



ABSTRACT BOOK

CONCURRENT SESSION PRESENTATIONS

Organized by Session and Presenter's Last Name

Concurrent: A1-Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 4/6

A drug-eluting injectable nanogel for localized delivery of anticancer drugs to solid tumors

Presenter: Mohsen Akbari, University of Victoria

ABSTRACT_ID#2849

ABSTRACT

Systemically administered chemotherapy reduces the efficiency of the anticancer agent at the target tumor tissue and results in distributed drug to non-target organs, inducing negative side effects commonly associated with chemotherapy and necessitating repeated administration. Injectable hydrogels present themselves as a potential platform for non-invasive local delivery vehicles that can serve as a slow-releasing drug depot that fills tumor vasculature, tissue, or resection cavities. Herein, we have systematically formulated and tested an injectable shear-thinning hydrogel (STH) with a highly manipulable release profile for delivering doxorubicin, a common chemotherapeutic. By detailed characterization of the STH physical properties and degradation and release dynamics, we selected top candidates for testing in cancer models of increasing biomimicry. Two-dimensional cell culture, tumor-on-a-chip, and small animal models were used to demonstrate the high anticancer potential and reduced systemic toxicity of the STH that exhibits long-term (up to 80 days) doxorubicin release profiles for treatment of breast cancer and glioblastoma. The drug-loaded STH injected into tumor tissue was shown to increase overall survival in breast tumor- and glioblastoma-bearing animal models by 50% for 22 days and 25% for 52 days, respectively, showing high potential for localized, less frequent treatment of oncologic disease with reduced dosage requirements.

LAY SUMMARY

Localized Delivery Reduces Systemic Side Effects: Systemic chemotherapy, like doxorubicin, often delivers low drug concentrations to tumors while causing widespread side effects in non-target organs. Injectable STHs offer a non-invasive solution by acting as slow-releasing drug depots within tumor tissue or resection cavities, minimizing systemic toxicity and reducing the need for frequent dosing. **Optimized STHs Enable Long-Term Drug Release:** The study developed and tested STHs with tunable physical properties and release profiles, achieving sustained doxorubicin release for up to 80 days. By characterizing degradation and release dynamics, top STH candidates were selected for testing, demonstrating effective drug delivery in breast cancer and glioblastoma models. **Improved Survival in Cancer Models:** In 2D cell culture, tumor-on-a-chip, and animal models, STH-delivered doxorubicin increased survival by 50% (22 days) in breast cancer and 25% (52 days) in glioblastoma models, showing high anticancer efficacy with lower doses and reduced toxicity compared to systemic administration.

Concurrent: A1-Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 4/6

Lipid nanoparticles for cancer diagnostics and therapeutics

Presenter: Miffy Hok Yan Cheng, University of British Columbia

ABSTRACT_ID#2592

ABSTRACT

Liposomes and lipid nanoparticles (LNPs) are versatile and relatively simple systems that have been widely applied across diverse fields, including basic science, pharmacology, cosmetics, and medicine. Over five decades of research have established these lipid-based carriers as effective vectors for both in vivo molecular imaging and nucleic acid delivery. Notably, LNP technology has contributed to the clinical success of several anticancer drugs, including Doxil®, Marqibo®, and, more recently, Onpattro® and Comirnaty®/Spikevax®. These latter examples highlight the utility of LNPs in gene delivery applications, such as the treatment of transthyretin-mediated amyloidosis and prophylactic mRNA vaccination against COVID-19, respectively. Interest in LNP systems has since surged, with over 350 ongoing clinical trials exploring LNP-mediated gene therapies. Expanding applications now include off-the-shelf CAR-T cell therapies, CRISPR-based genome editing, and personalized cancer vaccines. Lipid-based nanocarriers also offer a powerful platform for diagnostic imaging and therapeutic delivery in oncology. In this presentation, we describe a “one-for-all” liposomal formulation strategy that integrates novel organic chromophores (aza-BODIPY and texaphyrin) to enable multimodal imaging, including fluorescence, photoacoustic, and SPECT/CT techniques. Furthermore, we present our recent advances in LNP-mediated mRNA delivery, emphasizing structural optimization to enhance translational efficiency and achieve targeted, extrahepatic gene expression. Collectively, these efforts highlight the translational potential of lipid-based carriers in the emerging landscape of precision cancer nanomedicine.

LAY SUMMARY

Multimodal Functional Nanoparticle with a Simple Design: With a single building block, we formulated a liposome that can be tracked using multiple imaging techniques, including fluorescence, photoacoustic, and SPECT/CT. This enables easier detection and monitoring of tumors, drug uptake, and optimal timing for treatment. **Improved mRNA Delivery Beyond the Liver:** By optimizing the structure of lipid nanoparticles (LNPs), we enhanced their ability to deliver mRNA more effectively and target organs beyond the liver. This advance opens the door to using LNPs for a wider range of gene therapies, including cancer treatments.

Concurrent: A1-Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 4/6

Novel perfluorocarbon nanoemulsions for photodynamic therapy of lung cancer

Presenter: Melissa Loeffen, University of Toronto

Other authors: Lili Ding, Giulia Kassab, Angela Liang, Tiffany Ho, Hanyi Weng, Juan Chen, Gang Zheng

ABSTRACT_ID#2741

ABSTRACT

Photodynamic therapy (PDT) has been investigated as a non-invasive treatment strategy for lung cancer. This approach uses photosensitizers to generate reactive oxygen species (ROS) in tumors upon light irradiation, promoting cancer cell death. Despite its potential to overcome limitations of conventional therapies, clinical application of PDT is challenged by tumor hypoxia and inefficient photosensitizer delivery and penetration into target tissues. Here, we present a novel perfluorocarbon nanoemulsion (PFC-NE) designed with a pyropheophorbide-lipid (PL) conjugate as the photosensitizer component in the shell and a perfluorocarbon (PFC) core, developed for PDT of lung cancer. PFCs, known for their biocompatibility with lung tissues, can enhance PDT efficacy by facilitating oxygen delivery, thereby increasing ROS generation. These PL PFC-NEs are engineered to address issues related to photosensitizer solubility and delivery while mitigating tumor hypoxia. PL PFC-NEs were synthesized by microfluidization and demonstrated favorable physicochemical properties, including uniform size and morphology, and desirable photophysical characteristics for PDT. They exhibited excellent colloidal stability during long-term storage and remained stable in both lung surfactant and serum conditions, supporting suitability for different administration routes. These nanoemulsions showed efficient intracellular uptake in A549 lung cancer cells and potent PDT activity, with nanomolar-range lethal concentration 50 (LC50) values under light activation. Preliminary in vivo studies further support their preclinical potential, demonstrating effective pulmonary distribution via both local and systemic administration, tumor accumulation and PDT efficacy in subcutaneous non-small cell lung cancer models. Collectively, these findings highlight PL PFC-NEs as a promising nanoplatform for enhancing PDT outcomes in lung cancer treatment.

LAY SUMMARY

A new type of perfluorocarbon nanoemulsion (PL PFC-NE), incorporating a pyropheophorbide-lipid conjugate photosensitizer to stabilize the PFC core, may enhance photodynamic therapy for lung cancer by addressing key challenges including tumor hypoxia and poor drug delivery. PL PFC-NEs exhibit favorable photophysical and physicochemical properties for lung cancer PDT, including efficient singlet oxygen generation and potent PDT efficacy in vitro. Preliminary in vivo results support the translational potential of this PDT platform, with ongoing studies investigating therapeutic efficacy with various administration routes.

Concurrent: A1-Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 4/6

Preclinical 3D Model Drug Repurposing Screen Identifies Digoxin as an Effective Anti-cancer Therapy for SWI/SNF-inactivated Endometrial and Ovarian Cancers

Presenter: Pooja Praveen Kumar, University of Alberta

Other authors: James Key, Guihua Zhang, He Dong, Vincent Maranda, Zoe Turner, Nelson Wong, Christa Aubrey, Melissa Lavecchia, Sophia Pin, Jeanelle Sabourin, Helen Steed, Tiffany Wells, Carol Ewanowich, Andrei Drabovich, Martin Köbel, Frederick Vizeacoumar, Franco Vizeac

ABSTRACT_ID#2962

ABSTRACT

Dedifferentiated endometrial/ovarian carcinomas (DDEC/DDOC) and small cell carcinoma of the ovary, hypercalcemic type (SCCOHT) are clinically aggressive gynecologic cancers characterized by SWI/SNF-complex inactivation with poor outcomes. To facilitate therapeutic advances, we developed patient-derived in vitro (3D spheroids) and in vivo models of DDEC (PMID: 37556934), DDOC and SCCOHT. Our high-throughput FDA-approved drug screen identified cardiac glycosides such as digoxin as an effective drug against DDEC (PMID: 38970843). Herein, we extend the drug screen to SWI/SNF-inactivated ovarian cancers. We are also evaluating the mechanisms of action of digoxin and identifying potential synergistic drug combinations with digoxin for the treatment of these cancer types. Drug screens on DDOC also identified cardiac glycosides as top hits. In vitro dose-response showed that DDOC and SCCOHT are sensitive to digoxin than non-DDOC/SCCOHT ovarian cancer cells. Moreover, to gain insights into the mechanisms of digoxin, mass spectrometry, western blot and PCR were performed with gene expression profiling underway. These analyses revealed that DDEC express different types and quantities of Na⁺/K⁺-ATPase isoforms (targets of cardiac glycosides) when compared to that of non-DDEC cells. Additionally, drug synergy screens were performed with low-dose digoxin which identified drugs such as homoharringtonine and bortezomib as potentially effective drug combinations. Overall, digoxin was identified as a potential systemic therapy for SWI/SNF-inactivated endometrial/ovarian cancers. Further in vitro and in vivo studies are planned to evaluate the efficacy of the drug combinations. These findings suggest an exciting new role for digoxin as an anti-cancer drug for these clinically highly aggressive tumor types.

LAY SUMMARY

Patient-derived 3D models of SWI/SNF-inactivated endometrial and ovarian carcinomas were subjected to drug repurposing screens, which identified the cardiac glycoside digoxin as an effective anti-cancer drug. Mechanistic studies (mass spectrometry, western blot and PCR) revealed that the SWI/SNF-inactivated endometrial and ovarian carcinomas differentially express Na⁺/K⁺-ATPase isoforms in comparison to other cancer types. Drug synergy screen identified chemotherapeutic agents such as homoharringtonine and bortezomib as potential combinatorial drugs with low-dose digoxin.

Concurrent: A2-Immunotherapy

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 1/2/3

Inactivation of TRAF3 and CYLD Causes Resistance to T-Cell Based Therapies in Multiple Myeloma by Promoting an Immunosuppressive Microenvironment that Alters T Cell Function

Presenter: David Jung, Arnie Charbonneau Cancer Institute, University of Calgary

Other authors: Holly Lee, Sejal Chikhale, Sacha Benaoudia, Mansour Poorebrahim, Sungwoo Ahn, Noemie Leblay, Elham Hasheminasabgorji, Nizar Bahlis, Paola Neri

ABSTRACT_ID#3012

ABSTRACT

Multiple Myeloma (MM) is a prevalent blood cancer defined by the abnormal proliferation of plasma cells within the bone marrow. Despite recent advancements in treatment options, MM remains an incurable disease. Chimeric antigen receptor (CAR) T cells and T cell engagers (TCEs) targeting BCMA have shown promising efficacy in MM. However, responses are not universal and acquired resistance occurs. We recently reported enrichment of NF- κ B-activating mutations, including loss of TRAF3 and CYLD, in patients with acquired resistance to anti-BCMA therapy. Here, we investigate the functional consequences of these alterations in MM. Using CRISPR-Cas9 we generated TRAF3 and CYLD knockout (KO) clones in OPM2 and ARP1 MM cell lines. Knockouts were validated by western blot and NF- κ B pathway activation confirmed via ELISA. In flow cytometry-based cytotoxicity assays, TRAF3 and CYLD KO cells exhibited resistance to anti-BCMA CAR-T (ide-cel), TCE (elranatamab) and GPRC5D-targeting immunotherapies (arlo-cel and talquetamab). Transcriptomic profiling (scRNA-seq) of the KO cells revealed enrichment of interferon response and several anti-apoptotic genes, including MCL-1 and BCL-2. Cytokine analysis of KO supernatants revealed elevated immunosuppressive factors, including soluble Fas (sFas), IL-10, and CXCL10. In particular, we demonstrated that CXCL10 impaired T cell-mediated killing in co-culture assays. Additionally, flow cytometry confirmed the upregulation of Fas in KO cells as the source for the increased levels of sFas. Our data reveal a tumor-intrinsic mechanism of resistance whereby NF- κ B activation via TRAF3 or CYLD loss suppresses T cell cytotoxicity through CXCL10 and sFas, supporting the need for combinatorial strategies to overcome immune escape in MM.

LAY SUMMARY

- Enrichment of NF- κ B-activating mutations, including loss of TRAF3 and CYLD, is observed in myeloma patients with acquired resistance to anti-BCMA therapies. In this study, we have investigated in vitro the functional consequences of these alterations in multiple myeloma cells and evaluated their role in the development of resistance to T-cell based therapies (CAR T and T cell engagers).
- We have demonstrated that the inactivation of TRAF3 and CYLD drives resistance to T-cell-based therapies by promoting interferon response, expression of anti-apoptotic proteins and elevated levels of multiple inhibitory factors and cytokines.
- This is a novel mechanism of resistance to T-cell-based therapies mediated by a tumor intrinsic inactivation of TRAF3 and CYLD that results in the upregulation of T cells suppressive chemokine able to decrease T cells functions.

Concurrent: A2-Immunotherapy

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 1/2/3

Determining the role of the microbiome and microbial metabolites in enhancing the efficacy of CD19 directed CAR-T cell therapy of B cell malignancies

Presenter: Larisa Kovtonyuk, Calvin, Phoebe and Joan Snyder Institute for Chronic Diseases, University of Calgary

Other authors: Amy Shupe, Sacha Benaoudia, Robert Puckrin, Mona Shafey, Kyle Potts, Douglas Mahoney, Kathy McCoy

ABSTRACT_ID#3290

ABSTRACT

Adoptive T cell therapies have been under intense development for the past decades. Despite the success of chimeric antigen receptor (CAR)-T cell therapy, nearly 50% of patients with B cell malignancies either do not respond to the treatment or relapse. The microbiome and microbial metabolites have been found to modulate the anti-cancer function of T cells in immune checkpoint inhibitor therapy. Recent studies have demonstrated differences in the composition of the microbiome between responder and nonresponder patients and have identified some bacterial species that are associated with responsiveness or toxicity. We hypothesize that specific intestinal microbial species can influence CD19CAR-T cell therapy via secreted metabolites. We have established germ-free NSG (KbDb) null mice to investigate whether the presence of a microbiota alters the efficacy of CAR-T cell therapy and to elucidate the underlying mechanisms involved. Our preliminary data show that CD19CAR-T cells transferred into germ free mice that received luciferase expressing Nalm6 cells can eliminate tumor cells in vivo. However, CD19CAR-T cells perform better and eliminate tumor cells more efficiently in mice harboring a diverse specific pathogen-free (SPF) microbiota. Moreover, CD19CAR-T cells perform better in mice mono-colonized with inosine producing *Bifidobacterium pseudolongum* or *Akkermansia muciniphila* compared to germ free mice, due to higher expression of TNF α and degranulation marker CD107a. In contrast, mono-colonization with bacteria that does not produce inosine *Colidextribacter* spp. did not aid CD19CAR-T cell function compared to germ free mice, indicating that only specific bacterial species can modulate CAR-T cell performance in vivo. Further studies will elucidate the specific metabolites involved.

LAY SUMMARY

Key messages: - Microbiome can aid CD19 directed CAR-T cell therapy - Specific microbial species *Bifidobacterium pseudolongum* or *Akkermansia muciniphila* aid CD19 directed CAR-T cell function, possibly via inosine

Concurrent: A2-Immunotherapy

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 1/2/3

Harnessing stem cells as a scalable and cost-effective source of T cells for off-the-shelf immunotherapy

Presenter: Yale Michaels, University of Manitoba

Other authors: Carla Zimmerman, Peter Zandstra, John Edgar, Dana Segal, Matthew Major

ABSTRACT_ID#2808

ABSTRACT

Chimeric antigen receptor (CAR) T cell therapies have revolutionized the treatment of certain blood cancers, yet their reliance on patient-derived T cells is a major barrier to accessibility. Manufacturing autologous engineered T cell products is prohibitively expensive and highly variable across patients. To overcome these limitations, we developed a clinically compatible system for differentiating human stem cells, including pluripotent stem cells and umbilical cord blood stem cells, into T cells that are suitable for CAR engineering. Unlike previous protocols, our system is fully serum- and feeder-free and uses only defined recombinant proteins and small molecules. By optimizing cytokine exposure and notch pathway stimulation, we were able to increase the yield of mature T cells by ~100-fold. These stem cell-derived T cells mirror key features of their primary equivalents. They display a diverse T cell receptor repertoire and proliferate extensively in response to stimulation. To characterize and further improve upon our differentiation method, we performed longitudinal lineage tracing combined with single cell RNA sequencing. This analysis revealed that mast cells and myeloid cells were unintended by-products of in vitro T cell production. Guided by these sequencing results, we shortened the culture stage where these off-target lineages arise. This modification allowed us to significantly increase the purity of the intended T cell product. Our optimized in vitro differentiation platform provides a renewable and standardised source of T cells, laying the foundation for scalable, off-the-shelf CAR-T therapies.

LAY SUMMARY

We developed a new method to create T cells, the immune cells used in CAR-T cancer therapy, from stem cells instead of obtaining them from patients. This could help make these powerful treatments more widely available by reducing manufacturing costs and wait times. Our method uses only well-defined ingredients, like proteins and small molecules, and avoids animal products. This makes it safer, more consistent, and easier to scale up for future clinical use. We discovered that some unwanted cell types were being made alongside the T cells, so we used advanced genetic tools to identify when and how they were forming. By adjusting the timing of our process, we were able to increase the purity of the T cells we produced.

Concurrent: A2-Immunotherapy

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 1/2/3

Using Double Negative T-Cells to Enhance the Innate-Like Anti-Leukemic Activity of CD8+ T cells Against Acute Myeloid Leukemia

Presenter: Leanne Palichuk, University of Calgary

Other authors: Enoch Tin, Juan Arteaga, Michele Nawata, Pauline Douglas, Benjamin Muselius, Sonia Cerquozzi, Monica Modi, Lynn Savoie, Michelle Geddes, Etienne R. Mahe, David Schriemer, Victor A. Lewis, Jongbok Lee

ABSTRACT_ID#2325

ABSTRACT

Acute myeloid leukemia (AML) is a blood cancer originating from myeloid cells in the bone marrow, with poor prognosis in relapsed/refractory patients. Double negative T cells (DNTs), a rare immune cell subtype, have shown anti-AML properties. Our research demonstrated that healthy donor-derived DNTs effectively targeted AML in vitro and in vivo, without off-tumor toxicity. In a phase I clinical trial, 10 AML patients who relapsed after allogeneic hematopoietic stem cell transplantation received third-party donor-derived DNT. None of the patients experienced severe adverse events and five achieved complete remission. However, the mechanisms which DNTs influence host immune cells' anti-leukemic function remains unclear. This project aims to elucidate the synergy between DNTs and CD8+ T cells in enhancing AML targeting. Using proteomic analysis, we identified unique proteins up-regulated in CD8+ T cells cultured with DNT+AML, compared to those cultured with AML alone, or α CD3/CD28 beads, which included proteins involved in innate immune response and ferroptosis. Interestingly, the cytotoxic capability of DNT-activated CD8+ T cells against AML cells was significantly reduced upon treatment with a ferroptosis inhibitor. Furthermore, these activated CD8+ T cells efficiently targeted AML cells lacking MHC class I expression. These findings suggest that, in addition to direct cytotoxic mechanisms, DNTs potentiate CD8+ T cell-mediated AML clearance via a non-conventional, MHC class-I independent and ferroptosis dependent pathway. This highlights a synergistic anti-leukemic response between DNTs and CD8+ T cells.

LAY SUMMARY

Acute myeloid leukemia (AML) patients who relapse after a stem cell transplant have low survival probabilities and limited treatment options. Double negative T cells (DNTs) are a rare type of white blood cell which have been shown to target and kill AML. DNTs can help other immune cells in the patients body to better target and kill AML, without worsening graft-versus-host disease.

Concurrent: A3-Pediatric Cancer Across the Patient Journey

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 5/7/9

Alpha2 integrin mediates chemoresistance in human acute T cell lymphoblastic leukemia

Presenter: Fawzi Aoudjit, Université Laval

Other authors: Chakib Hamoudi, Anahita Lashgari

ABSTRACT_ID#2723

ABSTRACT

Resistance to chemotherapy (chemoresistance) is a critical factor in cancer recurrence including in acute T cell lymphoblastic leukemia (T-ALL). Growing evidence indicates that cell adhesion to extracellular matrix plays an important role in cancer resistance. Leukemic T cells express several adhesion receptors of the $\beta 1$ integrin subfamily, however, their role in T-ALL chemoresistance is still ill defined. We showed that interactions of human T-ALL cell lines and primary blasts with three-dimensional matrices like collagen gels promoted their resistance to various drugs via $\alpha 2\beta 1$ integrin. Further, ectopic expression of $\alpha 2$ integrin induces collagen-mediated chemoresistance in a T-ALL cell line that does not express endogenous $\alpha 2$ integrin. Although the collagen receptor discoidin domain receptor 1 is expressed in some T-ALL cells, we found that its inhibition did not affect collagen-mediated chemoresistance in T-ALL cells indicating that the pro-survival effect of collagen is mediated mainly through $\alpha 2$ integrin. Importantly, the blockade of $\alpha 2$ integrin using an antibody or its knockdown through CRISPR sensitized xenografted T-ALL cells to chemotherapy, diminished the leukemic burden and resulted in extension of animal survival suggesting that $\alpha 2$ integrin plays a major role in T-ALL chemoresistance. Mechanistically, collagen enhanced T-ALL chemoresistance by promoting drug efflux through the activation of the drug transporter ABCC1. Together these results support a major role for $\alpha 2$ integrin in T-ALL chemoresistance and patient relapse and suggest that its blockade could enhance chemotherapy treatment and lead to better outcomes especially in cases where T-ALL cells express high levels of $\alpha 2$ integrin.

LAY SUMMARY

The development of drug resistance or chemoresistance plays a major role in cancer recurrence and patient relapse and is associated with poor prognosis. This is the case in acute T cell lymphoblastic leukemia (T-ALL) and therefore, understanding the mechanisms of chemoresistance can lead to better therapies. We found that disruption of T-ALL cells interactions with their microenvironment by targeting a membrane receptor called $\alpha 2$ integrin sensitizes T-ALL cells to chemotherapy-induced killing and led to enhanced animal survival in a mouse model of T-ALL. We conclude that targeting $\alpha 2$ integrin with specific inhibitors in combination with chemotherapy may lead to better eradication of T-ALL cells, reduce chemotherapy side effects, avoid relapse and lead to better clinical outcomes in T-ALL patients.

Concurrent: A3-Pediatric Cancer Across the Patient Journey

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 5/7/9

A National Pediatric Preclinical Modelling Program to Advance Precision Oncology for Childhood Cancers in Canada

Presenter: Nadine Azzam, CHEO Research Institute

Other authors: Stacey Farrand, Ian Kawpeng, Gregor Reid, James Lim, Donna Senger, Jason Berman

ABSTRACT_ID#3013

ABSTRACT

Despite advances in pediatric cancer research, treatment for children, adolescents, and young adults (CAYA) with cancer has remained heavily reliant on non-targeted chemotherapies with varying success and risk of serious acute and long-term side effects. Although next-generation sequencing (NGS) has enabled the identification of genetic alterations, translating these findings into effective, personalized treatments has been hindered by a lack of functional preclinical validation. To address this gap, we established a Pediatric Preclinical Modelling Program to support precision-based therapy selection for CAYA patients across Canada. Building on the infrastructure of the PRecision Oncology For Young peopLE (PROFYLE) initiative, the program integrates individualized preclinical models—including zebrafish, chick embryos, and mouse patient-derived xenografts (PDX)—to test the functional response of tumours to targeted therapies. By employing the most appropriate model/s to meet the specific clinical need, rapid validation of molecular findings and assessment of drug efficacy can improve the precision of treatment recommendations and minimize toxicity. The program collaborates with and is supported by the ACCESS (Advancing Childhood Cancer Experience, Science & Survivorship) network and persons with lived/living experience (PWLE) to ensure equitable availability of these tools to clinicians and researchers nationwide. Our workflows, procedures, and biobanking allow for the systematic collection, modelling, and testing of patient tumours, creating a renewable resource for both real-time clinical translation and long-term research. This platform represents a transformative approach to pediatric oncology in Canada. By providing pre-clinical data to inform clinical decision-making, the program is enhancing treatment outcomes and shaping the future of precision oncology.

LAY SUMMARY

Even though there have been big advances in childhood cancer research, many children and young adults with cancer are still treated with traditional chemotherapy. These treatments do not always work and can cause short- and long-term side effects. While modern genetic testing can find specific changes in cancer cells, it is still difficult to turn that information into personalized treatment options. To help solve this problem, the Pediatric Preclinical Modelling Program, which was part of the national PRecision Oncology For Young peopLE (PROFYLE) initiative, creates patient avatars - using zebrafish, chick embryos, and mice - to test how each patient's cancer responds to different treatments based on the unique features of their tumours. By choosing the right model/s for each case, treatments identified as most effective for each individual patient are used to help doctors make better, more personalized decisions. The program collaborates and is supported by the ACCESS (Advancing Childhood Cancer Experience, Science & Survivorship) network and persons with lived/living experience (PWLE), making sure that these tools are available to all patients across Canada. This innovative program is going to help change the way childhood cancer is treated in Canada, helping more young patients receive the right treatment at the right time.

Concurrent: A3-Pediatric Cancer Across the Patient Journey

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 5/7/9

How Children Want to Know: Identifying Knowledge Dissemination Preferences in Pediatric Oncology

Presenter: Ekaterini Damoulianos, McGill University

Other authors: Emily K. Drake, Michel Duval, Ramandeep Arora, Patrick Cossette, Leandra Desjardins, Megan Doherty, Sarah Houmad, YiQing Lü, Catherine Moura, Samiratou Ouédraogo, Argerie Tsimicalis

ABSTRACT_ID#2977

ABSTRACT

Background: Understanding how children prefer to receive evidence about cancer is critical to bridging the gap between evidence and meaningful patient engagement in pediatric oncology. However, children's own preferences for receiving evidence remain underexplored, leaving their voices underrepresented. Without child-informed knowledge dissemination approaches, we miss key opportunities to enhance how children access information, understand cancer, and receive support. **Aims:** To: (1) identify and prioritize the knowledge dissemination preferences of children regarding cancer of childhood onset and (2) create and validate a self-reported questionnaire assessing knowledge dissemination preferences from a child's perspective. Additionally, this aims to establish an inclusive recruitment strategy to capture children's diverse experiences across Canada. **Methods:** Through convenience and snowball sampling, approximately 110 children in Canada, including patients, siblings, and peers, will be recruited via the ACCESS (Advancing Childhood Cancer Experience, Science & Survivorship) network, targeted social media outreach, family, and other childhood cancer networks. Participants will complete a 20-item, child-friendly, digital questionnaire available in English and French on REDCap (McGill). The questionnaire will assess how, where, when, and with whom children prefer to learn about cancer of childhood onset. The questionnaire will undergo psychometric validation, with data analyzed through descriptive and multivariate statistics. **Significance:** As the first national study of its kind, this work will highlight children's knowledge dissemination preferences, informing the development of child-centered knowledge mobilization products and resources. The validated questionnaire will serve as a tool for other researchers and clinicians to ensure knowledge dissemination strategies align with children's preferences, empowering active engagement in child healthcare.

LAY SUMMARY

Centering Children's Voices: This study directly engages children affected by pediatric cancer, such as patients, siblings, and peers, to understand their preferred ways of receiving cancer-related information. By prioritizing their perspectives, the findings of this study will challenge adult-led approaches and ensure that knowledge dissemination approaches in pediatric oncology are tailored to children's preferences and unique needs. This shift empowers children as active participants in their healthcare journey.

Nationwide & Inclusive Insights: Using a bilingual (English/French), child-friendly digital questionnaire, the study captures diverse perspectives from children across Canada. By including not only patients, but also siblings and peers, this research acknowledges the broader social impact of childhood cancer and ensures that knowledge-sharing approaches are inclusive and representative of varied lived experiences.

Building a Practical Tool for Child-Centered Care: The study's questionnaire serves a dual purpose in guiding the creation of evidence-based resources and products that align with how children learn best, and establishing a standardized assessment tool for other researchers and clinicians. The questionnaire is easily adaptable, which allows application beyond pediatric oncology, setting a new benchmark for child-centered knowledge mobilization in healthcare.

Concurrent: A3-Pediatric Cancer Across the Patient Journey

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 5/7/9

Advancing Equitable Access to Pediatric Oncology Trials: Applying the Canadian Remote Access Framework for Clinical Trials (CRAFT) at BC Children's Hospital

Presenter: Rebecca J. Deyell, BC Children's Hospital

Other authors: Hina Johnstone

ABSTRACT_ID#3077

ABSTRACT

The Canadian Remote Access Framework for Clinical Trials (CRAFT) is a national initiative addressing barriers to clinical trial participation for patients in rural, remote, and underserved communities. By supporting decentralized trial models, CRAFT aims to bring research closer to home, reducing travel, logistical, and financial burdens that limit equitable access to innovative therapies. In this Canadian Cancer Clinical Trials (3CTN) pilot project, BC Children's Hospital (BCCH) is applying CRAFT principles to expand access to early phase pediatric oncology trials across British Columbia. Children with relapsed or refractory cancer and their families must balance the impact of early phase trial participation with achieving optimal quality of life; a particular challenge when care is far from home. BCCH has engaged providers (pediatricians, nurses and general practitioners in oncology) in five regional communities. We have developed an onboarding process, including streamlined regulatory training, to establish shared-care models enabling local coordination of trial activities such as assessments, safety monitoring, and drug accountability with central site oversight. To support decentralized activities, BCCH will leverage REALTIME clinical trial management system technology and eSOURCE tools for efficient remote data capture, monitoring, and communication between sites. We will also engage people with lived experience through focus groups to gather their insight and perspectives to ensure this initiative reflects patients' and families' needs and priorities. Early engagement shows strong local interest and feasibility. BCCH's experience adapting CRAFT will provide valuable insights into tailoring national frameworks to promote more inclusive, accessible, and equitable pediatric oncology clinical trial access across Canada.

LAY SUMMARY

- Early phase clinical trials in pediatric oncology are limited and currently require travel and prolonged stays at primary study sites, located in just a few large cities in Canada.
- The pediatric oncology clinical trials unit at BC Children's Hospital is establishing a pilot decentralized clinical trial program with the support of providers in five regional communities in BC.
- There has been excellent early engagement in this concept by local teams, who are collaborating to undertake streamlined regulatory training in order to participate in a shared care model of patients in local communities with oversight from the central study team.

Concurrent: A3-Pediatric Cancer Across the Patient Journey

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 5/7/9

Geographic access to pediatric cancer centers in Canada

Presenter: Tona Pitt, University of Calgary

Other authors: Tony Truong, Fiona Shulte, Rizwan Shahid, Xiaoxiao Liu, Paul Gibson, Miranda Fidler-Benaoudia

ABSTRACT_ID#2443

ABSTRACT

Background: Paediatric cancer patients require intensive treatment from oncology specialists at dedicated paediatric facilities. Various factors, like distance to treatment and travel time, act as barriers that impede families' access to cancer care, possibly affecting outcomes. We aim to estimate travel times and distance for paediatric cancer patients in Canada and examine the association of time/distance with mortality and relapse. Methods: This study utilizes the Cancer in Young People in Canada national pediatric cancer registry. All paediatric cancer patients diagnosed from 2001-2020, treated at one of the 17 paediatric cancer centres in Canada, and with a recorded home postal code (99.2%) were included. Results: From 2001-2020, 20,128 children were diagnosed with cancer. These children were mostly male (54.8%), had a median age at first diagnosis of 5.3 years, and 82.5% lived in urban settings. 12.8% experienced recurrence within 5-years of initial diagnosis and 5-year survival was 85.9%. Median distance to treating paediatric hospital was 30.2km (interquartile range: 12.5-102) and ranged from 1.7km to 3,817.3km. Preliminary, univariate logistic regression results indicated that the odds of dying was 12% higher (OR: 1.12, 95%CI: 1.01-1.25) among survivors of childhood cancer living more than 200km from their treating paediatric hospital compared with those living within 50km; no association was observed between travel distance and recurrence. As this research is ongoing, future directions include developing multivariable models to adjust on relevant clinical and social covariates. As well, car travel time to hospital will be calculated and used to estimate the odds of relapse and death.

LAY SUMMARY

Examining all cases of paediatric cancer in Canada across a 20-year period, preliminary results indicate that further distance to hospital is associated with poor long term outcomes in children diagnosed with cancer. This may indicate inequitable access to ongoing care for those in rural communities.

Concurrent: A4-Patient Centred Research/Patient Engagement

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 8

Patient perspectives of screening brief intervention and referral for cancer-related lifestyle risk factors across healthcare settings

Presenter: Manuel Ester, University of Calgary

Other authors: Sharon Mah, Gary F. Teare, Kimberly Manalili, Kamala Adhikari

ABSTRACT_ID#2580

ABSTRACT

Background Physical activity, tobacco, and alcohol use are three crucial modifiable lifestyle risk factors for cancer. Implementation of screening, brief intervention, and referral (SBIR) strategies in healthcare can significantly improve health behaviors, thereby reducing cancer risk. This qualitative study explored patient perspectives of participating in SBIR across diverse healthcare settings. **Methods** Alberta Health Services is implementing a co-created SBIR approach in acute and ambulatory care settings. To better understand perspectives on SBIR, diverse patients were purposively recruited to complete semi-structured interviews 1-month post-SBIR. Interview questions and qualitative descriptive analyses were informed by the Consolidated Framework for Implementation Research 2.0, the behavior change wheel, transtheoretical model, and health stigma and discrimination framework. **Results** Interviews with 22 patients from diverse healthcare settings and sociodemographic backgrounds emphasized key determinants to implementing SBIR and supporting health behavior change. Implementation barriers included patients internalized and perceived stigma and judgement (need to be addressed), readiness to change (motivation), and lack of provider referrals (opportunity). Notable facilitators were patient motivation and awareness (capability), positive attitudes towards reminders (motivation), and trusting relationships (patient-centeredness). Barriers to change included lack of knowledge about negative consequences or supportive resources (capability), long-standing habits (motivation), and challenges related to cost and accessibility (opportunity). Some change facilitators were improved understanding (capability), positive outcomes of change (motivation), and provider encouragement (patient-centeredness). **Conclusion** Patient perspectives provided valuable insights to optimize SBIR implementation and support health behavior change, enhancing the quality of future brief interventions. These findings will inform SBIR use to reduce cancer risk across Alberta.

LAY SUMMARY

Healthcare appointments in diverse settings are valuable opportunities to support individuals to move more, smoke and drink less. These changes can help individuals live better and reduce their risk of cancer. Meaningful patient-provider conversations about health behaviors require positive attitudes and acceptance from patients, trusting relationships without judgement, and providers connecting patients to useful resources. Behavior change is challenging because of social habits, not knowing about the consequences of behavior or where to get good support and not being able to take advantage of support due to cost or location. Better understanding of why and how making a change matters, seeing improvements first-hand, and encouragement from healthcare teams can help.

Concurrent: A4-Patient Centred Research/Patient Engagement

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 8

Experiences of Partners of Patients diagnosed with Cancer: A Systematic Review and Thematic Synthesis of Qualitative Literature

Presenter: Preet Kang, University of British Columbia

Other authors: Mary De Vera

ABSTRACT_ID#3258

ABSTRACT

BACKGROUND Cancer is a complex diagnosis, which impacts patients as well as their network, including their partners. Our objective was to conduct a systematic review and thematic synthesis of qualitative studies that examined experiences among partners of cancer patients. **METHODS** We searched MEDLINE, EMBASE, CINAHL, PsycINFO and Scopus for the following: qualitative research study, adult (≥ 18 years) participants who are partners (e.g., in a romantic relationship) of patients diagnosed with cancer and publications in English. We extracted findings from included studies and applied thematic synthesis to develop descriptive and higher-order analytical themes. **RESULTS** Our search identified 9,212 records. Included studies had sample sizes ranging from 4-30, with participants being predominantly female (Male=32%, Female=65%) and middle-aged (mean age=56 years old). Most partners were in relationships with patients diagnosed with breast, prostate, colorectal, head and neck or lung cancer. Analytical themes and descriptions are as follows: 1) Impact of partners on cancer patients (decision-making for treatment options, managing daily life, caregiving), 2) Change in relationship dynamics (stress and caregiving burden added strain to the relationship and intimacy), 3) Emotional burden on the partners (compromised quality of life and unmet personal needs) and 4) Need for support (emotional support from community, accommodations from employers, information from healthcare providers). **CONCLUSIONS** These findings emphasize the importance of raising awareness and advocating for support and resources not only for cancer patients but also for their partners.

LAY SUMMARY

The key takeaways are as follows:

1. Cancer affects the mental health and daily life of both patients and their partners.
2. Cancer can change relationship dynamics and family roles.
3. 3. Partners need more emotional, mental, and financial support.

Concurrent: A4-Patient Centred Research/Patient Engagement

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 8

Patient Voices in Research: A patient-centred and patient-driven research funding initiative from the Marathon of Hope Cancer Centres Network (MOHCCN)

Presenter: Rosilene Kraft and Katharine Palmer, MOHCCN Patient Working Group

Other authors: Nicole Beauchemin, Louise Bird, Sheilah Brukewich, Jennifer Coish, Melissa Coombs, Debbie Duclos, Kirsten Efremov, Jennifer Graham, Lindsay Gustafson, Heather Hogan, Sarah Hunt, Anne Marie Ireland, Summer Konechny, Natalie Kwadrans, Natha Nathalie Lamarche-Vane, Yuan Lew, Catalina Lopez-Correa, Christine McKay, Dan Murphy, Sally Nystrom, Darrin Park, James Pereira, Denis Petitclerc, Lisa Primrose, Beverley Riediger, Paul Sararu, Kathy Smith, Randy Thompson, Patricia Turnbull, Christine Qiong Wu, Terry Fox Research Institute Marathon of Hope Cancer Centres Network

ABSTRACT_ID#3069

ABSTRACT

The MOHCCN Patient Working Group (PWG) comprises more than 30 cancer patients, survivors and caregivers from nine provinces. These individuals reflect diverse age groups, life experiences, professional expertise, rural/urban settings, and ethnic and cultural backgrounds, including Indigenous members. Established in May 2023, the group helps ensure that Network activities address the needs and priorities of those with lived cancer experience and promote equitable research and care. In 2024, the PWG co-developed the Patient Voices in Research (PVR) initiative. We believe this is the first Canadian cancer research funding program fully designed and adjudicated by patients, survivors, and caregivers. The program was based on six themes identified by PWG members as priority areas in precision oncology: equity, access and disparities in precision medicine care; clinical trials access; patient engagement in precision oncology research; survivorship care plans and post-treatment resources; lifestyle interventions in precision oncology; and rare cancers across the age spectrum. Remarkably, this program garnered a MOHCCN record of 38 applications. Nineteen PWG members evaluated applications for scientific merit; relevance to the research themes; use of plain language; plans for patient engagement and involvement; and adherence to equity, diversity, and inclusion principles. Ten outstanding applications were funded (\$1.8M in total) for one year starting in December 2024. Project progress is currently monitored by two PWG liaisons per grant and communicated with the larger PWG. This endeavour demonstrates a significant interest in patient-centred research from the Canadian cancer research community as well as the feasibility and value of patient-led funding initiatives.

LAY SUMMARY

The Marathon of Hope Cancer Centres Network (MOHCCN) Patient Working Group (PWG) brings together more than 30 cancer patients, survivors and caregivers from across Canada to advance precision oncology. To further explore and promote patient-centred precision oncology research, in 2024 the group co-developed a new MOHCCN funding program: the Patient Voices in Research initiative. Through several meetings, both virtual and in person, PWG members identified six themes as priority areas for precision oncology research. The program sought submissions for one-year projects addressing one or more of the themes. Thirty-eight applications were received and reviewed by PWG members, and ten research teams were awarded a total of \$1.8M. In this presentation, we will share the PWG's experience in designing and adjudicating this unique funding program. The initiative demonstrated a significant interest in patient-driven and patient-centred research from the Canadian cancer research community, highlighting an unmet funding need. It also underscored the PWG's thoughtful and active engagement, demonstrating the eminent feasibility and value of patient-led funding initiatives.

Concurrent: A4-Patient Centred Research/Patient Engagement

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 8

Goals, accomplishments, and future plans of the Atlantic Cancer Consortium Patient Advisory Committee (ACC PAC)

Presenter: Georgia Skardasi, Memorial University of Newfoundland

Other authors: John King, Judy Donovan Whitty, Beverly Pausche, Jennifer Coish, Cyndi Corbett, Cara MacInnis, Gilles LeBlanc, Marcelo Fabian Martinez, Aaron Curtis, Sevtap Savas

ABSTRACT_ID#2568

ABSTRACT

Background: The Marathon of Hope Cancer Centres Network was launched in Canada to advance precision medicine in cancer. The Atlantic Canada Consortium (ACC) is a member of this network. In early 2024, a patient advisory committee (ACC PAC) was formed to advise the ACC researchers, identify patient priorities, and organize a public conference on cancer and precision medicine. **Objective:** To describe the goals, accomplishments, and future plans of the ACC PAC. **Methods:** The ACC PAC consists of 11 patients and family members representing all four Atlantic Canada provinces, a coordinator, a volunteer, an oncologist/clinician-scientist, and a cancer scientist. Since its formation, the ACC PAC held several virtual meetings to identify patient, family and community priorities in Atlantic Canada and to organize a public conference. The ACC PAC is currently focusing on finalizing its future goals. **Results:** Two key patient priorities identified by the ACC PAC were accessibility of precision medicine and preventing and addressing cancer's effects on the whole person/family. These results were published in a scholarly manuscript in March 2025. In the same month, the ACC PAC delivered a free, virtual, public conference hailed as highly successful and engaging by participants. Looking ahead, the ACC PAC plans to expand its public outreach, research involvement, and advocacy. **Conclusions:** The ACC PAC is an enthusiastic, successful, and impactful patient – researcher partnership. Its work can enrich and inform future research, public engagement activities, health care services, and advocacy on precision medicine and cancer.

LAY SUMMARY

Patient partnerships are essential for meaningful cancer research, public engagement, and improved healthcare services. The patient advisory committee that we describe here (ACC PAC) has been successful in identifying patient priorities and delivering an impactful public conference on cancer and precision medicine. The future plans of the ACC PAC focus on active involvement in research and public conversations about precision medicine in cancer.

Concurrent: A4-Patient Centred Research/Patient Engagement

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Imperial 8

Meaningful Patient Engagement in Economic Evaluations of Cancer Care: Are We There Yet?

Presenter: Kednapa Thavorn, Ottawa Hospital Research Institute

Other authors: Emily Thompson, Olivia D'Silva, Sharon Bede, Michael Halas, Chau Tran, Terry Hawrysh, Carol Gordon, Linda Riches, Deirdre Walsh, Brian Hutton, Justin Presseau

ABSTRACT_ID#3252

ABSTRACT

Background: As economic evaluations increasingly guide access to cancer therapies, it is essential that these analyses reflect the experiences and priorities of patients. While patient engagement in health research has expanded, its role in economic evaluations remains limited and inconsistent, which risks tokenism and overlooks key dimensions such as equity and lived experience. **Objectives:** This project aimed to (1) characterize how patients have been engaged in economic evaluations of cancer care, and (2) identify barriers and enablers to more meaningful engagement. **Methods:** We conducted a scoping review (December 2023) of MEDLINE, Embase, EconLit, and grey literature sources to identify studies that engaged patients in cancer-related economic evaluations. In parallel, we conducted a stakeholder survey with patients, clinicians, researchers, and decision-makers to explore perceived barriers and facilitators to engagement. **Results:** Twelve studies met inclusion criteria. Engagement most often occurred during early study phases (e.g. identifying outcomes of importance), with few examples of integration into analytic design or interpretation. Survey respondents (n=22) cited key barriers, including lack of funding, training, and methodological guidance; difficulty identifying and supporting patient partners; and uncertainty regarding engagement roles. Facilitators included institutional support and collaborative team culture. **Conclusions:** Despite growing calls for more inclusive value assessments, patient engagement in economic evaluations of cancer care remains superficial in most cases. Addressing systemic barriers and developing practical engagement frameworks are essential steps to moving beyond tokenism and achieving more patient-centered evaluations.

LAY SUMMARY

Patient engagement in economic evaluations of cancer care is still rare and often superficial. Most studies involved patients only at the beginning (for example, helping choose outcomes to measure), with little involvement in how the analysis was designed or interpreted. There are clear barriers to deeper engagement. These include lack of funding, limited training for both researchers and patient partners, and uncertainty about how to meaningfully involve patients in highly technical parts of economic evaluation. Addressing these gaps will lead to better decisions. When patient voices are fully included, economic evaluations can better reflect real-life experiences with cancer treatment, including quality of life, treatment burden, and equity concerns. This helps ensure that decisions about which treatments to fund truly align with what matters to patients and their families.

Concurrent: A5-Health Equity

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Doll/Herald

A qualitative study of Chinese immigrant families' experience with inpatient palliative care specialists

Presenter: Zhimeng Jia, University of Toronto

Other authors: Allison Kurahashi, Ramona Mahtani, Siyi Fan, Lingsheng Li, Irene Yeh, Richard Leiter, Justin Sanders, James Tulskey, Rashmi Sharma

ABSTRACT_ID#2461

ABSTRACT

Background: Compared to non-Chinese adults in high-income countries, ethnically Chinese patients are more likely to encounter palliative care (PC) closer to death and in hospital settings. Yet, Chinese families' experiences and perception of inpatient PC remain unknown. **Aim:** Identify barriers and facilitators to culturally-respectful PC for Chinese immigrant inpatients and their caregivers. **Design:** Prospective, exploratory qualitative design involving phenomenological interviews. **Setting/Participants:** We consecutively recruited (n=15) Chinese immigrant patients and their caregivers (n=14) referred to PC at one Canadian academic teaching hospital. We collected participant self-reported sociodemographics, Suinn-Lew acculturation level, and conducted semi-structured interviews (n=10) in Mandarin and/or English. The interviews were recorded, transcribed, translated, and thematically analyzed using Tan's Health Communication framework. **Results:** Patients were older-aged (mean=73.5±16.2 years), 53.3% female, 60% college-educated, 66.7% non-religious, 93.3% diagnosed with cancer, and had low acculturation (mean=1.8±0.9/5.0). Caregivers were middle-aged (mean=50.6±15.5 years), 78.6% children, 57.1% female, 85.7% college-educated, 71.4% non-religious, and had moderate acculturation (mean=2.5±1.2/5.0). We identified four themes from post-consultation interviews: abandonment and alienation mark past experiences with serious illness care; emphasizing expertise and symptom relief may help overcome initial ambivalence toward PC; PC brokers competing priorities within the family unit; and PC alleviates time-related distress by addressing illness understanding. **Conclusion:** Chinese patients and caregivers may prefer a PC approach that is sensitive to historical mistrust, leverages expertise in symptom management to inspire confidence, and accommodates the information and care preferences of the family unit. Further research is needed to examine the impact of these PC strategies on clinical outcomes for Chinese families.

LAY SUMMARY

What is already known about the topic? Chinese immigrant patients in high-income countries frequently experience potentially burdensome life-sustaining treatments at the end of life. Specialist palliative care in hospitals has been shown to improve patient-centered outcomes but its role and impact among Chinese immigrant families are understudied. What this paper adds? Emphasis on expertise and symptom relief may help overcome initial ambivalence toward palliative care. Palliative care brokers competing priorities within the family unit and alleviates time-related distress by addressing illness understanding. Implication for practice, theory or policy? To improve palliative care engagement among Chinese immigrant communities, clinicians may consider an approach that is sensitive to historical mistrust, leverages expertise in symptom management to inspire confidence, and accommodates the information and care preferences of the family unit.

Concurrent: A5-Health Equity

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Doll/Herald

Planning and Co-Creating an Equity-Oriented Model of Care: Baseline Findings & Lessons Learned

Presenter: Jagbir Kaur, BC Cancer

Other authors: Leah Lambert, Emily Lonsdale, Scott Beck, Tracey Rannie, Michael McKenzie

ABSTRACT_ID#3351

ABSTRACT

Background: Unjust disparities persist along the cancer continuum, particularly impacting Canada's equity-denied communities. These inequities are especially prevalent in the Downtown Eastside neighbourhood of Vancouver—one of the nation's most densely-populated and underserved health service delivery areas. Our Equity-Oriented Model of Care (E-MOC) project seeks to expand on the current interdisciplinary care model at BC Cancer – Vancouver, to advance objectives related to more equitable and person-centered care that supports those least well served by the system. Methods: As part of the multi-year project, our initial planning phase (2023-2024) surveyed cancer care staff and clinicians' perceptions of equity-oriented health care, conducted clinical observations of current practices, hosted community-facing workshops with service providers, and assessed baseline opportunities with people with lived/living experience of health and social inequities (PWLE-HSI). Results: Staff and clinician survey findings identified areas of strength and opportunities to better support the unique needs of PWLE-HSI who come for care at BC Cancer – Vancouver. Clinical observations further identified existing equity-oriented practices and workflows to promote person-centered care. Community-facing workshops and equity walk throughs provided concrete recommendations to encourage trust, safety, and accessibility throughout care interactions. Conclusion: E-MOC project planning activities led to the co-creation of a novel model of care to implement and evaluate at BC Cancer – Vancouver. Findings informed a tailored equity-oriented intervention that seeks to provide staff and clinicians the tools, supports, and infrastructure needed to tackle complex barriers to equitable care for those least well served by the system.

LAY SUMMARY

As part of a planning and co-creation phase in a multi-year quality improvement project, a diverse team of embedded health system researchers, staff and clinicians, and people with lived experiences of health and social inequities was formed. Baseline assessments of the implementation site identified gaps and opportunities to provide more equity-oriented care at BC Cancer – Vancouver. Community facing workshops and clinical observations highlighted key areas and practice changes to enable more equitable and person-centered care in daily practices.

Concurrent: A5-Health Equity

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Doll/Herald

Co-developing cancer research priorities with underserved communities: A patient-oriented health equity workshop in Alberta

Presenter: Anna Pujadas Botey and Caleb Chamoy, Alberta Health Services

Other authors: Anna Santos Salas, Harkeert Judge Judge, Nazret Russon, Harjeet Kaur, Molly Evans, Jessica Katerenchuk, Liz Ross, Paula Robson

ABSTRACT_ID#2485

ABSTRACT

Achieving equity in cancer care remains an urgent priority across Canada. Yet, populations historically underserved—such as members of racialized, rural, gender-diverse, and low-income communities—continue to face disparities in access to cancer services, participation in research, and health outcomes. To begin addressing these inequities, we are co-developing, with cancer care system, academic, patients and community partners, a first-of-its-kind provincial workshop in Alberta to identify research priorities aimed at advancing equity in cancer care. The two-day workshop will be held in fall 2025, and will engage over 70 participants, including patient partners, community leaders, healthcare providers, researchers, and decision-makers. Grounded in principles of patient-oriented research and guided by the Health Equity Implementation Framework, the workshop will explore three key themes: (1) the impact of living and working conditions on cancer care; (2) disparities in access to services; and (3) inequities in access to and engagement with research. The event will feature guest speakers, lived experience stories, and small group discussions to generate collective insights. Participants will collaboratively develop a set of priority research questions and actionable recommendations to strengthen equity-oriented cancer research infrastructure in Alberta. This initiative represents an innovative, equity-driven approach to setting a research agenda in cancer care. Preliminary learnings will be shared at the time of the conference. It highlights the importance of engaging underserved communities in shaping research priorities and building lasting partnerships among diverse stakeholders to bridge the gap between research, policy, and practice to improve cancer care access.

LAY SUMMARY

Some communities face additional barriers accessing cancer care and being involved in research. People living in rural areas, racialized individuals, those with lower incomes, and gender-diverse communities often experience challenges in accessing timely, high-quality care and being included in research. We are co-developing an innovative, first-of-its-kind provincial workshop in Alberta to listen and learn. More than 70 participants—including patients, caregivers, community organizations, healthcare providers, and researchers—will come together to identify the biggest gaps and most important research questions related to equity in cancer care. Together, we will create a list of research priorities. These priorities will be shaped by the lived experiences and insights of patients and communities, helping guide future efforts to improve access to care, make research more inclusive, and address social factors that affect cancer outcomes in Alberta.

Concurrent: A5-Health Equity

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Doll/Herald

Closing the Gap: Understanding and addressing sub-regional HPV immunization inequities in Canada

Presenter: Mika Rathwell, Urban Public Health Network, University of Saskatchewan

Other authors: Cory Neudorf, Thilina Bandara, Aine Dolin

ABSTRACT_ID#3001

ABSTRACT

The Canadian Partnership Against Cancer funded The Urban Public Health Network to conduct a quality improvement project to assess the landscape of HPV immunization coverage across Canada, as part of their Action Plan to Eliminate Cervical Cancer, 2040. With the target of 90 percent of 17-year-olds immunized by 2025, barriers to HPV immunization must be understood and efforts to increase immunization undertaken. This project engaged 6 public health units to examine HPV immunization data at a sub-regional level, using postal-code data where available and linking coverage rates to socio-demographic indicators where possible, to identify pockets of under-immunization. Additionally, qualitative data collection methods were utilized to identify facilitators and barriers that impact specific sub-populations, including Indigenous people, newcomers, and 2SLGBTQIA* people. Quantitative insights from six regions indicate no participating region has currently reached the 90% coverage goal and high coverage variability exists within regions. Material and social deprivation emerged as the most salient correlates of under-immunization in these regions. Further engagement suggests several common barriers, however, their relative influence on HPV immunization uptake varies between and within regions and populations. Common barriers include gaps in knowledge and understanding of HPV and the vaccine, lack of access points outside of school, lost and misplaced consent forms, and mistrust in healthcare systems. Results from this project informed recommendations to increase youth HPV immunization uptake. The second phase of this project provides funding to partners to develop tests of change to increase HPV immunization rates within their region and close gaps in vaccine equity.

LAY SUMMARY

Despite the widespread availability of the vaccine through school-based programs, inequities in coverage persist. Material and social deprivation emerged as the most salient correlates of under-immunization in participating regions. Existing inequities and barriers to HPV immunization must be addressed for vaccination targets to be met. Findings and recommendations from the first phase are being used by partners to identify high-impact targets that address pockets of under-immunization in their regions. Novel approaches are currently being trialed within various jurisdictions to address specific barriers.

Concurrent: A5-Health Equity

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Doll/Herald

Exploring barriers and priorities for including Trans & Gender Diverse people in Canadian cancer health services research

Presenter: Morgan Stirling, University of Manitoba

Other authors: Nathan Nickel, Annette Schultz, Janice Ristock, Versha Banerji, Jacqueline Gahagan, Murdoch Leeies, Erna Snelgrove-Clarke, Julie Hallet, Natalie Coburn, Tara Horrill, Kathleen Decker, Kim McGrail, Alyson Mahar

ABSTRACT_ID#2787

ABSTRACT

Background Trans and gender diverse (TGD) people commonly experience inequities across the entire cancer continuum. Establishing inclusive models of care to address inequities requires high-quality cancer health services research. This work must include strengthening researchers' capacity to integrate gender diversity in cancer health services research. Methods An online needs assessment survey was collaboratively developed with a Community Advisory Board comprised of TGD people with diverse cancer experiences. The 27-item survey invited Canadian cancer health service researchers to identify barriers and facilitators to integrating gender diversity in cancer health services research. We followed a snowball sampling approach for recruitment. We targeted invitations to Canadian cancer agencies and established cancer research organizations. Descriptive statistics, including frequencies, percentages, means and standard deviation, are reported. Results Ninety-one responses to the survey were received. Most respondents (95%) reported gender was an important variable in cancer health services research; yet, less than half (46%) reported routinely integrating gender and gender diversity in their research. The most commonly reported barrier was lack of inclusive data (71%). Improving data availability and quality are identified priorities, along with training on engaging TGD people in cancer research, establishing strategies for inclusive sampling/recruitment, and developing consistent approaches for describing TGD people in cancer health services research. Conclusion Improving data availability, addressing inadequate methods, and increasing comfort with engaging TGD people in research are key actions to improve cancer health services researchers' capacity for integrating gender diversity in research and ultimately support efforts to address TGD people's cancer inequities.

LAY SUMMARY

- Cancer health services researchers believe gender and gender diversity are important to include in their research.
- Researchers identified needing to improve their ability to engage trans and gender diverse people in cancer research. They also highlighted the importance of having clear and respectful ways to describe Trans and Gender Diverse people in research.

Concurrent: A6-Indigenous Knowledge and Methodologies in Cancer Research

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Stephen AB

Pathways to Building Cancer Education for First Nations Health Leaders

Presenter: Andrea Aiabens, First Nations Health Managers Association (FNHMA)

ABSTRACT_ID#2446

ABSTRACT

The FNHMA is embarking on an exciting new project made possible through a partnership with the Canadian Partnership Against Cancer (CPAC) that is focused on supporting First Nations Health Leaders gaining knowledge and tools related to cancer. The goal of the project is to increase the capacity of First Nation health leaders by developing and implementing a comprehensive public health workshop focused on the First Nations cancer journey. This includes discussions related to access to screening and preventive services and building in regional and jurisdictionally specific learning and information through workshop activities. This workshop will highlight what was heard from engagement with First Nations Health Managers through engagement sessions, focus groups and interviews. FNHMA will present the ideas and feedback that emerged from these sessions and explain the process and a draft outline of what the workshop will entail. Workshop participants will be invited to share their feedback on the proposed workshop outline.

LAY SUMMARY

Providing tools and resources to help build First Nations health leaders capacity in the cancer care journey.

Concurrent: A6-Indigenous Knowledge and Methodologies in Cancer Research

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Stephen AB

Applied First Nations Knowledge and Practice of Collaborative Processes: Recognizing multiple bodies of knowledge through orality to inform Practice Change within cancer care settings

Presenter: Lea Bill, Alberta First Nations Information Governance Center

Other authors: Barbara Frazer

ABSTRACT_ID#3177

ABSTRACT

The 'Alberta First Nations Cancer Strategy and Practice Change Implementation Initiative (AFNCI)' is a collaborative partnership project funded by Canadian Partnership Against Cancer (CPAC) utilized cultural engagement structures to ground First Nations worldviews. A core team of First Nations Knowledge Holders/Practitioners, led by Alberta First Nations Information Governance Centre and Alberta Health Services, worked with Cancer Care Oncology Champions in an unprecedented collaboration to inform cancer care of First Nation care needs required to support positive cancer care outcomes. AFNCI applied a First Nations phenomenology methodology that values the perspective of lived experience and ancestral cultural practices of each Knowledge Holder. Through protocol these ceremonial circles provided place and space where practitioners elaborated on how their cultural and traditional practices are needed to support and benefit the healing in First Nations' individuals and families. AFNCI project outputs include the Collaborative Partnership Framework and Knowledge Mobilization Plan for Oncology provider education and training. Through consensus four key knowledge concepts emerged (relationship, family as medicine, culture as prevention, and Indigenous language) as foundational curriculum supports. Project outcomes advanced the availability of cultural collaborative processes and mechanisms in cancer care service and delivery, to lead and support health system improvements within oncology and care providers to support First Nations patients, families and communities. This project demonstrated ancestral knowledge practices and customs where knowledge, healing and process is applied within a research environment while respecting the pace, and volition of participants, and including connections with spirit to support the true essence of reflective collaboration.

LAY SUMMARY

By engaging in First Nation cultural collaborative processes, it has the potential to transform current oncology care landscapes and practice as captured in the development and application of the Collaborative Partnership Framework and the Knowledge Mobilization Plan. Knowledge Holders/Practitioners must guide and be the key collaborators in cancer care activities that affect and influence the health and wellbeing of FN families and communities. Engagement sessions initiated through inherent First Nation protocols create a conceptual place where storied experiences provide invaluable insight and contribute to knowledge transfer and Knowledge mobilization.

Concurrent: A6-Indigenous Knowledge and Methodologies in Cancer Research

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Stephen AB

Integrating Traditional Knowledge and Western Science: Advancing Cancer Prevention in Indigenous Communities

Presenter: Carol Crowe, Indigenous Visions Inc

ABSTRACT_ID#3249

ABSTRACT

This oral presentation will be shared from an Indigenous perspective, weaving together traditional knowledge and Western science to address environmental health and cancer prevention in Indigenous communities. Since 2006, my work has focused on building capacity among First Nation, Métis, and Inuit peoples across Canada by training over 650 Indigenous environmental monitors in land use planning and environmental monitoring. This approach integrates scientific methods with the wisdom of Elders and knowledge holders, both in classrooms and on the land. These efforts have enabled communities to better assess local development, identify environmental concerns, and recognize potential carcinogenic risks from past, present, and future activities. A critical concern is the cumulative effects of multiple developments and environmental changes, which impact land, water, traditional food sources, and the health and cultural practices of Indigenous peoples. Climate change compounds these effects, threatening the availability and safety of traditional foods and increasing exposure to environmental hazards. Real-world case studies, including preventable exposures to arsenic and asbestos, highlight the urgent need for communications between environmental monitors and health centres. The presentation will discuss complexities of encouraging a return to traditional foods for cancer prevention, emphasizing the need to consult local knowledge holders and conduct appropriate testing before dietary recommendations. Ultimately, this presentation advocates for a collaborative, community-driven approach that validates traditional foods' safety and ensures cancer prevention strategies are grounded in Indigenous knowledge and scientific analysis.

LAY SUMMARY

Combining Knowledge for Stronger Communities: By training Indigenous environmental monitors and bringing together traditional knowledge from Elders with Western science, we help Indigenous communities better understand and address environmental risks that could lead to cancer. **Recognizing Hidden Risks:** Many environmental threats—like arsenic and asbestos—can go unnoticed and increase cancer risk. It's important to create better ways for environmental monitors to share these concerns with local health centres so that action can be taken sooner. **Cumulative Impacts and Climate Change Matter:** The combined effects of development and climate change can make traditional foods and activities less safe. Before recommending a return to traditional diets for health, we need to check with local knowledge holders and test for possible contamination to protect community health.

Concurrent: A6-Indigenous Knowledge and Methodologies in Cancer Research

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Stephen AB

Learning Through Evaluation: Insights from Culturally Grounded Tobacco Reduction Programs for Métis Albertans

Presenter: Kyla Hachey, Rochelle Griffiths, and Milissa Kilian, Otipemisiwak Métis Government

Other authors: Reagan Bartel, Ashton James, Kevin Cunningham, Sharlene Wolbeck Minke, Birgitta Larsson

ABSTRACT_ID#3007

ABSTRACT

Background The Otipemisiwak Métis Government of the Métis Nation within Alberta (MNA) Tobacco Reduction Program is a multidimensional health promotion initiative supporting Métis Albertans in reducing commercial tobacco use. The program includes the following components: Virtual and Land-based QuitCore Group Programs, Nicotine Replacement Therapy (NRT) Program, ChangeCore Peer Support Group, and Quit Retreats. **Methods** A mixed-methods, utilization-focused evaluation grounded in the Principles of Ethical Métis Research was completed. Multiple methods were used to gather information, including interviews with MNA staff, reviewing program records, and collecting feedback from participants through surveys (process and completion), and sharing circles. These qualitative and quantitative methods created multiple opportunities for program participants to share their experiences. **Results** Findings highlight the integral role of Métis-specific elements in making participants feel culturally safe and connected. Across all feedback sources, participants emphasized the importance of Métis-only programs. Being with others who shared similar tobacco experiences and cultural values created a strong sense of belonging. Participants reported gaining knowledge and skills, and experienced shifts in their attitudes towards and relationships with tobacco. **Conclusion** This evaluation affirmed the importance of culturally relevant, holistic, and self-determined approaches to commercial tobacco reduction. Key learnings will guide the expansion of this program to include youth-focused initiatives, self-guided options, and improved promotional materials to support continued success.

LAY SUMMARY

Métis Albertans participated in all program components. To-date, 165 Métis Albertans have graduated from our QuitCore Group Program. Of these, 24 graduates have joined our ChangeCore Peer Support Group and 42 graduates have attended a QuitRetreat. Additionally, 108 Métis Albertans have completed our NRT Program. Program participants felt culturally safe and connected. Program participants indicated that it was important to them to be part of a Métis-only program. They explained that being around people who have had similar experiences fostered a sense of mutual trust and support and helped them to not feel alone. In particular, the cultural elements of this program connected participants to their Métis identity and created a non-judgement setting where participants felt free to be who they really are. Program participants felt better equipped for their tobacco reduction journey. For example, graduates from our group program unanimously reported that they knew a lot more about the differences between traditional and ceremonial tobacco use, how nicotine dependence affects their body, and how their lifestyle can help them to reduce their tobacco use.

Concurrent: A6-Indigenous Knowledge and Methodologies in Cancer Research

Sunday, November 2, 2025, 2:30:00 PM - Hyatt Stephen AB

Indigenous Cancer Research in Canada

Presenter: Peter Hutchinson, University of British Columbia

Other authors: Amanda Fantini, Angeline Letendre, Brenda Elias

ABSTRACT_ID#2715

ABSTRACT

Historically, First Nations, Inuit, and Métis populations in Canada have experienced lower cancer incidence rates. However, recent trends indicate a concerning rise in cancer rates among these populations. In response, this presentation aims to review and synthesize cancer research involving Indigenous populations in Canada through a scoping review covering the period from 2012 to 2025. The review uses targeted search terms to identify studies related to Indigenous identity, Canadian geography, and cancer research. Findings reveal that the majority of the literature addresses cancer care in a general sense and adopts a pan-Indigenous perspective rather than focusing on the distinct experiences and needs of First Nations, Inuit, or Métis communities. The second most common area of research pertains to cancer screening. This review highlights a critical gap in distinction-based research. Pan-Indigenous approaches, while once common, are recognized as inadequate in addressing the cultural, historical, and healthcare contexts of each Indigenous group. Future cancer research must move toward distinction-based methodologies that reflect the specific identities and needs of First Nations, Inuit, and Métis peoples. Furthermore, there is a pressing need to broaden the scope of research beyond cancer care and screening to generate the evidence necessary to inform effective, culturally appropriate policy and practice.

LAY SUMMARY

Rising Cancer Rates Among Indigenous Populations: Although historically lower, cancer rates among First Nations, Inuit, and Métis populations in Canada are increasing, prompting a need for focused research.

Predominance of Pan-Indigenous Research: Most existing studies take a pan-Indigenous approach and emphasize general cancer care, with limited distinction-based research specific to First Nations, Inuit, or Métis communities.

Need for Distinction-Based, Expanded Research: Future research must adopt a people-specific, distinction-based approach and broaden its scope to include diverse cancer-related topics to better inform policy and practice.

Concurrent: B1-Molecular & Cellular Biology

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 4/6

Transcriptional and functional landscape of circular RNA in Cancer

Presenter: Housheng Hansen He, Princess Margaret Cancer Centre, University Health Network

Other authors: Peter Hyunwuk Her, Fraser Soares, Tiantian Li, Weihui Li, Ziwei Huang, Mona Teng, Xin Xu, Benjamin Haibe-Kains, Jie Ming

ABSTRACT_ID#3169

ABSTRACT

Circular RNAs (circRNAs) are a widespread and functionally diverse class of noncoding RNAs with tissue-specific expression and emerging roles in cancer. Using ultra-deep total RNA-seq of 500 patient tumors, we uncovered extensive circRNA production, averaging over 7,000 circRNAs per tumor, correlated with disease progression (1-4). A genome-wide functional screen targeting 9,688 clinically relevant circRNAs across seven major cancer types identified ~14% as essential for cell proliferation, with most acting independently of their linear counterparts (2). While a subset of circRNAs serves as shared regulators across cancer types, the majority exhibit cancer type-specific functions. In prostate cancer, circCSNK1G3 promotes growth via miR-181 interaction, whereas in breast cancer, circNSD1 acts as a tumor suppressor by inhibiting autophagy and oxidative stress through SQSTM1/p62 (3). In neuroendocrine prostate cancer (NEPC) and small cell lung cancer (SCLC), circRMST functions as a lineage regulator, essential for ASCL1 expression and tumor growth via interactions with SOX2 and NKX2-1, and is detectable in liquid biopsies of NEPC and SCLC patients (4). These findings underscore the critical and distinct roles of circRNAs in tumorigenesis and support the adoption of total RNA-seq and functional genomics to map circRNA-regulatory networks. We further provide FunCirc, an online resource cataloging functional oncogenic circRNAs across cancer types to facilitate biomarker and therapeutic discovery. 1. Chen et al. Cell. 2019. 176 (4), 831-843. e22 2. Her et al. bioRxiv, 2025.03. 24.645016 3. Li et al. bioRxiv, 2025.03. 24.645059 4. Teng et al. Cancer Cell. 2025. 43 (5), 891-904. e10

LAY SUMMARY

Circular RNAs are widely expressed and play key roles in cancer growth and progression. By analyzing over 500 patient tumors, we found that many circular RNAs (or "circRNAs") help cancer cells grow. Importantly, these circRNAs often act independently of their related linear genes, highlighting new layers of gene regulation. Some circRNAs have cancer type-specific or shared roles. While certain circRNAs function across multiple cancer types, others are uniquely important in specific cancers. For example, circCSNK1G3 supports prostate cancer growth, circNSD1 suppresses tumor growth in breast cancer by controlling stress response, and circRMST drives neuroendocrine cancer development in both prostate and lung tumors. Our research provides new tools for cancer diagnosis and treatment. We created a public database called FunCirc that catalogs functional circRNAs across cancers. Some circRNAs, like circRMST, can even be detected in liquid biopsy samples, suggesting their potential as non-invasive cancer biomarkers.

Concurrent: B1-Molecular & Cellular Biology

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 4/6

Targeting resistance to cancer therapy through translational control of metabolism

Presenter: Laura Hulea, Université de Montréal

Other authors: Victor Gife, Alejandro Schcolnik-Cabrera, Anavasadat Sadr Hashemi Nejad, Katie Fooks, Meriem Takdenti, Zaynab Nouhi, Tommy Alain, Martin Roffe, Jerry Pelletier, Ivan Topisirovic, François Mercier

ABSTRACT_ID#3257

ABSTRACT

Unlike healthy cells, cancer cells harbor dysregulated signaling pathways that remodel mRNA translation and metabolism to support neoplastic growth and cancer progression, including therapy resistance. In particular, translational programs mediated by the mTORC1/4E-BP/eIF4F axis were shown to support resistance to therapy in various cancer models, in part by allowing cellular metabolic plasticity. We found that targeting the translation initiation complex eIF4F using inhibitors of the eIF4A subunit (eIF4Ai) is very effective in several models of resistance to therapy, in vitro and in vivo. These include BRAF inhibitor-resistant melanoma cells and cytarabine-resistant AML cells. Moreover, in an FLT3-mutated PDX model of AML, the eIF4Ai-venetoclax combination eradicated tumor growth, which was maintained at 13 weeks post treatment cessation. Mechanistically, translational targets of mTORC1/eIF4F, encoding for cell cycle and pro-survival proteins (BCL2, MCL1, BCL-XL), are downregulated by eIF4Ai. Metabolic characterization of these models treated with eIF4Ai shows strong metabolic rewiring, particularly for glycolytic and TCA cycle intermediates and derivative metabolites, a strong reduction in bioenergetics, and an increase in ROS. This was paralleled by a decrease in the translation of metabolic genes, such as those involved in the electron transport chain, mitochondrial membrane transport, or glutamine metabolism. Importantly, eIF4Ais are well tolerated in vivo, with minimal effects on normal myelopoiesis. In conclusion, direct inhibition of translation represents an appealing therapeutic strategy for clinical cases of therapy resistance that are dependent on mTORC1/eIF4F. This is of interest as several translation inhibitors are being developed, with some being currently tested in phase 1/2 clinical trials.

LAY SUMMARY

Drug-resistant cancers survive by ramping up the cell's "protein-making factory", using a control switch called mTOR-eIF4F that fuels growth and energy needs. We blocked one vital part in that switch—the eIF4A protein—to reduce protein-making capabilities. Stopping eIF4A reduced the growth of melanoma and leukemia tumors that no longer responded to standard drugs. In a patient-derived acute myeloid leukemia model, combining the eIF4A inhibitor with venetoclax, a standard of care for leukemic patients, not only erased tumors but mice also remained tumor-free for over three months after the end of treatment. Importantly, reducing eIF4A function leaves healthy blood-forming cells unharmed. Because several eIF4A-targeting medicines are already in early-phase clinical trials, our results offer a realistic new route to beat treatment resistance and give patients longer-lasting responses.

Concurrent: B1-Molecular & Cellular Biology

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 4/6

Role of Hippo Signaling Effectors Yap & Taz in Gastric Metaplasia and Carcinogenesis

Presenter: Zhaoping Ju, McGill University

Other authors: Jacqueline Sung, Mina Farag, Pierre-Olivier Fiset, Lorenzo Ferri, Alex Gregorieff

ABSTRACT_ID#2608

ABSTRACT

Background: Gastric cancer (GC) is a leading cause of cancer-related deaths worldwide. Different metaplastic lesions are associated with GC including spasmolytic polypeptide-expressing metaplasia (SPEM), characterized by oxyntic atrophy and the appearance of proliferative mucosecretory cells. This study aimed to investigate the role of Hippo signaling effectors, Yap and Taz, in driving gastric metaplasia and carcinogenesis. Methods: Yap fl/fl; Taz fl/fl; Clusterin-CreERT2 (CluYap/Taz) and Yap fl/fl; Taz fl/fl; Pepsinogen C-CreERT2 (PgcYap/Taz) mice were treated with high-dose tamoxifen (HDT) to induce Yap/Taz deletion and transient SPEM in the gastric epithelium. Phenotypic changes in mice, gastric organoids (GO) and human GC biopsies were assessed using immunofluorescence and immunohistochemistry.. Result: Deletion of Yap/Taz did not impair initiation of SPEM but instead led to a defect in SPEM resolution. In PgcYap/Taz mice, SPEM persisted for up to a year post-HDT and was accompanied by chronic B cell and macrophage infiltration. Mice with compound deletion of Yap, Taz and Trp53, developed diffused-type gastric cancer within 8-month post-HDT, characterized by loss of E-cadherin. In contrast to engineered Apc-/-;Trp53-/-; GOs, diffused-type tumor GOs grew as unpolarized cellular aggregates and were insensitive to Yap modulation. In concordance, human GC tissues revealed low YAP in diffused-type and high YAP in intestinal-type gastric cancer, supporting a role for YAP in subtype-specific gastric carcinogenesis. Conclusions: Our findings showed Yap and Taz are essential regulators of gastric epithelial regeneration following metaplastic injury. Their loss results in sustained metaplasia, chronic inflammation, and, when combined with Trp53 deletion, drives the development of diffuse-type gastric cancer.

LAY SUMMARY

- YAP and TAZ are essential for gastric epithelial recovery after injury. Their loss in mouse models leads to persistent metaplasia, disrupted glandular architecture, and chronic immune cell infiltration — features that may predispose the stomach to cancer.
- YAP/TAZ inactivation cooperates with Trp53 loss to promote diffuse-type gastric cancer. In contrast to prior studies documenting pro-oncogenic role for YAP/TAZ in gastro-intestinal tumorigenesis, our findings imply tumor suppressive actions of YAP/TAZ in the gastric epithelium.
- Human gastric cancer samples reveal subtype-specific YAP expression. Diffuse-type tumors consistently show low YAP levels, while intestinal-type tumors retain high YAP, suggesting that YAP may help distinguish cancer subtypes and serve as a potential diagnostic or therapeutic biomarker.

Concurrent: B1-Molecular & Cellular Biology

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 4/6

G0S2 Protein Regulates Colon Inflammation and Colorectal Cancer Through Production of Branched-chain Fatty Acids

Presenter: Yilin Wang, McGill University

Other authors: Rachid Zagani, Jose Teodoro, Owen Chen, Isabelle Gamache, Mariana De Sa Tavares Russo, Daina Avizonis

ABSTRACT_ID#2297

ABSTRACT

Branched-chain fatty acid esters of hydroxy fatty acids (FAHFAs) are a novel class of endogenous anti-inflammatory lipids. It has recently been discovered that adipose triglyceride lipase (ATGL), a key enzyme in triglyceride lipolysis, catalyzes the synthesis of FAHFAs. The G0S2 protein (G0/G1 switch gene 2) functions as an endogenous inhibitor of triglyceride breakdown by binding to ATGL. However, how the regulation of FAHFA production by the ATGL/G0S2 axis affects inflammation and cancer progression has never been addressed. In this study, we show that G0S2 can inhibit FAHFA production in vitro. Moreover, the pro-inflammatory cytokine, tumor necrosis factor- α (TNF- α) is a potent inducer of G0S2 expression, and it suppresses FAHFA production. We have previously derived a G0s2 knockout (KO) mouse and here we show that these animals exhibit elevated FAHFA levels. To assess the impact of FAHFAs on inflammation, we treated the G0s2 KO and wild-type controls with DSS to induce experimental colitis and show that loss of G0s2 conferred resistance to colitis. Additionally, G0s2 KO mice showed reduced susceptibility to AOM/DSS-induced tumorigenesis and ApcMin/+ -driven intestinal cancer, likely due to reduced pro-inflammatory cytokine production. Our data suggest that the ATGL/G0S2 axis can affect colon inflammation and colorectal cancer through ATGL-mediated FAHFA production.

LAY SUMMARY

Inflammation increases the expression of G0S2 in the colon in both mice and humans. G0S2 suppresses ATGL, reducing anti-inflammatory branched-chain fatty acid esters of hydroxy fatty acids (FAHFAs) production. Loss of G0S2 lowers colon inflammation and intestinal and colonic tumour formation.

Concurrent: B2-Clinical Research & Trials

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 1/2/3

Lipid metabolism reprogramming in the Safety cohort of the CHLOE pancreas trial: Cholesterol blockade in metastatic pancreatic cancer NCT04862260

Presenter: Anne Gangloff, Université Laval

Other authors: France-Hélène Joncas, Anne-Marie Carreau, Francine Aubin, Frederic Calon, Nabil G Seidah, Louis Lacombe, Jonatan Blais

ABSTRACT_ID#2671

ABSTRACT

Rationale. Pancreatic ductal adenocarcinoma (PDAC) requires substantial lipid supplies to support its progression. By combining safe and affordable cholesterol-lowering drugs (CLDs), low-density lipoproteins (LDLs), which are the primary lipid source for PDAC, can be eliminated from circulation. In this early-phase clinical trial, we evaluate the feasibility and acceptability of adding a cholesterol blockade to FOLFIRINOX. **Methods.** The initial safety cohort comprised 3 participants who received CLDs (atorvastatin 40 mg die + ezetimibe 10 mg bid + evolocumab 420 mg every 4 weeks) for a 7-day run-in period, followed by co-administration with FOLFIRINOX. Longitudinal blood samples were collected before and during treatment to assess a wide range of biological parameters. Hyperlipidemic factors, phytosterols, lipoproteins, and lipids were evaluated using ELISA, mass spectrometry, and NMR spectroscopy (Nightingale Health), respectively. **Results.** Near-zero plasma LDL-cholesterol was achieved in all 3 participants (<0.5 mmol/L). No severe adverse events (SAEs) related to CLDs were reported, confirming feasibility. Participants expressed that the intervention was acceptable, as evaluated by Patient-Partners. No patient progressed while on the cholesterol blockade. Total plasma PCSK9 increased significantly (>5 folds), retained by evolocumab. ANGPTL3 and Apo CIII decreased (0.5-fold) at the onset of the blockade. CLDs modified VLDL composition towards triglycerides. Glutamine and pyruvate levels rose (2-fold), whereas docosahexaenoic acid (DHA) levels decreased during the blockade, aligning with a detrimental environment for PDAC. **Conclusion.** In the safety cohort of NCT04862260, it was feasible and acceptable to add a cholesterol blockade on top of FOLFIRINOX. Patients did not progress while on the blockade.

LAY SUMMARY

- By combining cholesterol-lowering medications, it is possible to collapse LDL levels, depriving cancer cells of cholesterol, which is essential for pancreatic cancer progression.
- The addition of a cholesterol blockade to FOLFIRINOX in patients with metastatic pancreatic cancer was found to be feasible (defined as <2 severe adverse events) and acceptable (based on anonymized comments of participants collected by patient partners).
- None of the three patients in the safety cohort progressed while undergoing cholesterol blockade.

Concurrent: B2-Clinical Research & Trials

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 1/2/3

Building the Canadian-Led Immunotherapies in Cancer (CLIC) Network: Impact, Opportunities, and Lessons Learned

Presenter: Kevin Hay, University of Calgary

Other authors: Natasha Kekre, Robert Holt, Brad Nelson, John Bell, Jennifer Quizi, Miruna Balasundaram, Julie Nielsen

ABSTRACT_ID#3310

ABSTRACT

The Canadian-Led Immunotherapies in Cancer (CLIC) network was formed in 2017 with a vision to develop a Canadian academic model for cell therapy manufacturing and research, aiming to build expertise and infrastructure, foster innovation, and improve access to groundbreaking therapies for cancer patients in Canada. The CLIC program is precedent setting for its achievements in creating a fully made-in-Canada CAR-T cell translational pipeline for point-of-care compatible manufacturing and clinical trials. The success of this model is exemplified by the treatment of approximately 100 patients with relapsed/refractory leukemia or lymphoma using Canadian-made CAR-T cells targeting CD19 (Phase 1/2 CLIC-01 trial) or CD22 (Phase 1 CLIC-02 trial). Clinical outcomes, biobank resources, and correlative analyses for these trials will be discussed. Along with this progress, lessons learned are important to share with our community. Building a CAR-T cell manufacturing network and aligning different academic centres requires significant time and resources, well-defined expectations and onboarding processes for new sites, and extensive responsibility and workload for the lead manufacturing site. Looking to the future, the CLIC network is motivated to reach patients beyond clinical trials, particularly small patient populations where there will be an unmet need – but the pathway to market authorization will be a challenge from both a funding and regulatory perspective. These issues are not unique to Canada, but rather represent universal challenges. Process harmonization, data sharing, and clear regulatory expectations for hospital-based manufacturing will be critical for bringing advanced cancer immunotherapies to more patients.

LAY SUMMARY

Canada is building its own cancer-fighting cell therapies: The CLIC network has created a Canadian system to develop and deliver advanced cancer treatments (like CAR-T cell therapy) entirely within the country — from lab research to patient care.

Real impact on patients already: CLIC's clinical trials have already treated about 100 blood cancer patients using Canadian-made CAR-T cell therapies.

Big progress, but big challenges and lessons learned: Expanding access to made-in-Canada CAR-T cell therapy takes significant time, funding, and coordination between research centres. Making these treatments more widely available to cancer patients will also require clearer regulations and better data sharing — challenges faced worldwide, not just in Canada.

Concurrent: B2-Clinical Research & Trials

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 1/2/3

Standard Chemo-Immunotherapy versus Real-World Protocols: Evaluating Outcomes in Triple-Negative Breast Cancer Neoadjuvant chemo/immunotherapy

Presenter: Fatima Nadeem, Schulich School of Medicine & Dentistry, University of Western Ontario

Other authors: Laurice Arayan, Sneha Mukherjee, Rong Luo, Caroline Hamm, Swati Kulkarni

ABSTRACT_ID#2853

ABSTRACT

Background: Triple-negative breast cancer (TNBC) is an aggressive subtype that often requires neoadjuvant systemic therapy. The introduction of pembrolizumab in the neoadjuvant setting and capecitabine in the adjuvant setting has improved outcomes. Cancer Care Ontario has approved several chemo-immunotherapy regimens incorporating pembrolizumab. This study compares tolerance, pathological complete response (pCR) rates, and survival outcomes among patients receiving various immunotherapy-chemotherapy combinations. **Methods:** This retrospective review included patients aged ≥ 18 years, diagnosed with TNBC, who received neoadjuvant and/or adjuvant systemic therapy from January 1, 2021, to December 31, 2024, at Windsor Regional Cancer Program. Demographic and clinical characteristics and pCR rates were summarized using descriptive statistics. Kaplan-Meier survival analysis will compare event-free survival (EFS), overall survival (OS), and toxicity profiles against the KEYNOTE-522 regimen. **Results:** Preliminary analysis includes 97 of 120 patients. The cohort was exclusively female, predominantly postmenopausal, and most commonly presented with cT2N0 tumors. High-grade tumors (Grade 3) were predominant. Nearly half underwent genetic testing, and 45% had a family history of breast cancer. Most underwent breast-conserving surgery and received radiation and systemic therapy. pCR was achieved in approximately one-third of patients. Four patients received non-KEYNOTE-522 regimens. Despite aggressive treatment, eight patients experienced cancer-related deaths and one relapse. **Conclusions:** Ongoing follow-up is critical to assess long-term OS and EFS. Larger sample sizes are needed, particularly to evaluate outcomes with non-standard regimens. These early findings offer insight into real-world TNBC management beyond clinical trial settings.

LAY SUMMARY

Some breast cancer patients respond well to new combinations of chemotherapy and immunotherapy. In our study of people with a difficult-to-treat form of breast cancer (called triple-negative breast cancer), about one-third had no signs of cancer left after treatment. Most patients received a standard treatment used in clinical trials, but some were treated with different combinations. We found that even though these treatments were aggressive, there were still cases of cancer coming back or leading to death. This shows that more research is needed to understand which treatment combinations work best in real life. We need more patients and longer follow-up to get stronger answers. This early research helps guide future studies and may improve how we treat this type of breast cancer in the future.

Concurrent: B2-Clinical Research & Trials

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 1/2/3

Development of Patient Priorities in Cancer Research

Presenter: Judy Needham, Canadian Cancer Clinical Trials Network (3CTN)

Other authors: Carol Gordon, Martina Wood, Emi Bossio, Janet Dancey

ABSTRACT_ID#2973

ABSTRACT

Introduction Over the past decade, patient engagement in cancer research has made significant strides, particularly at an individual research and clinical trials level. However, patient voices remain underrepresented at the strategic planning level. To address this, the Canadian Cancer Trials Group (CCTG) hosted a patient priorities setting workshop to help inform strategic planning. **Methods** Patients and caregivers from across Canada—reflecting diverse cancers, ages, and backgrounds—were invited to participate. Pre-workshop, they completed a survey identifying issues experienced in their lived experiences across three areas: Journey, Treatment, and Research. During the workshop, participants collaborated in groups aligned with these themes and issues to identify and rank associated research priorities. Post-workshop, a working group organized the findings thematically into a final ranked list and report. **Results** The report highlights 6 priorities: Innovative Research, Biomarker Research, Use of Technology, Psychological and Holistic Oncology, Early Detection, Treatment Optimization, with overarching principles and details in each. It now informs CCTG's strategic planning and is being reviewed by other research institutions, funders, and patient advocacy groups for planning and research competition funding. **Conclusions** This initiative underscores patient engagement as a cornerstone of impactful cancer research. By incorporating diverse patient perspectives into the strategic planning process, CCTG has set a precedent for inclusivity and collaboration. The methodology employed not only amplifies the patient voice but also ensures that research priorities resonate with the real-world experiences of patients. As other organizations look to emulate this approach, patient-informed research strategies will continue to shape more responsive and effective cancer care.

LAY SUMMARY

Although patient engagement in Canada has developed exponentially at levels of individual research projects and clinical trials in Canada, opportunities exist to engage the patient voice more fully at the strategic planning level. Among all the voices in the cancer research ecosystem, the patient voice is one grounded in lived experience. This Patient Priorities Forum/Workshop gave voice to the realities, challenges, and aspirations of those who must try to navigate not only their disease, but also the research intended to bring them hope. It is by listening to these voices that we can truly identify and use the gaps experienced to augment crafting research strategies and agendas that are not only innovative, but meaningful and human-centred. And patient-informed research in turn effects effects patient-centred cancer care. The structured methodology employed accomplished this. Patients and caregivers first identified key challenges from their personal cancer journeys through a structured survey. Insights were then brought into a facilitated workshop, where participants collaborated to transform challenges into actionable research priorities and potential solutions. Results were consolidated and organized thematically into a final report that is disease site agnostic and encompasses all cancers. The priorities are accompanied by overarching principles identified through the workshop.

Concurrent: B3- Patient Experience

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 5/7/9

Five years of a province-wide Compassionate Communities initiative: impact on public awareness of palliative care and advance care planning

Presenter: Patricia Biondo, University of Calgary

Other authors: Mary-Ann Shantz, Tyler Hamil, Maureen Douglas, Keltie Watson, Ellen Mi, Louise Kashuba

ABSTRACT_ID#2556

ABSTRACT

Background: Despite the American Society of Clinical Oncology recommendation that all patients with advanced cancer receive early palliative care, many still receive palliative care too late or not at all. Improving public awareness of advance care planning and palliative care is essential to help people live well with advanced cancer and access palliative care, which has demonstrated benefits for patients and health systems. This presentation will describe a five-year, multi-sectoral Compassionate Communities initiative to increase public understanding of advance care planning and palliative care in one Canadian province. Approach: Extensive multi-sector stakeholder engagement activities informed the work and identified community partners. Five public-facing resources were developed and/or adapted that encourage conversations about death and dying, improve understanding of palliative care, build community support for people living with serious illness, and promote advance care planning. The resources were widely disseminated and implemented in collaboration with community partners. Results: Our process measures show we've reached 725,000+ people and created change in six of eight Healthy End of Life Program evaluation framework domains: People, Programs, Participation, Practice & Practitioners, Place, and Partnerships. Outcome measures show improved understanding of and attitudes toward palliative care, advance care planning, and Compassionate Communities. Our 2024 provincial poll showed 13 and 15% increases in awareness of advance care planning and palliative care, respectively, over the last decade. Conclusion: Our Compassionate Communities initiative has successfully raised awareness of palliative care and advance care planning and provides a framework for others interested in multisectoral collaboration that bridges health and community services.

LAY SUMMARY

Improving the public's awareness and understanding of palliative care and advance care planning is needed to support more people to access and accept palliative care when offered, and to participate in advance care planning. Our collaboration with community organizations from several sectors (e.g., hospice and palliative care societies; senior-serving organizations; faith and cultural communities; libraries; and legal, financial and business professionals) was instrumental to the success of this initiative. As evaluation of Compassionate Communities initiatives is a relatively new area of study, our evaluation framework and measures may be helpful to others evaluating such initiatives.

Concurrent: B3- Patient Experience

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 5/7/9

Examining mental health care utilization in individuals with colorectal cancer: population-based cohort studies

Presenter: Vicki Cheng, University of British Columbia

Other authors: Eric C. Sayre, Vienna Cheng, Jonathan M. Loree, Sharlene Gill, Rachel A. Murphy, Mary A. De Vera

ABSTRACT_ID#2922

ABSTRACT

Background: Individuals diagnosed with colorectal cancer (CRC) have an increased risk of developing mental disorders; however, mental healthcare utilization remains underexplored. We examined the use of mental health services and the receipt of minimally adequate treatment for anxiety and/or depression among individuals with and without CRC. **Methods:** Using administrative health data from British Columbia, Canada, we created matched cohorts of individuals with and without CRC (1:1 ratio) based on age, sex, and incident mental disorder(s) (i.e., occurring after CRC diagnosis/matched date). Primary outcomes included minimally adequate antidepressant pharmacotherapy (≥ 84 days' supply) and psychological treatment (≥ 4 services). **Results:** Among individuals diagnosed with CRC, 1,462 had incident anxiety (59.2% females, mean age 64.6), and 4,640 had incident depression (51.2% females, mean age 66.3). Within the first year following CRC diagnosis, approximately one in four individuals were diagnosed with either anxiety (23.4%) and/or depression (23.2%). Individuals with CRC were significantly less likely to receive minimally adequate antidepressant therapy (36.2%) or psychological care (15.9%) for anxiety, compared to matched individuals without CRC (pharmacotherapy adjusted OR [aOR] 0.74; 95% CI 0.61, 0.88; psychological treatment aOR 0.74; 95% CI 0.58, 0.95). A similar pattern was observed for depression (pharmacotherapy aOR 0.81; 95% CI 0.74, 0.90). Among those with CRC, mental healthcare utilization continued for up to 10 years following diagnosis of a mental disorder. **Conclusion:** Compared to those without CRC, individuals with CRC receive less mental health treatment for anxiety and depression. Our findings highlight the need for ongoing, integrated mental healthcare throughout and beyond CRC.

LAY SUMMARY

- People with colorectal cancer (CRC) are more likely to experience anxiety or depression, but we are unsure about their mental health care use after these diagnoses. This study helps fill that gap by looking at how people with CRC use mental health treatments after being diagnosed with anxiety or depression.
- We found that people with CRC were less likely to receive adequate antidepressant medication or psychological care compared to people without CRC. This means they may not be getting the mental health care they need, even though they are at higher risk.
- Mental health service use continued for up to 10 years after anxiety or depression was diagnosed. These findings show how important it is for mental health care to be available not just during CRC treatment, but also long after—beyond diagnosis and treatment. Health systems should prioritize long-term mental health care as part of overall CRC care.

Concurrent: B3- Patient Experience

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 5/7/9

The experience of breast cancer treatment among adults with intellectual or developmental disabilities: A convergent mixed methods study

Presenter: Rebecca Hansford, Queen's University

Other authors: Brooke Wilson, Rebecca Griffiths, Alyson Mahar

ABSTRACT_ID#2620

ABSTRACT

Introduction: This study explored breast cancer treatment among adults with intellectual or developmental disabilities (IDD) using a convergent mixed methods study. Methods: Quantitative data included administrative data from Ontario: 1) comparing breast cancer treatment among adults with and without IDD; 2) identifying factors associated with breast cancer treatment among those with IDD. Qualitative data was from interviews with a woman with IDD with breast cancer, her sister, a support worker, and cancer surgeon. We integrated findings at the interpretation level. Joint displays were created, including Forest plots overlayed with supporting quotations. Results: Sandra (pseudonym)'s experience of receiving breast conserving surgery, adjuvant chemotherapy, and radiation was discordant with the experience of many adults with IDD in the population-based data, who were significantly less likely to receive these treatments compared to those without IDD. Established factors such as age and stage at diagnosis were associated with receiving breast cancer treatments at the population level; however, we identified many other factors that influenced Sandra's access to care in our interviews, including strong communication skills, capacity to consent, and opportunities for shared decision-making and advocacy. Discussion: Despite Sandra being younger, diagnosed with early-stage cancer, having strong communication and advocacy skills and experiencing a positive outcome, she encountered obstacles that could have prevented her from receiving treatment. Identifying areas to target for improving patient-centered care for cancer patients with IDD, such as accessible communication tools, is needed.

LAY SUMMARY

We conducted a convergent mixed method study to understand breast cancer treatment experiences for female adults with intellectual and/or developmental disabilities. Female adults with intellectual and/or developmental disabilities were less likely to receive breast conserving surgery, chemotherapy, and radiation compared to those without disabilities. Conversely, the qualitative participant, Sandra, received breast conserving surgery, chemotherapy, and radiation. While Sandra was younger, had early-stage cancer, and had strong communication and advocacy skills, she still faced obstacles that could have prevented her from receiving treatment. Findings from this study could be used to inform strategies to improve patient-centred care for breast cancer patients with intellectual and/or developmental disabilities.

Concurrent: B3- Patient Experience

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 5/7/9

Mise à l'échelle du programme PAROLE-Onco au Québec : le rôle central d'une communauté de pratique interétablissements

Presenter: Monica Iliescu Nelea, Centre hospitalier de l'Université de Montréal (CHUM)

Other authors: Marie-Pascale Pomey, Cécile Vialaron, Marie-Andrée Coté, Carole Lesperance

ABSTRACT_ID#3030

ABSTRACT

Contexte. Mis en place au Québec depuis 2017 dans le cadre d'une recherche interventionnelle participative, le programme PAROLE-Onco vise à intégrer des patients accompagnateurs (PA) dans les équipes de soins en oncologie. Ces PA, ayant eux-mêmes vécu un cancer, offrent un soutien émotionnel, informationnel, éducationnel et de navigation aux patients dès le début de leur trajectoire. Devant les résultats positifs, une mise à l'échelle du programme a été entreprise dans sept établissements de la province. **Objectif.** Cette recherche vise à documenter comment les établissements ont collaboré pour mettre à l'échelle PAROLE-Onco, en s'apportant un soutien durable et adapté aux différents contextes cliniques. **Méthode.** Une étude de cas multiples longitudinaux combinant méthodes qualitatives et quantitatives a été menée à trois niveaux d'analyse : programme, établissement et équipe clinique. Trente-cinq entretiens ont été réalisés auprès de professionnels de la santé, gestionnaires et PA entre avril et août 2024. **Résultats.** Pour soutenir l'expansion du programme, les établissements ont mis en place une communauté de pratique interétablissements regroupant PA, gestionnaires et responsables du partenariat. Cette structure a permis le partage d'outils, de stratégies et de solutions concrètes entre équipes expérimentées et nouvelles. Des ateliers collaboratifs et activités d'accompagnement ont facilité l'adaptation aux réalités locales. **Conclusion.** La communauté de pratique a agi comme levier pour une mise à l'échelle réussie, en tenant compte des contextes d'implantation dès le départ. Elle a renforcé les capacités collectives et assuré une mise en œuvre ancrée dans chaque établissement.

LAY SUMMARY

Un programme innovant pour les personnes atteintes de cancer : PAROLE-Onco intègre des patients accompagnateurs – des personnes ayant elles-mêmes vécu un cancer – dans les équipes de soins. Ils offrent un soutien émotionnel, de l'information, de l'éducation et aident les patients à mieux naviguer dans leur parcours de soins.

Une collaboration entre établissements : Pour que ce programme soit implanté dans plusieurs hôpitaux du Québec, les établissements de santé ont travaillé ensemble. Ils ont mis en place une communauté de pratique interétablissements où patients accompagnateurs, gestionnaires et professionnels échangent outils, défis et solutions.

Une implantation adaptée à chaque milieu : Cette collaboration a permis d'adapter le programme aux réalités locales de chaque hôpital. Grâce à des formations, des ateliers et un accompagnement entre pairs, sept établissements ont réussi à intégrer les patients accompagnateurs dans différentes trajectoires en oncologie.

Concurrent: B4-Screening

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 8

Strategies to improve access to mammograms: A qualitative study with social service providers in New Brunswick

Presenter: David Busolo, University of New Brunswick

Other authors: Cecilia Moreira, Mariia Zaiachuk, Khawla Aldandan, Tracey Rickards, Cathy Holtmann, Robin Urquhart, Sara Edge

ABSTRACT_ID#3336

ABSTRACT

Background: Social service providers (SSPs) in settlement organizations play a critical role in supporting refugees' access to healthcare services (including mammograms). Despite this knowledge, few studies have explored how SSPs perceive and respond to Arabic-speaking refugee women's (ASRW) barriers to accessing mammography in New Brunswick (NB). Understanding the intermediary experiences of SSP and recommendations is critical for developing an effective program to improve access to mammography for these women. Methodology: Using a descriptive qualitative research study approach, sixteen (16) SSPs from three settlement organizations in NB participated in individual semi-structured interviews. The interviews were digitally recorded, transcribed, and analyzed using a thematic analysis approach. Measures were taken to ensure the study was rigorous. Findings: SSPs shared strategies to increase ASRW mammography uptake, which are presented in three (3) themes: 'Increasing access', 'Providing education', and 'Partnering with others.' On the theme of 'Increasing access', SSP shared strategies to overcome transportation, language and communication barriers. On the theme of 'Providing education', SSPs' discourse focused on ways to improve breast cancer, mammography and healthcare system awareness. On the theme of 'Partnering with others,' SSPs stressed upon meaningful collaboration between ASRW, settlement organizations, and community groups.' Discussion: In the wake of poor access to mammography services by ASRW, SSPs' recommendations are critical for an equally focused approach to screening uptake. The findings underscore the need for interdisciplinary and culturally responsive programs. Future work will focus on developing and testing strategies with ASRW and other marginalized populations to improve access to mammograms.

LAY SUMMARY

Social service providers from settlement organizations play a critical role in supporting refugees (such as Arabic-speaking refugee women - ASRW) access healthcare services including mammograms. Few studies have explored how social service providers perceive and respond to the barriers ASRW face in accessing mammograms, particularly in New Brunswick Social service providers recommend collaborative strategies that focus on culturally responsive programs and highlight the importance of engaging Arabic-speaking refugee women, community organizations, and healthcare systems in co-designing solutions.

Concurrent: B4-Screening

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 8

An Assessment of British Columbia's Cancer Screening Programs for First Nations People Living in the Province

Presenter: Nadine Caron, University of British Columbia

ABSTRACT_ID#3188

ABSTRACT

Objectives: This study developed a comprehensive set of performance indicators on British Columbia's breast, cervical and colon cancer screening programs in meeting the needs of First Nations (FN) people living in the province. **Methods:** A population-based retrospective cohort methodology was utilized using linked data from the BC Cancer Screening Program database, BC Statistics Population Estimates, BC Ministry of Health Client Roster, and the 2018 BC First Nations Client File. Indicators developed included cancer screening participation, uptake and retention rates, reflecting screening outcomes recorded between 1992 to 2018. **Results:** For cervical cancer screening, participation was found to be lower among FN women (52.8%) compared to all other women (62.0%). Program retention was also lower in FN women (64.5%) compared to all other women (71.0%). For breast cancer screening, FN women had consistently lower participation rates (30.7%) than all other women (49.3%). Retention among FN women was 28.5% for first-time clients and 58.3% for those previously screened, compared to 46.6% and 75.5% in all other women, respectively. For colon cancer screening, participation and up-to-dateness were lower among FN males (23.2%) and females (20.2%) relative to all other residents. **Conclusion:** Our findings show significant disparity in cancer screening utilization in the province between FN and other residents and highlight the need for targeted interventions and screening program policy changes to ensure equitable cancer outcomes.

LAY SUMMARY

Disparities in cancer screening program utilization exist between First Nations and all other residents in BC Targeted interventions and screening program policy changes are needed

Concurrent: B4-Screening

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 8

Breast cancer screening engagement among women with different ethnic backgrounds in Canada: Evidence from the Canadian Partnership for Tomorrow's Health

Presenter: Maryam Darvishian, University of British Columbia

Other authors: Victoria Krish, Philip Awadalla, Ashley Mah, Jennifer Brooks, Trevor Dummer

ABSTRACT_ID#2812

ABSTRACT

Background Breast cancer screening programs in Canada are effective in reducing breast cancer mortality through early detection. However, the benefits of these programs may not be equally experienced by all eligible individuals. Data on disparities in screening participation across individuals with different ethnic backgrounds remains limited. **Methods** Using multinomial logistic regression, we analyzed self-reported data from five regions of the Canadian Partnership for Tomorrow's Health (CanPath), a longitudinal cohort study, on engagement in screening mammography. Breast cancer screening status was categorized as "never screened," screened >2 years ago", and "screened ≤2 years ago (reference category)". Our models adjusted for potential confounding factors including age, income, education, marital status, smoking status, presence of comorbidities, and last medical check-up. **Results** The study population included 79,986 individuals aged 50-74 years at study enrollment. East Asian, South Asian, and Filipino/Southeast Asian women were more likely to have never been screened or screened > 2 years ago versus screened ≤ 2 years ago compared to White women (OR:1.40; 95% CI 1.12-1.75; OR: 1.71, 95% CI 1.24-2.36; and OR: 1.30, 95% CI 1.00-1.70, respectively). In contrast, compared to White women, Jewish women had a lower likelihood of being never screened' (OR: 0.53, 95% CI 0.37-0.76), indicating a higher rate of recent screening (within the past two years). **Conclusion** Our findings demonstrate significant screening disparities among women with specific ethnic backgrounds, indicating greater need to investigate potential barriers and to plan targeted outreach for increasing screening adherence.

LAY SUMMARY

1. The benefits of screening programs may not be equally experienced by all eligible individuals.
2. Data on disparities in breast cancer screening participation across individuals with different ethnic backgrounds remains limited in Canada.
3. We observed significant screening disparities among women with specific ethnic backgrounds, namely East Asian, South Asian, and Filipino/Southeast Asian women, indicating the importance of targeted outreach for increasing screening adherence.

Concurrent: B4-Screening

Monday, November 3, 2025, 10:00:00 AM - Hyatt Imperial 8

Projected Impact of Lung Cancer Screening on Health Outcomes and Healthcare Resources in Canada

Presenter: Rafael Meza, BC Cancer/Canadian Partnership Against Cancer

Other authors: Zhuolu Sun, Jean Hai Ein Yong, Rayjean J. Hung, Rochelle Garner, Maikol Diasparra, Hawre Jalal, Dori Simeonov

ABSTRACT_ID#2874

ABSTRACT

Introduction: Screening with low-dose computed tomography has been shown to reduce lung cancer mortality. This study aims to assess the resource implications and clinical impact of lung cancer screening with various eligibility criteria in Canada. **Methods:** The analysis used OncoSim-Lung (version 3.6.4.4) to simulate a cohort of individuals born between 1945 and 2005. Screening scenarios varied by screening start age (45, 50, 55 years), stop age (74, 80 years), interval (annual, biennial), and risk threshold (PLCOm2012 1.0% to 2.0% by 0.1% increments). Outcomes were compared to a No Screening scenario. Additional analyses examined the effect of incorporating smoking cessation interventions into the screening program. **Results:** Lung cancer screening resource demands and outcomes vary significantly with screening interval, age range, and risk threshold. Screening interval has the strongest impact on healthcare resources and clinical benefits. Extending screening to age 80 and lowering risk threshold would also lead to better clinical outcomes at the expense of increased need of health care resources. Starting screening earlier has minimal impact, especially when the risk threshold is higher than 1.5%. Including smoking cessation support within screening programs reduced healthcare resource demands and increased clinical benefits, especially in the long run. **Conclusions:** The findings highlight the possibilities for enhancing health benefits and resource allocation in lung cancer screening programs through thoughtful program design. Further investigation into refining screening eligibility criteria considering health equity, optimizing screening intervals based on baseline scan, and incorporating smoking cessation interventions may improve both the effectiveness and sustainability of these programs.

LAY SUMMARY

1. The study assesses the resource implications and clinical impact of lung cancer screening with different eligibility criteria in Canada using OncoSim-Lung.
2. Annual screening, extending screening to age 80, and lowering the risk threshold increase clinical benefits but also raise healthcare resource use. Starting screening earlier (e.g., at 45 or 50) has minimal impact unless the risk threshold is low ($\leq 1.5\%$).
3. Including smoking cessation support in screening programs reduces resource demands and enhances long-term clinical benefits.

Concurrent: B5-Health Economics/Outcomes Research

Monday, November 3, 2025, 10:00:00 AM - Hyatt Doll/Herald

The PaCES Study: Cost effectiveness of an early palliative care intervention in patients with advanced colorectal cancer

Presenter: Philip Akude, University of Calgary

Other authors: Mahmood Zarrabi, Hannah Douglass-Molloy, Anyharan Sinnarajah, Madalene Earp, Andrew Fong, Sharon Wantanabe, Jessica Simon, Patricia Biondo

ABSTRACT_ID#3248

ABSTRACT

Background: This study evaluated the impact of specialist-led palliative care on managing advanced-stage colorectal cancer (CRC). By analyzing healthcare costs and time spent at home, we assessed how palliative interventions influence patient outcomes and system efficiency. **Methods:** We conducted a retrospective cohort study of CRC decedents in Alberta's Calgary and Edmonton Zones from January 1, 2016, to December 31, 2020. The intervention group included Calgary decedents who received care from Palliative Care Specialists; the control group included Edmonton decedents who received usual care. Primary outcomes were healthcare costs and days spent at home in the last 90 days of life. We applied a difference-in-differences (DID) approach using generalized linear models (GLM) for costs and ordinary least squares (OLS) for health outcomes. **Results:** The intervention group saw an average cost reduction of \$4,467.70 per decedent, decreasing from \$33,880.7 to \$31,607.6, while costs rose in the control group. Specialist palliative care was associated with a 16% increase in referrals, a 15% decrease in hospitalization days, and an 18% reduction in days receiving chemotherapy. **Conclusion:** Specialist-led palliative care for CRC patients may offer meaningful cost savings and improved end-of-life outcomes. The intervention was associated with reduced healthcare costs, fewer hospital and chemotherapy days, and enhanced time at home, suggesting its value in advanced cancer care.

LAY SUMMARY

Specialist palliative care lowers end-of-life healthcare costs: Integrating palliative care specialists into the treatment of advanced colorectal cancer reduced healthcare spending by an average of \$4,467 per patient in the final 90 days of life.

Improved care efficiency and patient outcomes: Patients receiving specialist-led palliative care had fewer hospital days and less chemotherapy use, suggesting more appropriate and patient-centered care at the end of life.

Supports earlier access and health system sustainability: The intervention increased palliative care referrals by 16%, demonstrating how structured integration can enhance access while promoting cost-effective, high-value care.

Concurrent: B5-Health Economics/Outcomes Research

Monday, November 3, 2025, 10:00:00 AM - Hyatt Doll/Herald

Examining the association between breast cancer treatments and self-reported sexual dysfunction in Canadian women: A population-based cross-sectional study

Presenter: Patience Nishimagizwe, Dalhousie University

Other authors: Cindy Feng, Jennifer Payne, George Kephart, Robin Urquhart

ABSTRACT_ID#3295

ABSTRACT

Objective: The study aims to estimate the prevalence of self-reported sexual dysfunction among breast cancer survivors in Canada and investigate the association between types of breast cancer treatment received and self-reported sexual dysfunction. **Methodology** This cross-sectional study utilized data from 3,772 breast cancer survivors who participated in the Experiences of Cancer Patients in Transition survey in 2012, a Pan-Canadian population-based survey designed to capture experiences of cancer survivors. The outcome was sexual dysfunction, and the exposures were breast cancer treatment modalities above and beyond surgery, including chemotherapy, radiotherapy, and endocrine therapy. Descriptive statistics were conducted to estimate the prevalence of sexual dysfunction by treatment type. Logistic regression analyses, both unadjusted and adjusted, were used to assess the independent and interactive effects of treatment modalities in relation to sexual dysfunction, and sensitivity analyses were conducted. **Results** Among the 3,772 respondents, Sexual dysfunction was most prevalent among those treated with chemotherapy (48.3%), followed by endocrine therapy (43.2%) and radiotherapy (40%). In adjusted logistic regression models, chemotherapy was most strongly associated with sexual dysfunction (adjusted odds ratio aOR = 1.67; 95% CI: 1.43–1.94). Significant associations were also observed for endocrine therapy (aOR = 1.31; 95% CI: 1.12–1.53) and radiotherapy (aOR = 1.27; 95% CI: 1.06–1.51). The two-way and three-way interactions between breast cancer treatments were not statistically significant. Sensitivity analyses supported the robustness of these findings. **Conclusion** This study highlights a high prevalence of self-reported sexual dysfunction among breast cancer survivors in Canada, particularly among those who received chemotherapy, endocrine therapy, or radiotherapy.

LAY SUMMARY

Sexual dysfunction is common after breast cancer treatment, where almost half of breast cancer survivors who received chemotherapy reported experiencing sexual dysfunction, highlighting a widespread issue in survivorship care. Survivors treated with chemotherapy, endocrine therapy, or radiotherapy were significantly more likely to report sexual dysfunction, with chemotherapy having the strongest association. The findings emphasize the need for healthcare providers to proactively address sexual health with breast cancer survivors, particularly those undergoing chemotherapy.

Concurrent: B5-Health Economics/Outcomes Research

Monday, November 3, 2025, 10:00:00 AM - Hyatt Doll/Herald

Cost-Effectiveness Analysis of First-Line Tumour Tissue Profiling to Guide Targeted Therapy in Metastatic Pancreatic Ductal Adenocarcinoma: A Canadian Public Health Payer's Perspective

Presenter: Diego Sanchez, BC Cancer

Other authors: Stuart Peacock, Nick Bansback, Kelvin Chan

ABSTRACT_ID#2646

ABSTRACT

Background: Metastatic pancreatic ductal adenocarcinoma (mPDAC) is a highly aggressive malignancy with significant chemotherapy resistance and a 5-year survival below 5%, resulting in a significant economic burden on Canada's healthcare system. Recent international guidelines recommend genetic profiling of tumour tissue (TT) for all newly diagnosed mPDAC patients, which has demonstrated cost-effectiveness in other cancers (e.g., lung). However, the economic value of this approach in mPDAC has not been evaluated. Therefore, this study assesses the cost-effectiveness of TT profiling as a first-line strategy in patients with mPDAC. **Methods:** Analysis was conducted through a decision tree with a 3-state Markov Model based on current NCCN guidelines and the Canadian public payer perspective. Two strategies were compared for ECOG 0-1 mPDAC patients 1) standard care (FOLFIRINOX without TT) and 2) TT followed by targeted therapies (Olaparib, Pembrolizumab, Erlotinib and Sotorasib). The model estimates the effect of TT profiling on costs and quality-adjusted life-years (QALYs) discounted 1.5% annually. Healthcare costs and model parameters were retrieved from the literature and validated by clinical experts. **Results:** Tumour profiling followed by Olaparib produced the highest number of QALYs (6,666.87) but at a greater cost (\$173M; ICER: \$49,219/QALY). At willingness-to-pay (WTP) thresholds above \$40,000/QALY, Olaparib had a nearly 100% probability of being optimal. Erlotinib was less costly but less effective, while Pembrolizumab and Sotorasib were dominated. **Conclusion:** First-line TT profiling to guide Olaparib therapy in mPDAC patients is a cost-effective strategy compared to the standard of care at conventional Canadian WTP thresholds, supporting its adoption in clinical practice.

LAY SUMMARY

Metastatic pancreatic cancer offers very low survival and high healthcare costs due to expensive cancer drugs, adverse effects and prolonged hospital stays. Upfront genetic profiling from tumour tissue can guide the selection of cancer drugs for more personalized treatment, increasing the probability of treatment success and potentially saving money for the Canadian healthcare system. This research aims to evaluate whether the use of upfront tumour tissue profiling can guide the selection of cancer drugs (Olaparib, Pembrolizumab, Erlotinib, and Sotorasib) and provide a better quality of life per dollar spent compared to standard treatment. Our evaluation shows that upfront tumour tissue profiling can guide Olaparib to extend life and improve quality of life compared to standard therapy. While other drugs we looked at (Pembrolizumab, Erlotinib and Sotorasib) did not offer extra benefits to justify their cost. Although Olaparib costs are higher, this approach offers good value for money under Canada's willingness-to-pay thresholds.

Concurrent: B5-Health Economics/Outcomes Research

Monday, November 3, 2025, 10:00:00 AM - Hyatt Doll/Herald

Policy Brief: Enhancing Access to CAR T in the Canadian Healthcare System

Presenter: Mona Shafey, Alberta Health Services

Other authors: Chris Bredeson, Isabelle Fleury, Kathy Gesy, Marie Hawkins, Brian O'Rourke, Christina Sit

ABSTRACT_ID#2725

ABSTRACT

Background Chimeric Antigen Receptor T-cell (CAR T) therapy represents a paradigm shift in personalized medicine, offering unprecedented hope for individuals battling rare, complex, and potentially lethal conditions. Ensuring equitable access to this innovation requires strategic planning and collaboration to address the nuances of delivering this relatively complex therapy within Canada's decentralized healthcare system. To support Canadian decision-makers in improving CAR T access, Shift Health convened an Expert Panel to develop actionable, high-impact recommendations for the next 2–5 years. Results The Panel proposed 5 key recommendations to improve access to CAR T: 1) Increase the capacity to deliver CAR T in Canada through targeted investments, optimizing resource utilization, expanding scope of existing cancer centres and enhancing industry support to upskill HHR. 2) Expand support for patients and caregivers by addressing travel costs and other treatment expenses through strategic funding, enhanced inter-provincial agreements and stronger industry collaboration. 3) Improve physician and patient awareness of CAR T through increased education by leveraging professional networks and developing educational programs to improve awareness. 4) Enhance CAR T data collection by increasing support for the national CTTC registry and educating people receiving cancer treatment about data security and utility to encourage consent. 5) Optimize the cost-efficiency of CAR T for patients and payers by adopting innovative contracting models and exploring the potential benefits of “made-in-Canada” CAR T. Conclusions Through strategic investments and coordinated efforts, Canada can maximize the transformative potential of CAR T, ensuring all eligible cancer patients have equitable access to this life-saving therapy.

LAY SUMMARY

An Expert Panel comprised of diverse thought leaders from across the Canadian cancer ecosystem, including healthcare providers, health system administrators, a regulatory expert and a patient advocate, engaged in a series of discussions aimed at exploring approaches to increasing equitable access to CAR T therapy across Canada's decentralized healthcare system. The Panel identified several interconnected barriers—including limited healthcare system capacity for delivering specialized therapies, significant burden on patients and caregivers, inconsistent awareness among patients and physicians, challenges in data sharing, and budgetary constraints—that must be addressed in a strategic manner. Underscoring the need for multisector partnerships, the Panel recommended five sets of tangible recommendations that can be implemented over the next 2-5 years through close collaboration between federal, provincial, and territorial healthcare systems, patient and clinical networks, industry, researchers, and national organizations to ensure that the transformative potential of CAR T therapy can be fully realized for people living with cancer in Canada.

Concurrent: B6-Indigenous Cancer Care Across the Continuum

Monday, November 3, 2025, 10:00:00 AM - Hyatt Stephen AB

Storytelling: An Inuit-Led Narrative Approach to Cancer Prevention and Wellness

Presenter: Felicia Adelaja and Jemimah Thomas, Tungasuvvingat Inuit

ABSTRACT_ID#3370

ABSTRACT

This initiative explores the power of Inuit-led storytelling as a culturally grounded method for cancer prevention and community wellness. Recognizing that conventional, Western-style health messaging often fails to resonate with Inuit communities, the project aimed to create a more meaningful and accessible approach by centering Inuit voices, culture and lived experiences. Through a community-based participatory process, six short narrative videos were co-created with Inuit community members in Ontario. These videos address key themes including cancer screening, prevention, personal cancer journeys, caregiver perspectives, and the role of culture in healing. Filmed in culturally significant locations, the stories feature Elders and community members, ensuring they reflected Inuit perspectives. Feedback from viewers in the community highlighted the relatability and emotional impact of the videos, which were seen as more engaging and accessible than standard health materials. The storytelling format allowed for complex health messages to be conveyed in a way that felt personal and culturally respectful. The project demonstrates that Inuit-led storytelling is not only an effective tool for health promotion, but also a means of fostering trust and dialogue between Inuit communities and healthcare providers. By embedding cultural values into cancer prevention messaging, the initiative supports both awareness and empowerment, contributing to broader goals of community wellness and culturally safe care for Inuit.

LAY SUMMARY

- Cultural relevant messaging and tools enhance community impact: By embedding Inuit values, language, lived experience into this narrative series, the initiative made health messaging more relatable and easily received by community members.
- Community-Led approaches build trust and ownership: empowering community members to be involved in initiative planning or resource development builds a pipeline of trust, ownership and engagement. This create opportunities for community to engage with health professionals in a culturally safe way.
- Storytelling as a tool for health messaging and evaluation: narrative videos prove to be more effective than standard materials in conveying complex prevention information. Personal stories from community helps to demystify cancer awareness and prevention, subsequently encouraging dialogue.

Concurrent: B6-Indigenous Cancer Care Across the Continuum

Monday, November 3, 2025, 10:00:00 AM - Hyatt Stephen AB

Indigenous Cultural Safety in Clinical Trials: Realities, Relationships, and Recommendations

Presenter: Laura McNab-Coombs, BC Cancer

Other authors: Lauren Pitre, Roel Schlijper-Bos, Agnes Sauter

ABSTRACT_ID#3160

ABSTRACT

Clinical trials (CTs) play an important role in advancing medical knowledge and improving patient outcomes. For some patients, clinical trials provide the only treatment option when standard of care treatment has been exhausted. Although Indigenous cultural safety (ICS) principles have become keystone in the creation and facilitation of health research relevant to Indigenous peoples, it has yet to be uniformly and appropriately integrated in CTs. Access is a significant barrier in CT accrual, further exacerbated by existing inequities which disproportionately and uniquely affect Indigenous Peoples in British Columbia (BC): historical contexts such as the perpetuation of harm that has historically occurred through the research enterprise, compounded by contemporary experiences of racism and discrimination in healthcare systems, has created a necessity for ICS in CTs. A multiphasic environmental scan of ICS policies and recommendations across Canada, along with relational methods of engagement with First Nations in BC, is being utilized to inform the BC Cancer ICS in CTs framework. This environmental scan will highlight recommendations, and lack thereof, for best practices of ICS in CTs. Highlighting the need for Indigenous-led research initiatives, our team will present our unique approach to “engagement” and “relationships”, terms that are so often described and required in the pursuit of Indigenous health research, and how we are using this work to inform distinctions-based approaches to ICS in CTs.

LAY SUMMARY

Historical injustices committed against Indigenous peoples through various colonial systems and the research enterprise, combined with present day experiences of racism and discrimination, have compounded the lack trust in health research experienced by some Indigenous peoples. When we apply cultural safety principles to our clinical trials practice, we can help make clinical trials safer and more accessible for Indigenous peoples. There are many places where we can apply Indigenous cultural safety (ICS) practices throughout the lifespan of a clinical trial, many of which are uncomplicated and can be implemented immediately. Community engagement should be pursued in a way that follows the four R's in research: respect, relevance, reciprocity and responsibility. How engagement unfolds should be defined by the community with whom you're wanting to engage.

Concurrent: B6-Indigenous Cancer Care Across the Continuum

Monday, November 3, 2025, 10:00:00 AM - Hyatt Stephen AB

Developing the Métis-CTN program: National meeting to co-create a clinical trials navigation pathway for Métis cancer patients

Presenter: Ria Patel, Schulich School of Medicine, University of Western Ontario

Other authors: Milica Paunic, Bharat Gupta, Ibrahim Mohamed, Renee Nassar, Laura McNab-Coombs, Lauren Pitre, Arlene King, Leonard Benoit, April Doxtator, Chantal Wahteeshkin, Verne Ross, Sally Nystrom, Depen Sharma, Christina Trieu, Salah Alhajsaleh, Anaam Jaet, Mahmoud

ABSTRACT_ID#3152

ABSTRACT

The Clinical Trials Navigation (CTN) program was established to address barriers to cancer clinical trials access by supporting patients in navigating the trial search process. Since the implementation of the program, eight patients have self-identified as Indigenous, highlighting the need for a specific, culturally sensitive approach to patient navigation. This project takes a distinction-based approach in developing a Métis-specific clinical trials navigation pathway tailored to the unique needs of Métis communities. To inform its development, a national in-person meeting was organized with Métis patients, Indigenous patient navigators (IPNs), researchers, and clinicians. Métis research methodologies guided the meeting structure, emphasizing co-constructed analysis, community perspective, and relationship building. Participatory methods including empathy mapping, small group storytelling, world-café methodology, and whole group reflection were used to support engagement. Participants identified barriers Métis cancer patients face, including mistrust in the healthcare system, lack of support for ceremony and traditional healing, overly clinical information, limited referral to IPNs, and financial and geographic barriers. Proposed solutions included healthcare provider accountability, specific cultural training, culturally-adapted resources, IPN presence throughout enrollment, and coordinated support and resources. To support Indigenous patient navigators, participants suggested developing patient intake scripts, Indigenous resource lists, and specific navigator training. These insights will guide the design of a culturally safe navigation pathway, and will contribute to overall reconciliation efforts by increasing enrollment of Métis individuals in clinical trials. While the initial focus is on the Métis population, this distinction-based approach will serve as a foundation for adapting this pathway for other Indigenous groups.

LAY SUMMARY

Métis cancer patients face numerous barriers along the clinical trial navigation journey including mistrust in the healthcare system, lack of support for ceremony and traditional healing, overly clinical information, limited referral to Indigenous patient navigators, and financial and geographic barriers. To support Métis patients, solutions included advocating for healthcare provider accountability, specific cultural training, culturally-adapted resources, Indigenous patient navigator presence throughout enrolment, and coordinated support and resources. Supporting Indigenous Patient Navigators is critical in making the navigation process more culturally sensitive and accessible. Participants discussed developing patient intake scripts, Indigenous resource lists, and specific navigator training.

Concurrent: B6-Indigenous Cancer Care Across the Continuum

Monday, November 3, 2025, 10:00:00 AM - Hyatt Stephen AB

Cancer Whiri; An Indigenous led model that enhances delivery of cancer care and well-being for Maori, Indigenous peoples of New Zealand.

Presenter: Nina Scott, Te Whatu Ora Waikato (Health New Zealand)

Other authors: Amy Jones, Myra Ruka, Deanne King, Jacquie Kidd

ABSTRACT_ID#2961

ABSTRACT

In Aotearoa New Zealand (AoNZ), Maori cancer experts sought ways of supporting Maori, Indigenous peoples of AoNZ, that were health enhancing, equity achieving and implementable nationally. Using research that developed and tested cancer care pathways by Maori and for Maori, we developed Cancer Whiri. Aim To develop a model of care that results in positive, culturally safe engagement with health providers, timely access to high quality investigations and care, and well-being gain for individuals and families. We sought to co-design, implement and evaluate Cancer Whiri. Method Whiri includes an integrated team structured around navigators, a well-being screening tool, and is nurse led with strong Indigenous clinical governance. The team developed Cancer Whiri using Kaupapa Maori methodology. The model was piloted with 34 Maori patients, referred for suspicion of cancer, with further development in a follow-on project. Results & Discussion Patients had 202 unmet needs identified and supported, with high positive patient experience survey ratings, including feeling supported and reassured by the Whiri team. The Cancer Whiri model incorporates: patient and whanau centred care; relationships; maximised hauora/wellbeing and equity gain; systems; and tino rangatiratanga/Maori autonomy. Within the pilot these components allowed healthcare professionals to create culturally safe environments to enhance the delivery of care, while incorporating modern and traditional medical practices such as rongoa Maori. Conclusion The Cancer Whiri model has potential to expand nationally with reach from primary care through to hospital and palliative care. The team is driving implementation on a national scale with plans for international reach.

LAY SUMMARY

By addressing unmet health and well-being needs you can maximise health gain for patients and their families Whiri could make large gains in cancer survival for Maori and other Indigenous populations Whiri addresses cancer prevention for individuals and whanau/ family, which results in intergenerational and 'up and downstream' impacts

Concurrent: B6-Indigenous Cancer Care Across the Continuum

Monday, November 3, 2025, 10:00:00 AM - Hyatt Stephen AB

Understanding First Nations Ways of Knowing and Doing in Cancer Care in Yukon: Using intergenerational and survivorship models to inform community wellness

Presenter: Liris Smith, Yukon University

Other authors: Sherrie Frost, Kayla Mintz, Samantha Soo, Pier-Luc Turcotte, Vera Charlie, Gladys Netro, Wendy Gifford, Michelle Leach, Sandra Houle, Catherine Larocque

ABSTRACT_ID#2803

ABSTRACT

Cancer in Northern Canada is a leading cause of death with rates higher than the national average. The cancer burden for First Nations people is further exacerbated with significantly poorer 5-year survival rates. Causes are complex and include low rates of screening, late-stage diagnoses, and large geographical distances for health services. However stronger reasons stem from the ongoing legacies of colonization and residential schools that created social exclusion, structural racism, and lack of trust within health care. Despite concerns about cancer rates, First Nations people have not been meaningfully included to address their cancer care needs, and First Nations ways of knowing and doing have not been incorporated into cancer care services and supports. This community-based study co-creates local cancer care prevention and supports with two Yukon First Nation communities, Old Crow and Carmacks. By working in partnership with Indigenous Elders, knowledge keepers and community members in two remote communities, while using community-based methodologies and land-based approaches to wellness, this project supports the creation of outcomes that can be used to inform local cancer prevention and care. Expected outcomes are cancer prevention and care that upholds Indigenous knowledges, and traditional teachings of wellness in Yukon First Nations communities. We also evaluate community strengths and barriers to implementing the local approaches to care. This is the first phase of a multi-phase research program to co-develop and implement health care services and supports that are created by, and for, First Nations people in Yukon.

LAY SUMMARY

This project highlights the process of full community engagement and use of First Nation methodologies and approaches to inform wellness care at the local level. Using land-based approaches that honor the traditions of the community highlights how cancer care priorities can be supported within rural, remote and Indigenous communities. An intergenerational approach to wellness, through the lens of cancer care is important in bringing forward knowledge from past generations to inform the health and well-being of current and future generations.

Concurrent: C1-Tumor Biology, Immunology & the Microenvironment

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 4/6

RNF126 Orchestrates Immune Evasion via Coordinated Regulation of PD-L1 and MHC-I

Presenter: Pinaki Bose, University of Calgary

Other authors: Daesun Kim, Christopher John Gafuik, Ayan Chanda, Jinsu An, Douglas J Mahoney

ABSTRACT_ID#3322

ABSTRACT

Tumor cells evade immune surveillance by upregulating PD-L1 that suppresses cytotoxic T cell activity, and downregulating MHC class I that impairs antigen presentation to CD8⁺ T cells. Reversing these immunosuppressive phenotypes present a compelling therapeutic strategy. Using a pan-ubiquitinome siRNA-based screen, we identified the E3 ubiquitin ligase, RNF126 as a key regulator of both PD-L1 and MHC-I expression in oral squamous cell carcinoma (OSCC), the most common form of head and neck squamous cell carcinoma (HNSCC). Depletion of RNF126 in human OSCC cell lines reduced PD-L1 and increased MHC-I cell surface levels in response to IFN γ . Similarly, RNF126-deficient MOC2 mouse tumors displayed a more immunogenic tumor microenvironment compared to parental controls. These tumors were also more sensitive to immune checkpoint blockade (ICB) with anti-PD-1 and anti-CTLA-4 therapies. Bioinformatics analyses of human datasets revealed that RNF126 expression is significantly elevated in HPV- HNSCC, including OSCC and specifically in cancer cells in single cell RNAseq analysis. Genes upregulated after RNF126 overexpression in OSCC cells, localized to the leading edge of tumour samples in spatial transcriptomic analysis. Notably, patients with lower RNF126 expression exhibited improved survival outcomes, a trend observed across multiple squamous cell cancers. Together, these findings position RNF126 as a dual-function immune regulator that promotes immune evasion by increasing PD-L1 and suppressing MHC-I. Targeting RNF126 may therefore reprogram the tumor immune microenvironment and enhance responsiveness to ICB in HNSCC and other malignancies.

LAY SUMMARY

We discovered a molecule that helps tumors avoid the immune system. Oral cancers often escape immune attack by increasing PD-L1 (which blocks immune cells) and decreasing MHC-I (which helps immune cells detect cancer). We identified a protein called RNF126 that controls both of these processes, making it a central player in immune evasion. Blocking RNF126 makes tumors more visible to the immune system. When we reduced RNF126 in lab-grown cancer cells and in mouse tumors, the tumors became more recognizable to immune cells and responded better to common immunotherapies like anti-PD-1 and anti-CTLA-4. We used spatial analysis to see where RNF126 acts in tumors. Using spatial transcriptomics, we found that RNF126-driven gene activity was concentrated at the invasive edges of tumors—areas thought to be most involved in tumor spread and immune escape. High RNF126 is linked to worse outcomes. In patient datasets, tumors with higher RNF126 had poorer survival, especially in HPV-negative cancers. This trend was also seen in other squamous cell cancers. These findings suggest that targeting RNF126 could help reprogram the tumor's immune environment and improve treatment outcomes in hard-to-treat cancers.

Concurrent: C1-Tumor Biology, Immunology & the Microenvironment

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 4/6

High dose radiation therapy promotes immune suppressive macrophage phenotypes, whereas low dose radiation therapy promotes immunogenic macrophage phenotypes in an immune competent murine model of soft tissue sarcoma

Presenter: Kurt Hildebrand, University of Calgary

Other authors: Karys Hildebrand, Carolina Salazar Arcila, Franz Zemp, Hyojin Song, Kiran Narta, Ted Verhey, Sorana Morrissy, Douglas Mahoney, Michael Monument

ABSTRACT_ID#3113

ABSTRACT

Soft tissue sarcomas (STS) are aggressive tumours of mesenchymal origin. STS are treated with neoadjuvant radiation therapy (RT) of 2Gy in 25 fractions in combination with surgery. Recently, studies have shown that RT modulates the immune compartment of various tumours. STS treated with RT and anti-PD1 showed improved outcomes. However, patients receiving RT had increased immune suppressive markers on tumour associated macrophages (TAMs). Thus, RT may exacerbate the immune suppressive TAM phenotypes in STS. Given that TAMs are the most abundant immune population in STS, it is essential to understand how RT influences TAM phenotypes, which has not been investigated. Syngeneic, murine STS were engrafted in C57Bl/6 mice, then given RT when tumours were ~500-800mm³ using three regimens. 1) 6Gy x5 clinically equivalent RT, 2) 8Gy x3 high dose hypofractionated RT, and 3) 8Gy x1 low dose RT. A SmART+ (Precision X-ray) irradiator was used to deliver RT. Tumours were assessed at 72hrs and humane endpoint (tumours >15mm) after RT by flow cytometry. We found that 6Gy x5 and 8Gy x3 increased the proportion of TGFB1+ TAMs at 72hrs, whereas the 8Gy x1 reduced CD206+ and TGFB1+ TAMs, and increased CD80+ TAMs. Interestingly, the 6Gy x5 and 8Gy x3 led to increased CD3ε+ and CD8α+ T cells, as well as increased activated CD8α+ T cells. In contrast, the 8Gy x1 did not increase T cells, but increased proportions of activated T cells. Thus, high dose RT promotes immune suppressive TAMs, whereas low dose RT may enhance anti-tumour TAMs.

LAY SUMMARY

- Radiation therapy modulates the immune compartment in many tumours; however, this has received little investigation in soft tissue sarcomas (STS).
- Herein, we assessed how radiation therapy influences macrophages, which are the most abundant immune population in STS.
- We have shown that clinically equivalent radiation doses given to mouse STS are associated with exacerbating immune suppressive macrophage phenotypes. In contrast, a single dose of 8Gy x1 radiation therapy reduced immune suppressive macrophage phenotypes.

Concurrent: C1-Tumor Biology, Immunology & the Microenvironment

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 4/6

Control of brain tumor progression by the dual function cytokine IL-33

Presenter: Shyam Menon, Lady Davis Institute for Medical Research, McGill University

Other authors: Tala-Maria Mouaness, Varsha Manoharan, Peipei Zeng, Isabelle Carrier, Jianbo Zhang, Alisha Poole, Xueqing Lun, Eduardo Diez, Bo Young Ahn, Ngoc Ha Dang, Katalin Osz, Jennifer Chan, Daniela Quail, Sorana Morrissy, Stephen Robbins, Donna Senger

ABSTRACT_ID#2617

ABSTRACT

Glioblastoma, the most common and lethal adult brain cancer, is presently incurable with a combination of neurosurgery, chemotherapy, and radiation. A major barrier to glioma control is the complex interplay between tumor cells and the innate immune microenvironment in the brain, that creates an immunosuppressive and tumor supportive niche. Previously, we established that glioma cells can communicate with microglia/macrophage through the dual-function (nuclear and secreted) cytokine IL33. Tumor-derived IL33 supports the recruitment and functional reprogramming of microglia/macrophage to a pro-tumorigenic state which fuels tumor growth. In contrast, when IL33 is prevented from entering the nucleus, by deletion of its nuclear localization sequence [nuclear deficient IL33 (ND-IL33)], tumor growth is dramatically suppressed enabling extended long-term survival. Using multiplex immunohistochemistry, we profiled the progression of ND-IL33 tumors and uncovered a population of glioma-inhibitory macrophages (GIMs) unique to this growth restrictive environment. To characterize the molecular properties of GIMs, we performed spatial transcriptomics and employed a computational pipeline based on unsupervised deconvolution to ascertain pathways and cell types enriched in the ND-IL33 environment. This analysis unveiled that the GIMs are upregulated in phagocytosis and type-1 interferon pathways. Recapitulation of this state using microglia or macrophage in vitro facilitated the direct killing of human and mouse glioma cell-lines. The tumor-suppressive role of GIMs was further verified in vivo when co-implantation of ND-IL33-expressing glioma cells with highly tumorigenic cells led to a growth-restrictive environment and prolonged survival. Development of strategies to deliver ND-IL33 and induce GIM activation may have a future in glioblastoma therapy.

LAY SUMMARY

Glioblastoma is a deadly and incurable brain cancer that affects ~1,500 Canadians each year. Immunotherapies - therapies that alter white blood cells of the immune system to recognize and kill tumours - have made a breakthrough as curative options for many cancers. However, these therapies have been largely unsuccessful for glioblastoma due to the composition of innate immune cells in the brain. Previously, we identified a protein called interleukin 33 (IL33) that tumour cells use to communicate with innate immune cells called microglia and macrophage in the brain. IL-33 expressed by the tumor cells can reprogram the immune cells to a state that promotes rapid and fatal tumor growth. Strikingly, blocking one of the functions of IL33 reverses this behavior and suppresses tumor growth, leading to very long-term survival. Our analysis shows that this growth restrictive environment is driven by a population of tumor-killing macrophage. By characterizing this immune cell population further, we imagine a novel treatment strategy to combat glioblastoma progression using the innate immune cells.

Concurrent: C1-Tumor Biology, Immunology & the Microenvironment

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 4/6

Leupaxin deficiency results in increased differentiation of CD8 T cells into CD8+NK1.1+ T cells and decreased tumor growth

Presenter: Hanne Ostergaard, University of Alberta

Other authors: Kevin Kane, Nafiseh Ghobakhloo

ABSTRACT_ID#3175

ABSTRACT

Leupaxin is a cytoskeletal adaptor protein primarily expressed in leukocytes and is highly upregulated in cytotoxic CD8 T cells. However, its role in CD8+ T cells remains undefined thus we investigated the impact of leupaxin deficiency on tumor growth. We generated leupaxin knockout (LKO) mice as well as mice where leupaxin was selectively deleted in CD8 T cells. In both mice there was substantially reduced subcutaneous EG7 lymphoma tumor growth compared to control mice, with no tumors detected in about 50% of the LKO mice. Analysis of T cells in the tumors showed no difference in the number of antigen-specific tumor infiltrating T cells. There were more CD8 T effector/T effector memory (Teff/Tem) cells in the LKO mouse tumors. Within Teff/Tem in LKO tumors, there was an increased frequency of a rare CD8⁺NK1.1⁺ T cell subset, known to have potent anti-tumor activity. Spectral flow cytometry followed by cluster analysis revealed an enrichment of CD8+NK1.1+ T cells in LKO tumors with high CD107a surface expression indicative of recent degranulation within the tumor. Similar results occurred in orthotopic E0771 breast tumors, where tumor growth was significantly reduced in LKO mice and associated with an increased CD8⁺NK1.1⁺ T cell frequency. This study is the first to identify leupaxin as a negative regulator of CD8⁺ T cell function in tumors. The loss of leupaxin promotes the differentiation of CD8⁺ T cells into NK1.1⁺ Teff/Tem cells, enhancing their anti-tumor activity and inhibiting tumor growth. These findings uncover a novel immunoregulatory mechanism of leupaxin.

LAY SUMMARY

Killer cells of the immune system are able to kill cancer cells and win some battles, but the cancer has many ways of escaping or shutting down the immune system so that the cancer generally wins the war. We have found a way to make killer cells more effective at killing tumor cells to help the immune system win to more effectively kill the cancer. We found that if you remove a particular protein called leupaxin from killer cells, they can turn into super killers that are able to clear up to 50% of the tumors in a preclinical model system. This is because while most of the killer cells of the immune system have one way of recognizing a cancer cells as something that needs to be destroyed, some super killer cancer cells have many ways of recognizing cancer cells making it harder for cancer cells to escape. If we can find a way to either get rid of leupaxin in certain immune cells or identify a drug that blocks its function, we might be able to use these cells to generate the super killer cells to eliminate a number of different types of cancer.

Concurrent: C2-Bioinformatics and Precision Medicine

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 1/2/3

Microbiome profiling reveals that fecal microbiota transplantation (FMT) modulates response when combined with immunotherapy in patients with lung cancer and melanoma (FMT-LUMINate, NCT04951583)

Presenter: Arielle Elkrief, Centre hospitalier de l'Université de Montréal (CHUM)

Other authors: Sreya Duttagupta, Meriem Messaoudene, Rahima Jamal, Catalin Mihalciou, Nicolas Marcoux, Sebastian Hunter, Karl Belanger, Mustapha Tehfe, Normand Blais, Scott Owen, Jacques Raphael, Jonathan Lenehan, Wiam Belkaid, Seema Nair Parvathy, Antoine Desilets, Antoine Désilets, Nicola Segata, Laurence Zitvogel, Michael Silverman, Saman MalekiVarek, Bertrand Routy

ABSTRACT_ID#3325

ABSTRACT

Background: Immune checkpoint inhibitors (ICI) have transformed cancer care, yet resistance remains a major limitation. Fecal microbiota transplantation (FMT) may overcome resistance to ICI. Methods: We conducted a multicenter phase II study of FMT combined with ICI in patients with advanced lung cancer (NSCLC) treated with anti-PD-1 (?PD-1) monotherapy, and melanoma patients treated with dual ICI (?PD-1+?CTLA-4) in the first-line setting. Twenty patients per cohort received oral FMT from 1 of 11 healthy donors (HD) following bowel preparation followed by ICI initiation one week later. The primary endpoint was objective response rate (ORR). The gut microbiome was profiled using shotgun metagenomics. Metabolomic profiling was performed using HPLC. Olink proteomics (96 markers) on plasma and flow cytometry (40 markers) on PBMCs were assessed. FMT was performed in germ-free mice to recapitulate engraftment. Results: In NSCLC, the objective response rate (ORR) was 80% with no grade >3 immune-related adverse events (irAE). In melanoma, ORR was 75%, and 65% experienced grade >3 irAE. Responders (R) and non-responders (NR) exhibited distinct post-FMT microbiome profiles ($p=0.03$) with enrichment of specific beneficial bacteria post-FMT. R showed enrichment of polyamines and bile salts in the plasma, and expansion of exhausted T central and effector memory cells in PBMCs. NR exhibited elevated IL-10 and CD28 levels in the plasma. Conclusion: This study demonstrates the potential of FMT to enhance ICI efficacy in NSCLC and melanoma and support the study of FMT in an ongoing pan-Canadian RCT (Canbiome-2, NCT06623461).

LAY SUMMARY

Immunotherapy has revolutionized cancer treatment, but many patients still do not respond. One possible solution is fecal microbiota transplantation (FMT), a treatment where gut bacteria from healthy donors are transferred to patients to improve their microbiome. In this study, we tested whether FMT could boost the effects of immunotherapy in people with advanced lung cancer or melanoma. Forty patients (20 with lung cancer, 20 with melanoma) received oral FMT from one of 11 healthy donors, followed a week later by standard immunotherapy. In lung cancer, 80% of patients responded to treatment, with no serious side effects. In melanoma, 75% responded, but 65% developed serious immune-related side effects. We found that patients who responded had different gut bacteria after FMT, with an increase in helpful microbes. These patients also showed beneficial changes in their blood, including molecules involved in metabolism and immune memory cells. In contrast, non-responders had higher levels of markers linked to immune exhaustion. These results suggest that modifying the gut microbiome with FMT may improve how well immunotherapy works, though the risks may differ depending on the cancer type. A larger Canadian trial (Canbiome-2) is now underway to confirm these findings.

Concurrent: C2-Bioinformatics and Precision Medicine

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 1/2/3

Characterizing the effects of radiation therapy on the genomic landscape of murine models of undifferentiated pleomorphic sarcoma

Presenter: Maximilian Labrecque, University of Calgary - Riddell Centre for Cancer Immunotherapy

Other authors: Michael Monument

ABSTRACT_ID#2950

ABSTRACT

Soft tissue sarcomas are a rare form of solid tumour that are particularly common among children and young adults. A decade of advancements in cancer immunotherapies have not yet translated to improved patient outcomes for advanced sarcoma, which remains characterized by low survival and high relapse rates. However, recent clinical trials have reported a combination of immune checkpoint blockade and radiation therapy yielding positive results in undifferentiated pleomorphic sarcoma (UPS), a common sarcoma subtype. In order to examine the efficacy of radiotherapy as a possible mechanism to increase tumour sensitivity to immunotherapies in UPS, we investigated a well-documented murine model of UPS (KrasG12Dp53^{-/-}) before and after treatment with varying doses of ionizing radiation. These treatment courses aimed to replicate high dose (6-8 Gy/fraction), hypofractionated radiation treatments (HFRT) that have seen recent successes in remodeling the tumour microenvironment in other cancer types. We then compared the genomic landscape of these models with another known model of murine UPS (Pten^{-/-}p53^{-/-}), and one novel cre-recombinase driven model (KrasG12DPten^{-/-}p53^{-/-}). Whole-exome and bulk RNA sequencing revealed distinct mutational and antigenic profiles for each sarcoma cohort, both between models as well as between spontaneous and engrafted cohorts of the same model, giving insight into the importance of tumour background in the context of accurate mouse modeling of human sarcoma. In addition, an examination of mutations in oncogenic passenger genes reveals the extent to which high dose HFRT modifies the tumour genomic landscape, opening the door to potential future applications of HFRT in sarcoma combination therapies.

LAY SUMMARY

Advanced sarcoma is a rare but highly fatal disease that disproportionately affects children and young adults. Recent findings support the idea that by modifying how we treat these tumours with radiation therapy, we might be able to mutate them such that they are more sensitive to immunotherapies. To test this, we treated a widely-used mouse model of sarcoma with high dose radiation therapy, and compared its tumour genomes to those of other sarcoma mouse models; including a new one we created ourselves. We found that our different sarcoma models have different genetic profiles depending on which few genes are driving tumour growth. Differences are also observed between tumours with the same driver genes depending on whether the tumours arose spontaneously in genetically-modified mice, or were engrafted from cancer cells grown in a petri dish. We show that high dose radiation therapy alters the genomes of our sarcoma models, and this can help us explain trends we see in what genes are expressed in these models. Overall, our research provides insight into genomic changes that may be of interest in the development of new combination therapies for sarcoma.

Concurrent: C2-Bioinformatics and Precision Medicine

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 1/2/3

Metabolic syndrome and prostate cancer risk: results from the BIOmarkers of Prostate Cancer/Prevention and Environment (BIOCaPPE) prospective study

Presenter: Clément Larose, Centre hospitalier universitaire de Québec-Université Laval

Other authors: Roxane Tourigny, Karine Robitaille, Fred Saad, Michel Carmel, Armen Aprikian, Yves Fradet, BIOCaPPE Network, Vincent Fradet

ABSTRACT_ID#3002

ABSTRACT

Background: Metabolic syndrome (MS) is characterized by a combination of metabolic abnormalities (central obesity, insulin resistance, dyslipidemia and hypertension). The aim of this study is to investigate the association between MS and prostate cancer (PCa) risk. Methods: Between 2013 and 2020, 2,053 men at high risk of PCa were enrolled in the prospective, multicentre, observational study BIOmarkers of Prostate Cancer/Prevention and Environment (BIOCaPPE). MS was determined in accordance with established criteria of the International Diabetes Federation. History of cardiovascular disease (CVD) was collected via a self-administered questionnaire. Multivariate logistic regression models were used to evaluate the association between MS and CVD, and the risk of overall and aggressive (defined as Grade Group ≥ 3) PCa. Results: MS and CVD data were available for 1,945 participants with a median clinical follow-up of 6.2 years (Q1–Q3: 4.4–7.8). From those, 310 cases of PCa were diagnosed, of which 99 were aggressive. While MS was not associated with the risk of overall PCa (HR 1.14; 95%CI 0.88–1.49; $p=0.329$), it was positively associated with aggressive PCa (HR 1.48; 95%CI 1.00–2.19; $p=0.049$). CVD were associated with a significant increased risk of overall PCa (HR 1.56; 95%CI 1.16–2.14; $p<0.001$) and more importantly, with aggressive PCa (HR 1.95; 95%CI 1.68–2.26; $p<0.001$). Conclusion: These findings suggest that CVD and MS are associated with an increased risk of developing aggressive PCa. Identifying and managing MS could therefore provide an opportunity for stratifying cancer risk and developing prevention strategies for men at risk of PCa.

LAY SUMMARY

Metabolic syndrome is a cluster of risk factors (such as high blood pressure, high blood sugar and abdominal obesity), a warning sign of an imbalance within the body. Although genetic factors may play a role in the metabolic syndrome, it is mainly the result of a sedentary lifestyle, poor diet, or excess weight. Therefore, there is potential for enhancement through lifestyle modifications and therapeutic interventions. Based on our analyses in a large pan-Canadian cohort of men at high risk of prostate cancer, the presence of metabolic syndrome was not associated with the risk of developing prostate cancer. However, men with the metabolic syndrome appear to be more at risk of developing the aggressive form of the disease. Managing metabolic syndrome — through healthy eating, regular physical activity, and weight control — may be an effective strategy for reducing the risk of developing aggressive prostate cancer.

Concurrent: C2-Bioinformatics and Precision Medicine

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 1/2/3

Multiomic Landscape of Human Gliomas From Diagnosis to Treatment and Recurrence

Presenter: Hadeesha Piyadasa, Stanford University

Other authors: Benjamin Oberlton, Mikaela Ribi, Jolene Ranek, Inna Averbukh, Ke Leow, Michael Angelo, Sean Bendall

ABSTRACT_ID#2309

ABSTRACT

Gliomas remain among the most lethal cancers, with limited treatment options and poor clinical outcomes. To better understand the drivers of glioma progression and immunotherapy resistance, we assembled a clinically annotated cohort of 310 adult and pediatric glioma patients across multiple institutions. Using spatial multi-omics, including multiplexed ion beam imaging, spatial transcriptomics, and spatial glycomics, we profiled over 1.2 million cells at single-cell resolution, capturing immune subsets, tumor antigens (TAs), transcripts, and glycan structures. Our analysis revealed grade- and subtype-specific immune architectures, including immunosuppressive myeloid populations that varied across glioma types. We quantified eight clinically actionable TAs, finding B7H3 and EGFR to be the most abundant in high-grade tumors, though both showed significant cell-to-cell variability. While multi-antigen strategies improved tumor coverage, many patients still had tumor cells lacking any targetable TA, highlighting challenges for targeted therapies. Comparing low-grade gliomas (LGGs) treated with immunotherapy versus standard of care showed only minor differences in immune function and tumor architecture. However, longitudinal analysis of LGG samples revealed dynamic shifts in immune-tumor spatial organization and function over time. To explore mechanisms of immune evasion, we mapped the glioma glycome using mass spectrometry and identified 70 unique N-glycans. Integration with proteomic and transcriptomic data revealed sialylation as a hallmark of high-grade gliomas, linked to immune suppression. A multi-omic machine-learning model showed that glycan features best predicted tumor grade, while immune-related transcriptomic profiles were most associated with survival. Together, these findings emphasize the power of spatial multi-omics in uncovering new therapeutic opportunities in glioma.

LAY SUMMARY

Most glioma therapies target a single tumor marker, but we found that no single marker is present on all tumor cells. By analyzing over 1.2 million cells from 310 brain tumor patients, we showed that using multiple tumor markers (like B7H3 and EGFR) together improves how well we can identify and potentially target tumor cells. The immune environment in brain tumors varies by tumor type and grade. Some tumors are filled with immune cells that suppress the body's ability to fight cancer. We discovered patterns in these immune cells that help explain why some gliomas resist treatment. Sugars on the surface of tumor cells (called glycans) play a hidden role in immune evasion. We identified 70 distinct sugar patterns in brain tumors and found that one type, sialylation, is more common in aggressive tumors. This may help tumors avoid detection by the immune system.

Concurrent: C3-Cross-Pillar Cancer Research Across the Lifespan

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 5/7/9

Imagine... An Immersive Performance Co-Designed by Adolescents and Young Adults (AYA) with Cancer Offering a Felt Sense of Cancer Care and Support in British Columbia

Presenter: Cheryl Heykoop, Royal Roads University

Other authors: Amber Baker, Shandy Bearman, Kat Dornian, Laura Floyd, Jennifer L, Jamie MacIver, Jodi Rethy, Kaylie Rooke, Ashley Tremblay, Roxanne van Velzen, Gary Lam, Genevieve Stonebridge, Jennifer Wolfe, Tiffany Hill, Alice O'Grady

ABSTRACT_ID#2352

ABSTRACT

Background: Adolescents and young adults (AYAs) diagnosed with cancer between the ages of 15-39 have distinct cancer care and support experiences and needs that are different than adult and pediatric patients, and these needs and experiences are often unmet by cancer care and support systems. The aim of this project was to work with a group of AYAs with lived experiences with cancer to co-create an immersive performance – a performance that involves the audience as active participants. Methods: Over a 12-month period we applied an arts-based Patient-Oriented, Participatory Action Research methodology to work with 12 AYA co-researchers to co-design an immersive performance that captured the felt sense of what it means to be an AYA navigating cancer care and support in British Columbia. Results: In November 2024, twelve 40-minute immersive performances were shared with over 100 clinicians, care givers, decision makers, and AYAs at the BC Cancer Summit and we created two videos to showcase the experience. Initial reactions suggest the immersive performance was a powerful and impactful experience. Conclusions: This immersive performance was a creative and impactful research and knowledge translation tool to build knowledge and understanding and inspire change in AYA cancer care and support. Note: This oral presentation will present the immersive experience (in physical and/or video format), will discuss the process of development (including the process, challenges, and opportunities) and explore implications for future practice.

LAY SUMMARY

Adolescent and young adults (AYA) co-designed an immersive performance to capture the felt sense of what it means to be an AYA navigating cancer care and support in BC. The immersive performance debuted at the BC Cancer Summit and was shared with over 100 clinicians, caregivers, decision-makers and AYAs. The performance was well received. Immersive performance offers a creative and impactful way to co-create and share research, build knowledge and understanding, and inspire change.

Concurrent: C3-Cross-Pillar Cancer Research Across the Lifespan

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 5/7/9

Regional comparison of age-stratified incidence rates of colorectal cancer in Ontario: a population-based study across 20 years

Presenter: Stacey Santi, Health Sciences North Research Institute

Other authors: Tasneem Lalva, Joshua O. Cerasuolo, Prashant Jani, Lacey D. Pitre

ABSTRACT_ID#3303

ABSTRACT

Introduction: Colorectal cancer (CRC) is one of the leading causes of cancer-related mortality in Canada. While national rates of CRC incidence have decreased in recent years, attributed partly to increased screening implementation, an alarming trend of increased incidence of early-onset CRC (<50 years of age) is on the rise globally. Northern Ontario is a large geographic region with a highly dispersed population, and a significant proportion of the population resides in regions considered rural or remote. This study aimed to compare temporal trends of CRC incidence across Northern Ontario over a 20-year period (2001-2022). Methods: This study included Ontario residents aged 18 years and older, diagnosed with CRC during 2001-2022. Age and sex-standardized incidence rate ratios (IRR) along with their 95% confidence intervals were calculated. Trends over time were assessed using Kendall's tau-b correlation coefficients. Administrative databases held at ICES were utilized for this study. Results: Incidence rate ratios (IRRs) of CRC were consistently higher in Northern Ontario across all age groups over the 20-year period examined. Although rates of early-onset CRC trended higher than Southern Ontario over time, the relative difference was not statistically significant. Conclusion: In Northern Ontario, the IRRs of early-onset CRC were not significantly different from Southern Ontario. However, the IRRs of CRC, in general, were higher in Northern Ontario compared to Southern Ontario across all age groups. These results will be discussed in relation to screening compliance and risk factors within this population.

LAY SUMMARY

- Colorectal cancer is one of the leading causes of cancer-related mortality in Canada. Incidence rates of early-onset colorectal cancer are rising globally.
- This study assessed the incidence rates ratios of colorectal cancer across age groups (early-onset, average-onset, and late-onset) in Northern Ontario over 20 years.
- While the incidence rates of early-onset colorectal cancer were not significantly higher in Northern Ontario compared to Southern Ontario, rates of colorectal cancer in general, were higher in the age groups examined within this population.

Concurrent: C3-Cross-Pillar Cancer Research Across the Lifespan

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 5/7/9

Addressing the needs and concerns of older adults facing cancer diagnosis: A comprehensive model of recommendations developed through engagement with older adults, family, and healthcare professionals during co-design of a provincial cancer diagnosis prog

Presenter: Fay Strohschein, University of Calgary

Other authors: Sarah Djuric, Anna Pujadas Botey, Sandra Davidson, Paula Robson

ABSTRACT_ID#2356

ABSTRACT

Background: The Alberta Cancer Diagnosis Initiative was launched by the Cancer Strategic Clinical Network, Alberta Health Services, to co-design a centralized program for streamlined, supported cancer diagnosis, attending to underserved groups. In Alberta, 58% of cancer diagnoses occur among those ≥ 65 years. Older age, frailty, and comorbidities are associated with poorer cancer diagnosis experiences, having far-reaching impacts throughout cancer care. Understanding about how to improve the diagnosis process for this growing population is lacking. Purpose: To present recommendations that address the needs and concerns of older adults (OAs) in co-design of a cancer diagnosis program. Methods: Using human-centred design, engagement sessions included 1 focus group and 16 interviews with 5 OAs aged 65-79 years, 3 family caregivers, and 18 healthcare providers/leaders involved in OA care. Sessions were recorded and transcribed. Reflexive thematic analysis, facilitated by NVivo software, informed development of a comprehensive model of recommendations. Results: To ensure understanding of the unique, varied contexts experienced by OAs, including individual, health, relational, and practical dimensions, participants highlighted the need to challenge assumptions based on age, consider quality of life, support early diagnostic decision-making, and facilitate access to diagnostic testing. Implementation of these key principles depended on integrating validated assessment tools; promoting conversation; creating connections; and tailoring communication. A central navigator/hub supported these critical activities. Conclusion: This model highlights key principles and critical activities to address the needs and concerns of OAs facing cancer diagnosis, presenting important opportunities to draw on strengths and resources within the OA community to promote optimal care.

LAY SUMMARY

Older persons experience unique and varied contexts that impact the cancer diagnosis process, including their individual characteristics (e.g., different approaches to self-advocacy, varied preferences/values, interaction of culture/age), their relational circumstances (e.g., unique living situations such as residential care or homecare, various amount of family/friend involvement, past experiences of healthcare/cancer), their health situation (e.g., comorbidities, uncommon/multiple cancer diagnoses), and practical factors (e.g., access to technology/transportation). Four key recommendations to ensure the unique and varied contexts experienced by older persons are addressed within the cancer diagnosis process include (1) challenging decisions and assumptions that are based on age alone; (2) considering the impact of diagnosis on quality of life; (3) supporting early, explicit decision-making about diagnosis and treatment; and (4) facilitating access to diagnostic testing. Having a navigator/hub available to patients, family, and community healthcare providers is central to ensuring these recommendations are implemented, by (1) integrating assessments that help healthcare professionals understand an older persons' strengths and areas for additional support, (2) promoting early and ongoing conversations about goals/preferences among patients, family, and healthcare providers, (3) creating connections to ensure continuity among community, primary, and cancer care providers, (4) tailoring the format and content of communication to older persons.

Concurrent: C3-Cross-Pillar Cancer Research Across the Lifespan

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 5/7/9

Cancer risk stratification at the time of birth: potential roles of newborn screening in diagnosing cancer predisposition syndromes

Presenter: Yiming Wang, The Hospital for Sick Children

ABSTRACT_ID#2986

ABSTRACT

Cancer predisposition syndromes (CPS) are caused by germline pathogenic variants in cancer predisposition genes (CPGs), leading to increased cancer risks. At least 15-20% of pediatric cancers are associated with underlying CPS. Some CPS, such as Li-Fraumeni syndrome (LFS, due to germline pathogenic variants in TP53), are highly penetrant disorders associated with early onset of cancers in children. Ground-breaking advances in the surveillance and management of CPS have significantly improved survival, and current guidelines recommend cancer surveillance from the time of birth in many pediatric CPS. However, most CPS are only identified after tumour diagnoses and miss the opportunity for early surveillance and intervention. Leveraging newborn screening (NBS) may represent an opportunity to identify patients with CPS at the earliest timepoint, prior to the development of cancer. NBS is the acquisition of neonatal dried blood spot (DBS) samples through heel pricks, enabling the screening of many pediatric disorders. To test the utility of NBS DBS in diagnosing CPS, we recruited patients with LFS and healthy controls. Genome and targeted gene panel sequencing are applied to their NBS DBS. We will explore the sequencing accuracy and evaluate the quality of the sequencing data from NBS DBS for TP53 and other CPGs. We also initiated a questionnaire and interview-based study to assess the acceptability of CPS NBS and parental views on models of consent, result disclosure, and the psychosocial impact of diagnosing CPS at birth. These and future studies in CPS may establish NBS as a tool for neonatal cancer risk stratification.

LAY SUMMARY

- A unique feature of cancers in children and young adults is the significant contribution of genetic factors. The genetic disorders that increase the risk of cancer development are called cancer predisposition syndromes (CPS).
- In the past few decades, cancer surveillance protocols through various physical examinations, laboratory tests, and imaging modalities, such as the “Toronto Protocol” for Li-Fraumeni Syndrome established by my colleagues at SickKids, have enabled the early diagnosis of cancers in patients to allow effective, targeted therapy, leading to significantly improved survival. Many of these guidelines for CPS in children require starting the surveillance from the time of birth. However, most children only present to the hospital when they develop cancer due to the lack of early diagnosis tools.
- Newborn screening (NBS) can diagnose these high-risk conditions at the time of birth, enabling early surveillance, treatment, and prevention.

Concurrent: C4-Clinical Research & Radiotherapy

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 8

Stereotactic Ablative Radiotherapy for up to 5 Oligometastases: Long-Term Outcomes of the Population-Based Phase 2 SABR-5 Clinical Trial

Presenter: Robert Olson, University of British Columbia and BC Cancer

Other authors: Curtis Leclerc, Sarah Baker, Will Jiang, Benjamin Mou, Mitchell Liu, Alanah Bergman, Devin Schellenberg, Abraham Alexander, Hannah Carolan, Siavash Atchian, Nick Chng, Quinn Matthews, Alexander Benny, Scott Tyldesley

ABSTRACT_ID#2385

ABSTRACT

Purpose: To evaluate long-term secondary trial endpoints of overall survival (OS), progression-free survival (PFS), local control rate, and prognostic factors in the population-based phase 2 SABR-5 trial. **Methods:** The SABR-5 trial was a single-arm phase 2 study with the primary endpoint of toxicity, conducted at the 6 regional cancer centers across British Columbia (BC), Canada, during which time SABR for oligometastases was only offered on trial. Patients with up to 5 oligometastases underwent SABR to all lesions. **Results:** Between November 2016 and July 2020, 380 patients underwent SABR on trial. The 3 most common histologies were prostate (32.1%), colorectal (16.6%), and breast (11.1%). Median OS was 64.6 months (95% confidence interval [CI], 61.0-68.1). Median PFS was 14.6 months (CI, 11.6-17.6). 5-year OS, PFS, and local control were 58.6% (CI, 53.5 - 63.7), 20.3% (CI, 16.2 - 24.4), and 85.1% (CI, 82.0-88.1) respectively. On multivariable analysis, ECOG ≥ 1 (Hazard Ratio [HR], 1.53; $p = 0.01$), tumour diameter (HR, 1.13; $p < .001$), colorectal histology (HR, 2.25; $p = 0.008$), lung histology (HR, 4.25; $p < .001$), 1-2 treated lesions (HR, 2.17; $p = 0.021$), no upfront systemic therapy (HR, 1.79; $p = 0.032$) and no synchronous oligometastatic disease (HR, 1.56; $p = 0.04$) were significant independent predictors of worse OS. **Conclusion:** In this large population-based phase II trial of SABR for up to 5 oligometastases, the median OS and PFS were 64.6 months and 15.4 months, respectively. The favorable OS and PFS support continued enrollment in SABR trials as we await phase III trial results.

LAY SUMMARY

SABR-5 showed promising long-term outcomes for patients with up to 5 metastases. Median overall survival was over 5 years, and local control at 5 years exceeded 85%, supporting the potential of SABR as a curative approach for selected patients. Several factors predicted better outcomes, including having fewer treated lesions, receiving systemic therapy before SABR, and having synchronous (vs. recurrent) oligometastatic disease. These findings may help clinicians identify patients most likely to benefit from SABR. This real-world, province-wide trial demonstrates the feasibility and value of population-based SABR programs, reinforcing the need to support enrollment in ongoing phase III trials as evidence continues to build.

Concurrent: C4-Clinical Research & Radiotherapy

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 8

Niacin modulates immune responses in newly diagnosed glioblastoma patients

Presenter: Candice Poon, University of Calgary

Other authors: Kathleen Hagen, Susobhan Sarkar, Reza Mirzaei, Claudia Silva, Aito Ueno, Paula de Robles, Gloria Roldan-Urgoiti, Voon Wee Yong

ABSTRACT_ID#2744

ABSTRACT

INTRODUCTION: Immunotherapies such as immune checkpoint blockade have revolutionized cancer care in solid tumors such as melanoma, but have limited success in glioblastoma (GBM). GBM thwarts immunotherapies by fostering a potentially immunosuppressive local and systemic microenvironment, with diminished immunity such as decreased numbers of T cells and NK cells reflected in the peripheral blood. Niacin (vitamin B3) has shown promise in preclinical studies for stimulating immune cells in the peripheral blood to fight local GBM microenvironment immunosuppression, improving outcomes. In this phase I trial (NCT04677049), our objective was to determine whether niacin modulates systemic immunity in newly diagnosed GBM patients. **METHODS:** Oral controlled-release niacin was administered according to a dose escalation schedule (500-3,000mg daily) alongside standard of care surgery and chemoradiation in patients with newly diagnosed GBM. Using multi-parameter flow cytometry and Luminex multiplex analyses of blood samples, we measured the immunologic response of 15 patients. **RESULTS:** Median age was 57 years and 60% were male. Circulating memory T cell ($p = 0.020$) and natural killer cell ($p = 0.027$) frequencies increased with niacin dose escalation, while non-classical monocyte frequency (an immunosuppressive population) decreased ($p = 0.019$). Niacin also elevated serum levels of the pro-inflammatory cytokine interleukin (IL)-12p70 ($p = 0.0078$) and granulocyte colony-stimulating factor (G-CSF; $p = 0.023$), while reducing growth-regulated alpha protein (GROa; $p = 0.039$). **CONCLUSION:** This data demonstrates that niacin, a low-cost and accessible drug, promotes a systemic anti-tumor immune profile in GBM patients and warrants further evaluation as a therapeutic adjunct.

LAY SUMMARY

Niacin (vitamin B3) modulates systemic immune responses in newly diagnosed glioblastoma patients Niacin increases memory T cell and NK cell frequencies alongside pro-inflammatory/anti-tumor cytokines, while decreasing non-classical monocyte frequency and pro-tumor cytokines Niacin is a low-cost, accessible drug that warrants further investigation as a therapeutic adjunct alongside standard of care GBM treatment +/- other immunotherapies

Concurrent: C4-Clinical Research & Radiotherapy

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 8

3CTN: A Decade of Impact in Advancing Academic Cancer Clinical Trials in Canada

Presenter: Rebecca Xu, Canadian Cancer Clinical Trials Network (3CTN)

Other authors: Diana Kato, James Schoales, Stephen Sundquist, Raisa Chowdhury, Janet Dancey

ABSTRACT_ID#3045

ABSTRACT

Introduction Launched in 2014, the Canadian Cancer Clinical Trials Network (3CTN) is a collaboration of 51 cancer centres across eight provinces, aligned to improve access, recruitment, and performance of multicentre academic cancer clinical trials (ACCTs) on the 3CTN Portfolio. With an annual budget of \$4.3 million, the Network provides core and incentive funding to support research capacity and facilitates the development and sharing of resources and best practices to enhance trial conduct. 3CTN's impact has been evaluated across three cumulative phases: foundational growth (2014–2018), structural optimization (2018–2022), and system innovation (2022–2027). **Methods** Recruitment and performance data from April 2015 to March 2025 were assessed against pre-3CTN baseline metrics (2011–2013) and across program phases. A standardized framework described Portfolio trials and evaluated outcomes and impact measures across the trial lifecycle. Key initiatives were also reviewed. **Results** Between 2015 and 2025, 43,167 patients were enrolled in 592 active Portfolio trials, with centres achieving a 130% increase over baseline in 2021. Of 342 completed trials, 46% reported positive outcomes, 32% articles in high-impact journals, and 6% influenced clinical practice. Since 2020, metrics have captured participation from adolescents/young adults and patients in rural and remote areas. Network-wide informatics and initiatives such as the Canadian Remote Access Framework for Clinical Trials and Trial Finder enhanced access and visibility. Cross-provincial and pediatric–adult collaboration strengthened the national research environment. **Conclusions** 3CTN has enhanced ACCT performance and access. Continued funding beyond 2027 is essential to sustain progress and strengthen Canada's ACCT ecosystem.

LAY SUMMARY

A Stronger National Research System: By connecting cancer centres across provinces and supporting both adult and pediatric trials, 3CTN has built a stronger, more collaborative research system in Canada. Network-wide informatics and initiatives have enhanced access and visibility for trials. **Better Trial Results and Research Impact:** Over 43,000 patients joined nearly 600 trials, with many studies leading to important results, publications in top journals, and even changes in how cancer is treated. **Increased Access for Clinical Trials:** Since 2014, 3CTN has helped more people across Canada—including those in rural areas—join cancer clinical trials by improving access and making it easier to find and participate in studies. The Network also supports participation through site-level incentives.

Concurrent: C4-Clinical Research & Radiotherapy

Monday, November 3, 2025, 12:45:00 PM - Hyatt Imperial 8

Glutamate and Glutamine (Glx) Measurements in High Grade Glioma in Response to Radiation Therapy: A Magnetic Resonance Spectroscopic Imaging Study at 3 T

Presenter: Atiyah Yahya, Cross Cancer Institute, University of Alberta

Other authors: John Amanie, Samir Patel, Wilson Roa, B. Gino Fallone, Albert Murtha

ABSTRACT_ID#2582

ABSTRACT

Collective levels of tumour glutamate and glutamine (Glx) in response to radiation treatment (RT) has been previously measured in animal studies. In this work, we employ magnetic resonance spectroscopic imaging (MRSI) at 3 T to investigate Glx levels, normalized to Cr (creatine), in high grade glioma patients pre- and mid-RT. The protocol was approved by our local ethics board. Seven patients received 60 Gy in 30 fractions while two others (elderly) received 40 Gy in 15 fractions. MRSI scans were acquired before RT (baseline) and then mid-RT (approximately after 3-5 weeks) using a sequence tailored to target the 3.75 ppm Glx protons. Peak areas for the 3.75ppm Glx and 3.9ppm Cr peaks were measured from voxels in both the tumour and corresponding normal tissue regions. Average Glx/Cr values were calculated in the glioma and healthy tissue for each patient. In one patient, metabolites were not detected in the glioma, possibly due to necrosis. Glx/Cr values were averaged over the other eight patients. The average ($\pm$ standard deviation) values in the gliomas and healthy tissue were 1.40 ($\pm$ 0.50) and 0.48 ($\pm$ 0.12), respectively. Mid-RT, the average value dropped to 0.97 ($\pm$ 0.26) in glioma but remained similar, 0.46 ($\pm$ 0.06), in healthy tissue. Glx/Cr levels in glioma were on average about three times higher than in healthy tissue at baseline and were reduced by about 31 % in glioma at mid-RT. Variation of Glx/Cr levels with RT suggests that it could potentially serve as a biomarker for assessing response to RT.

LAY SUMMARY

Collective levels of glutamate and glutamine relative to that of creatine (Glx/Cr) were found to be higher in high grade glioma compared to healthy brain tissue. Glx/Cr levels decreased in glioma in response to radiation treatment (RT) Glx/Cr could potentially serve as a biomarker for assessing glioma response to RT.

Concurrent: C5-Environmental Impacts on Cancer

Monday, November 3, 2025, 12:45:00 PM - Hyatt Doll/Herald

Wavelength-resolved measures of outdoor artificial light at night and breast cancer risk

Presenter: Parveen Bhatti, BC Cancer Research Institute

Other authors: Emma Quinn, Jaclyn Parks, Thomas Gagne, Cheryl Peters, Christopher McLeod, Grace Shen-Tu, Jennifer Vena, Martin Aube

ABSTRACT_ID#2404

ABSTRACT

Background. By suppressing melatonin secretion, outdoor artificial light at night (ALAN) may increase the risk of breast cancer. Most studies of ALAN and breast cancer have not accounted for wavelength of light even though suppression of melatonin is influenced by both light intensity and wavelength. **Objectives.** We evaluated the association of multiple ALAN measures, including those that captured wavelength, with breast cancer risk. **Methods.** We pooled data from two prospective cohort studies (n=24,793 female participants, 674 incident breast cancer cases). ALAN measures were generated from images taken from the International Space Station and linked to participant residential addresses. Measures included visual radiance (i.e., brightness), melatonin suppression index (MSI), which quantifies the extent to which light spectra suppress melatonin, and the product of visual radiance and MSI (impact MSI). Cox proportional hazards regression was used to generate hazard ratios with 95% confidence intervals (CI) for the associations between ALAN metrics and breast cancer incidence. Analyses were adjusted for breast cancer risk factors and various built environment factors. **Results.** In fully adjusted analyses restricted to participants that self-reported light entering their rooms while sleeping, a 1.53-fold increased hazard of breast cancer was observed when comparing the highest to lowest tertile of impact MSI (95% CI: 1.18-1.98). Increased hazards of invasive ductal, luminal A, and postmenopausal breast cancers were also observed. **Discussion.** Our results demonstrated that outdoor ALAN is associated with increased risk of breast cancer and that both intensity and wavelength of light should be considered when evaluating ALAN exposures.

Concurrent: C5-Environmental Impacts on Cancer

Monday, November 3, 2025, 12:45:00 PM - Hyatt Doll/Herald

Mobilizing sun safety messages in the construction industry

Presenter: Lindsay Forsman-Phillips, University of British Columbia (CAREX Canada)

Other authors: Raissa Shrestha, Joanne Telfer, D. Linn Holness, Sunil Kalia, Thomas Tenkate, Cheryl E Peters

ABSTRACT_ID#2587

ABSTRACT

Skin cancer is on the rise in Canada. Outdoor workers are at particularly high risk of exposure to solar ultraviolet radiation (UVR), and there are 1.7M outdoor workers in Canada. Construction workers are at an increased risk due to long outdoor working hours and limited ability to change activities during peak exposure times. Understanding the risk of exposure to solar UVR and practicing sun safe behaviours can help reduce the associated health risks; however, there is a lack of awareness about this occupational hazard. Most sun protection strategies, including messaging and resources, largely target the general population. To address this gap, we developed a core set of evidence-based, practical, harm-reducing sun safety messages that are likely to resonate with outdoor construction workers. The primary goal was to mobilize the sun safety messages and influence behaviour change among outdoor workers in the construction industry in British Columbia. We convened and surveyed workers and experts from industry, labour, exposure assessment, epidemiology, dermatology, and policy, building consensus through sequential rounds of voting to ensure that the sun safety messages were appropriate for construction workers. We also identified knowledge translation (KT) products that would be most effective in mobilizing the sun safety messages. We co-developed evidence-based KT products with key stakeholders in the construction industry, and produced a series of workplace sun safety images that reflect the diversity of outdoor workers in Canada. We continue to coordinate efforts to mobilize the KT products and help build a culture of sun safety in the workplace.

LAY SUMMARY

We successfully produced a set of 28 evidence-based, expert-approved, and stakeholder workshopped messages, tailored to be feasible and practical for outdoor construction workers. These fall under the categories of sun safety facts, recommended actions (for skin, eye, and heat illness protection), and tips (for shade, clothing, sunscreen, and eye protection). Our collaboration and execution of the KT plan with key stakeholders led to the creation of a publicly available toolkit consisting of several KT products, including posters on recommended personal protective equipment (PPE) use and shade utilization, and a toolbox talk. The sun safety images showcasing the diversity of outdoor workers in Canada from various industries (i.e. agriculture, recreation, construction) can be further customized with sun safety messages and organizational logos and incorporated into workplace sun safety plans and programs as needed. Moving forward, we will continue to leverage our relationships and explore potential partnerships with regulators, occupational health and safety organizations, and other relevant stakeholders to further disseminate the messages and KT products.

Concurrent: C5-Environmental Impacts on Cancer

Monday, November 3, 2025, 12:45:00 PM - Hyatt Doll/Herald

Lifetime occupational and domestic environmental exposures and breast cancer risk among women in the CARTaGENE study

Presenter: Vikki Ho, Sainte-Justine Hospital Research Center, CARTaGENE, Montréal

Other authors: Mengting Xu, Simon Gravel, Guillaume Lettre

ABSTRACT_ID#3127

ABSTRACT

Objective: To investigate associations between lifetime occupational and domestic environmental exposures and breast cancer risk in women. **Methods:** We conducted a nested case-control study (278 cases; 5,285 controls) within the Quebec-based CARTaGENE prospective cohort, leveraging lifetime occupational and domestic exposure data. Occupational exposures to >200 agents were assessed using a female-specific version of the Canadian Job Exposure Matrix (CANJEM), applied to self-reported jobs. Domestic exposure durations to traditional heating sources (gas, oil, wood, and coal) were collected from questionnaires. Unconditional logistic regression adjusted for age, income, ethnicity, and other established breast cancer risk factors. **Results:** Occupational exposure to calcium carbonate (OR = 1.94, [1.33–2.77]) and cosmetic talc (OR = 1.72, [1.08–2.64]) was associated with increased breast cancer risk. Prolonged domestic exposure (>10 years vs. 0 years) to gas (OR = 1.95, [1.20–3.03]), oil (OR = 1.55, [1.14–2.09]), and coal (OR = 6.43, [2.36–14.88]) heating sources also showed elevated risks. No significant associations were found for other occupational or domestic exposures examined. **Conclusion:** Preliminary findings suggest that occupational (calcium carbonate, cosmetic talc) and domestic (gas, oil, coal heating) exposures may contribute to breast cancer risk in this Quebec population, warranting further investigation.

LAY SUMMARY

1. Occupational Exposures: Lifetime occupational exposure to calcium carbonate and cosmetic talc were associated with elevated breast cancer risks.
2. Domestic Exposures: Prolonged domestic exposure to gas/oil/coal heating sources were associated with elevated breast cancer risks.

Concurrent: C5-Environmental Impacts on Cancer

Monday, November 3, 2025, 12:45:00 PM - Hyatt Doll/Herald

Investigating the Impact of Environmental Exposures on Lung Cancer Promotion

Presenter: Emilia Lim, The University of British Columbia

Other authors: Lucas Fung, Brenden Joseph, Theodora Lo, Taysia Nikaido-Landry, Honor Cheung, Anna McGuire, Julia Naso

ABSTRACT_ID#2295

ABSTRACT

BACKGROUND: A mechanistic basis for the formation of non-small cell lung cancer (NSCLC) in never smokers is unclear. Air pollution particulate matter (PM) is known to be associated with the risk of NSCLC, however a direct cause and mechanism of pollution remain elusive. While pollution-associated anthracotic pigment (black carbon deposits) can be detected in Hematoxylin and Eosin (H & E) slides of lung tissue and are associated with oncogenic clonal expansions, it is unclear how they contribute to lung cancer initiation and development. **METHODS:** To quantify anthracotic pigment in 32 archival H & E slides, we developed an automated pixel classifier method using QuPath. In 4 cases, to further understand the transcriptional dysregulation associated with anthracosis, we used NanoString DSP GeoMx spatial transcriptomics to examine regions of interest (ROI) close to (n=96) and distant (n=95) from anthracotic pigment. **RESULTS:** We observed a significant correlation between the quantity of anthracosis and 3-year PM_{2.5} exposure estimated based on patient residence ($R=0.25$, $p<0.05$). We noted a trend toward an association between lower levels of anthracosis and a less-aggressive lepidic predominant growth pattern ($p<0.05$). Preliminary analyses of our DSP GeoMx data suggested that anthracosis tends to disrupt centrilobular lung tissue regions. Ongoing work involves identifying dysregulated genes that may be targeted to prevent pollution-promoted lung cancer. **CONCLUSION:** Our results indicate that anthracotic pigment may be representative of geographically estimated PM_{2.5} exposure, and suggest that its quantification can enable research into pollution-promoted lung cancer research using archival slides.

LAY SUMMARY

- Air pollution can promote lung cancer, but we are only beginning to understand how this occurs.
- We have found a way to automatically quantify pollution-related black carbon pigments in lung tissue slides routinely obtained by pathologists at lung cancer surgeries. This enables us to study the consequences of pollution exposure in large numbers of patients.
- We observed that the quantity of these pigments is associated not only with geographical estimates of pollution exposure but also with expansions of cells with cancer-causing mutations. We noted a trend toward a low quantity of pigments being associated with a less aggressive type of lung cancer.
- Ongoing work involves using spatial transcriptomics technology to identify which cellular processes these pigments disrupt to promote cancer.

Concurrent: C5-Environmental Impacts on Cancer

Monday, November 3, 2025, 12:45:00 PM - Hyatt Doll/Herald

Environmental Determinants of Cancer Disparities in a Black Canadian Community

Presenter: Paola A. Marignani, Dalhousie University

Other authors: Ariel Provo, Nathalie Yuen, Jinhong Kim, Samantha Wilson, Jong Sung Kim, Ingrid Waldron, Juliet Daniel

ABSTRACT_ID#3180

ABSTRACT

According to the Canadian Cancer Society, in 2024, an estimated 247,100 new cancer cases were diagnosed, with 88,100 cancer deaths. Notably absent from these statistics, is data about cancer rates amongst Black, and other ethnic Canadian communities. These observed disparities are often attributed to a complex interplay of socioeconomic status, lifestyle, environmental factors, and genetics. Objective: Our study focused on the marginalized Black community of Shelburne, Nova Scotia that is located adjacent to a landfill used for military and hospital waste. Shelburne has reported an unusually high incidence of different cancers. The objective of our study was to investigate the environmental impact on the high cancer incidence and mortality within this community. Methods and Results: Consent was obtained before the collection of biospecimens (buccal swabs and toenails). Biospecimens were prepared for analysis of epigenetic marks and heavy metals associated with carcinogenesis. Community members also answered a lifestyle questionnaire that included living distance from the landfill, how long they lived in South Shelburne, smoking status, age, sex and gender. This demographic data were aligned with geographical data related to landfill proximity. Summary: Our first-of-its-kind study provides novel pioneering data about environmental impact and epigenetics on cancer disparities in Black Nova Scotians. Our study creates a framework for transformative, inclusive biomedical research, creating a model for future large-scale studies of health disparities in other chronic diseases across Canada. JD, IW and PM are supported by the New Frontiers Research Fund-Exploration

LAY SUMMARY

- Lack of statistics associated with cancer rates in Black Canadians High rates of cancer in marginalized communities.
- Epigenetic marks associated with carcinogen exposure were identified in biological samples from Shelburne Black community members.

Concurrent: C6: First Nations, Inuit and Métis Cancer Prevention and Screening

Monday, November 3, 2025, 12:45:00 PM - Hyatt Stephen AB

Enhancing Alberta Métis Settlements Participation in Cancer Screening: A Distinctions-Based, Community-Grounded Approach

Presenter: S. Trevor Baxter, Primary Health Alberta/Alberta Health Services

Other authors: Jessica Smith, Angeline Letendre, Brenda Roland

ABSTRACT_ID#2735

ABSTRACT

Métis Settlement communities in Alberta experience systemic barriers in accessing culturally appropriate cancer screening services. The Enhancing Alberta Métis Settlements Participation in Cancer Screening (EAMSS) project applies a distinctions-based, Indigenous-led approach to address these disparities that recognizes the unique histories, rights, and needs of Métis Settlement communities. The project aims to co-develop a Métis Settlements Cancer Prevention and Screening Strategy by: (1) gathering evidence to inform the strategy; (2) piloting culturally appropriate screening innovations; and (3) identifying Indigenous-specific indicators to enhance screening participation. This presentation will share how distinctions-based, community-led approaches have addressed inequities in cancer screening and advanced sustainable, community-driven solutions for Métis Settlements in Alberta. Key project components, including the application of Indigenous methodologies, community-based research principles, and Indigenous data governance will be described. Partnerships with Elders, healthcare providers, and community leadership have guided the integration of cultural protocols such as tobacco offering, smudging, ceremony, and gift-giving throughout the research process. Multiple data collection activities—environmental scans, literature reviews, sharing circles, community gatherings, and healthcare provider surveys—have supported meaningful community engagement that informed the development of new innovations. The pilot of these innovations are expected to demonstrate promising practices for improved screening participation, strengthened capacity, and an informed comprehensive, distinctions-based Cancer Prevention and Screening Strategy. EAMSS focus on positive healthy lifestyle choices will further reinforce these community-led solutions in promoting health and preventing cancer.

LAY SUMMARY

Métis Settlement communities in Alberta experience unique barriers to cancer screening, shaped by historical, systemic, and cultural factors. The EAMSS project partnered directly with Métis Settlements to co-develop a distinctions-based Cancer Prevention and Screening Strategy that integrates Indigenous knowledge, cultural protocols, and community leadership. Through Indigenous-led research methods, pilot interventions improved screening participation, strengthened community capacity, and promoted positive healthy lifestyle choices to support long-term cancer prevention.

Concurrent: C6: First Nations, Inuit and Métis Cancer Prevention and Screening

Monday, November 3, 2025, 12:45:00 PM - Hyatt Stephen AB

Knowledge into Action: Implementation Testing of Cancer prevention and Screening Practices Grounded in First Nation Knowledge Transfer Practices

Presenter: Barbara Frazer, Alberta First Nations Information Governance Center

Other authors: Lea Bill

ABSTRACT_ID#3349

ABSTRACT

Inherently, cancer prevention and screening tools that embed First Nation cultural knowledge and teachings have a greater potential to effect prevention and screening participation and improve cancer outcomes within First Nations communities. The First Nations Cancer Prevention and Screening Practices Projects (FN Practices Project Phase I and II) were a collaborative partnership with six Alberta First Nations. Supported by Alberta Health Services – Alberta Cancer Prevention Legacy Fund/Cancer Prevention and Screening Innovation and the Alberta First Nations Information Governance Centre, Phase II of the FNCPSF partnered with three First Nation communities for implementation testing of a Community Support Team Model (CSTM). During the proof-of-concept Phase I, key guides and learning resources were developed to accompany the CSTM implementation and direct the approach for improving cancer prevention and screening within/by First Nations communities. Phase II tested the CSTM within three First Nations communities. A model for action planning, aimed to promote healthy lifestyle behavior's while simultaneously decreasing significant gaps related to risk factors for cancer. For example, engagement-oriented community activities, demonstrates the essential role of a Community-Action-Team as the implementors. The CSTM aligned actions with primary values and principles with a First Nation world view while respecting the diversity of kinship and knowledge systems among First Nations. This project facilitated the testing of a guide prepared from a First Nation knowledge base to demonstrate the strength of knowledge to action from a community-driven perspective that positions intergenerational knowledge transfer principles into engagement processes and cultural concepts that support cancer prevention and screening.

LAY SUMMARY

The First Nations Community Support Team Model (CSTM) is deemed by First Nation community partners as a highly transferrable approach.

- Engagement-oriented activities that follow First Nations ways of being were incorporated as key activities, demonstrating the strength of the CSTM as First Nation customized community-based resources using their language as a equity capacity-building tools, by integrating traditional teachings. · The creation of guides were recognized as a major achievement toward improved cancer prevention and screening action planning within/by Alberta First Nations communities.
- The Guide to Preparing Indigenous Communities for Cancer Prevention: A Pathway to Healthier Communities (©2019 AHS, Alberta Cancer Prevention Legacy Fund and Canadian Indigenous Nurses Association) and the Indigenous Community Readiness Model for Cancer Prevention and Screening: A Pathway Towards Community Wellness (Adaptation of the Community Readiness Model for Community Change for use in First Nations, Métis, Inuit Communities, Version 2, 2020. Alberta Cancer Prevention Legacy Fund & Canadian Indigenous Nurses Association, Alberta Health Services, Alberta) was developed to better understand First Nations community preparedness to engage in cancer prevention and screening activities.
- Emphasis on strategic partnerships and Elder/Knowledge Holder support is key to ground strategies and action in culturally relevant cancer prevent and screening solutions.

Concurrent: C6: First Nations, Inuit and Métis Cancer Prevention and Screening

Monday, November 3, 2025, 12:45:00 PM - Hyatt Stephen AB

Exercising Self-Determination & Governance through Métis Culture, Knowledge & Science Research to Build Youth Capacity to Prevent Cancer

Presenter: KD King, University of Alberta

Other authors: Angeline Letendre, Peter Hutchinson

ABSTRACT_ID#3263

ABSTRACT

The Métis are one of three distinct Indigenous Peoples recognized in Canada. Under Canadian Law (1982 Constitution Act, Section 35), Indigenous Peoples are recognized and affirmed as treaty rights holders, (for those who signed treaty), and as rights-holders for future agreements. Section 35 also confirms that Indigenous peoples have rights that predate colonization and the formation of Canada. These include rights to land, culture, governance, and cultural practices. Unlike First Nations and Inuit, whose rights and relationships with the federal government have been more explicitly recognized in legislation and treaties, the Métis have historically faced marginalization and legal ambiguity in federal policy and law. The Métis Settlements Cancer Prevention project provides an exemplary framework for Métis self-determination using a Métis-led community-based participatory approach to cancer research in Canada. From the inception of the project, through to the ethics, funding agreements and legal frameworks through which the project has operated, Métis researchers and their Settlement partners have had near-total control over every aspect of the research. This presentation will explore a Métis Settlement intergenerational knowledge transfer model grounded in Indigenous science using Cree Michif language to describe and define concepts. The objectives are to: describe the governance model that provides leadership for the project, share how culture and language provide the fertile earth from which the project has grown, and explain how the Métis Settlements' and research team's graduate trainee's capacity building needs were realized throughout the project delivery.

LAY SUMMARY

- A novel process for sustainable Métis self-determination in cancer research and governance is shared.
- Ways of enhancing intergenerational knowledge transfer related to cancer prevention based in Métis science is shared.
- Building capacity in communities requires real investment in communities to meet their cancer prevention needs.

Concurrent: C6: First Nations, Inuit and Métis Cancer Prevention and Screening

Monday, November 3, 2025, 12:45:00 PM - Hyatt Stephen AB

Métis Culture, Knowledge & Science as a Framework for Building Youth Capacity to Prevent Cancer: A Métis Settlement Panel Presentation

Presenter: Angeline Letendre, Canadian Indigenous Nurses of Canada (CINA)

Other authors: Peter Hutchinson, Keith King, Edna Desjarlais, Julia Auger, Tom Durocher, Elmer Ghostkeeper, Jessica Smith

ABSTRACT_ID#3191

ABSTRACT

This presentation brings together a panel of Youth and Elder/Knowledge Keepers participating in the Canadian Partnership Against Cancer funded initiative: “Métis Culture, Knowledge & Science as a Framework for Building Youth Capacity to Prevent Cancer: A Métis Settlement Project”. Representing 3 Métis Settlement communities in Northern Alberta, Elders play key roles throughout the project to address the overall health and wellbeing of Youth by providing mentorship grounded in Indigenous wisdom, knowledge, science, and relationship between land and language. Youth from each partner community act as Ambassadors through engaging in activities designed to support the creation of learning and teaching sessions to be co-led by Youth and Elders during a series of Health Camps. These sessions are expected to focus on lifestyle changes, behaviours and ethics that promote health and prevent cancer among Youth based on the transfer of Métis-specific knowledge and concepts, inclusive of beliefs, values, behaviours, relationships and inter-relationships. These concepts will be discussed as aspects of a Métis Intergenerational Knowledge Transfer Model, providing a foundation for teaching a new way of decision-making to Youth, promoting health, and preventing cancer. Objectives include: 1) Project co-leads will provide an overview of the project, 2) Elders will discuss how and why the transfer of Indigenous knowledges in relation to land and language are critical to healthy decision-making by Métis Youth, 3) Métis Youth describe learning and teaching sessions presented during a Health Camp and how their work aspires to support other Youth in living a lifestyle that promotes health and prevents cancer.

LAY SUMMARY

1. Métis Science and Culture are the foundation of this research with Métis Settlement Youth.
2. Elders ground teachings in land and language to demonstrate a new way of healthy decision-making to Métis Youth.
3. Métis Youth act as Ambassadors in presenting learning and teaching sessions during a Health Camp to support other Youth in living a lifestyle that promotes health and prevents cancer.

Concurrent: C6: First Nations, Inuit and Métis Cancer Prevention and Screening

Monday, November 3, 2025, 12:45:00 PM - Hyatt Stephen AB

Living Well Together: Integrating Two Perspectives into Cancer Prevention Programming for Indigenous Communities

Presenter: Samantha Morris, Elliott Fiddler, and Jason Mawakeesick, Lakehead University

Other authors: Ian Newhouse, Jason Mawakeesic, Elliot Fiddler

ABSTRACT_ID#3082

ABSTRACT

Despite substantially higher rates of cancer among Indigenous people, there is limited evidence on culturally relevant health initiatives to prevent cancer. This project employed a community-based participatory action research (CBPAR) approach to co-develop a culturally tailored cancer prevention initiative with a remote First Nations community. Building on insights from earlier program phases, Phase III culminated in a one-day community health event, Living Well Together, which promoted cancer prevention and healthy living. Although no significant changes in cancer prevention knowledge were observed, participant feedback indicated high satisfaction and an increased awareness of healthy practices. In Phase IV, in-depth interviews with two community champions provided critical reflections on the event's impact, identified strengths and gaps, and emphasized the importance of community ownership in health programming. Key themes included the value of two-eyed seeing, the need for sustained community engagement, and culturally grounded approaches to physical activity and wellness. These findings highlight the importance of embedding community narratives in prevention strategies to enhance cultural alignment, sustainability, and impact. This work offers valuable insights for future program development to ensure cancer prevention efforts remain responsive, respectful, and rooted in Indigenous contexts.

LAY SUMMARY

Building programs with the community matters. We worked with a remote First Nations community to create a cancer prevention program that respects and reflects their culture and knowledge. This kind of partnership, called community-based participatory action research, helps ensure that health programs are meaningful and locally relevant. Cultural connections strengthen health messaging. Our Living Well Together event combined both Indigenous and Western ways of knowing (a concept called two-eyed seeing) to promote healthy living and cancer prevention. While the event did not significantly change knowledge about cancer prevention right away, it increased awareness of healthy practices and was very well received by participants. Community leadership and ownership are key for lasting impact. Interviews with local community champions showed that programs work best when they are led and owned by the community itself. They also stressed the importance of ongoing engagement and including cultural traditions, such as land-based activities and traditional wellness practices, in future health programming.

Concurrent: D1-Precision oncology continuum: From molecular markers to therapeutic resistance pathways

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 4/6

Proteomics: complementary insights in precision oncology

Presenter: Kirsty M. Ferguson, University of British Columbia

Other authors: Verena Goebeler, Enes K. Ergin, Agustina Conrrero, Georgina D. Barnabas, Tariq A. Bhat, Pascal Leclair, Jonathan W. Bush, C. James Lim, Philipp F. Lange

ABSTRACT_ID#3073

ABSTRACT

Children and young adults with cancer face unique challenges in access to the latest scientific advances, with approved therapies historically trailing behind adult cancers. The Canadian network ACCESS (Advancing Childhood Cancer Experience, Science and Survivorship) is tackling this through the driver project PROFYLE (PRrecision Oncology For Young peopLE), where molecular profiling is used to find personalized therapies for children, adolescents and young adults whose cancers have returned or are resistant to conventional treatments. Identifying alterations in tumour DNA and RNA has already shown great benefit in guiding targeted therapies for patients enrolled in PROFYLE. However, as most drugs target proteins, proteomic analyses could provide complementary information closer to the patient's individual cancer biology, as well as identify treatment options where tumors are unresponsive to genome-guided therapies. Here, we present proteomics as a complementary technique in precision oncology for paediatric cancers. We highlight a recent case study where proteomics and personalized tumour models found a drug target in a young person's rare metastatic tumour, not seen by DNA or RNA analyses. This identified a treatment option within an actionable timeframe, demonstrating proof-of-concept that proteomics can support clinical decision making. We outline our multi-disciplinary process, from bedside-to-bench and back again, and the optimization steps needed to integrate proteomics into precision oncology pipelines and bring new molecular insights to more patients across Canada.

LAY SUMMARY

PRrecision Oncology For Young peopLE (PROFYLE) is a pan-Canadian network using molecular profiling to improve treatments for children, adolescents and young adults with difficult-to-treat cancers. Molecular profiling is traditionally focused on the study of genes, the instructions to make proteins which are the functional building blocks of our cells. Studying proteins (proteomics) directly may provide complementary information on what is driving tumours; we highlight a case where proteomics identified a drug target in a young patient's tumour and detail our plans to expand proteomics to reach more childhood cancer patients.

Concurrent: D1-Precision oncology continuum: From molecular markers to therapeutic resistance pathways

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 4/6

Highly selective inhibition of ALDH1A3 impedes breast cancer growth and metastasis by blocking ALDH1A3-driven transcriptional programs

Presenter: Maya MacLean, Dalhousie University

Other authors: Bianca Bernardoni, Wasundara Fernando, Giovanni Petrarolo, Ilaria D'Agostino, Cheryl Dean, Jaganathan Venkatesh, Christopher Hughes, Kerry Goralski, Geetha Subramanian, Raj Pranap Arun, Olivia Walker, Hannah Cahill, Concettina La Motta, Paola Marcato

ABSTRACT_ID#3212

ABSTRACT

Triple-negative breast cancer (TNBC) is an aggressive subtype of breast cancer with limited treatment options and poor survival outcomes. Aldehyde dehydrogenase 1A3 (ALDH1A3) is a key driver of tumor growth, metastasis, and chemoresistance across several cancers, including TNBC, glioblastoma, melanoma, and lung cancer. Despite its clinical relevance, no ALDH1A3-selective inhibitors are currently approved for use in clinics. We developed a novel, first-in-class small molecule that selectively inhibits ALDH1A3 and evaluated its therapeutic potential in preclinical models of TNBC. In vitro assays using TNBC cell lines (MDA-MB-231, MDA-MB-468, HCC1806) demonstrated potent ALDH1A3 inhibition ($IC_{50} = 2 \text{ nM}$) with no detectable activity against the closely related ALDH1A1 isoform. RNA sequencing confirmed high selectivity, showing targeted downregulation of ALDH1A3-driven transcriptional programs. In vivo, daily intraperitoneal administration of the inhibitor significantly reduced tumor burden and lung metastases in MDA-MB-231 tumor-bearing NOD-SCID mice, with no observable toxicity. Pharmacokinetic studies revealed favorable biodistribution, including penetration into tumor tissue, lungs, liver, and brain, with a half-life exceeding 8 hours and sustained physiological concentrations beyond 24 hours following both intraperitoneal and oral dosing. These findings suggest the compound achieves therapeutically relevant exposure and may be suitable for oral administration. Collectively, our data support the continued development of this ALDH1A3 inhibitor as an oral precision oncology treatment, with the potential to suppress metastasis and recurrence in ALDH1A3-expressing tumors.

LAY SUMMARY

We developed a new drug that targets a specific protein (ALDH1A3) linked to the spread and treatment resistance of aggressive cancers like triple-negative breast cancer, lung cancer, and melanoma. This protein helps cancer cells survive and grow, and until now, there have been no approved drugs that target it directly. Our drug works in lab models at extremely low doses and doesn't affect similar proteins which suggests it could have fewer side effects compared to less targeted treatments. In animal studies, the drug successfully shrank breast cancer tumors and stopped them from spreading to the lungs, and it did so without causing harm to the mice. It also reached key organs like the brain, liver, and lungs, and stayed active in the body for over 24 hours, showing potential for once-a-day oral dosing.

Concurrent: D1-Precision oncology continuum: From molecular markers to therapeutic resistance pathways

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 4/6

THSD4 is a novel mediator of T cell exclusion and anti-PD-1 resistance in breast cancer

Presenter: Paola Marcato, Dalhousie University

Other authors: Olivia Walker, Marie-Claire Wasson, Vishnupriyan Kumar, Sarah Nersesian, Jaganathan Venkatesh, Vishnu Vijayan, Raj Pranup Arun, Hannah Cahill, Wasundara Fernando, Elizabeth Baker, Margaret Dahn, Penelope Barnes, Daniel Gaston, Gregory Knapp, Jennifer Melvin, Shashi Gujar, Jeanette Boudreau, Gillian Bethune

ABSTRACT_ID#3178

ABSTRACT

Breast cancer remains the most prevalent cancer among women, with hormone receptor-positive (HR+) tumors accounting for approximately 70% of cases. While the immune checkpoint inhibitor (ICI) anti-programmed cell death 1 (PD-1) pembrolizumab has demonstrated efficacy in triple-negative breast cancers (TNBCs), its benefit in HR+ subtypes is limited. ICI resistance in breast cancer is largely due to the “cold” tumor immune microenvironment characterized by low tumor-infiltrating lymphocytes (TILs). To identify novel genetic determinants of immune exclusion and pembrolizumab resistance, we analyzed multi-omics and clinical datasets from the I-SPY2 trial and The Cancer Genome Atlas (TCGA), focusing on genes associated with low T cell infiltration and poor response to pembrolizumab. We identified thrombospondin type-1 domain containing 4 (THSD4), as a top candidate. THSD4 expression was significantly elevated in breast tumors with low T cells and in breast cancer patients exhibiting resistance to pembrolizumab, particularly within the HR+ subtype. THSD4 expression is enriched in HR+ breast cancers. Validation in local patient cohorts using RNA sequencing and multiplex immunofluorescence confirmed that both high THSD4 expression and anti-THSD4 antibody staining correlated with reduced T cell infiltration in the tumor epithelium and associations with poorer clinical outcomes. Functional studies in a syngeneic mouse HR+ tumor model demonstrated that THSD4 promotes an immunosuppressive tumor microenvironment, with reduced T cells, resistance to anti-PD-1, and altered collagen fibre abundance. Collectively, these findings establish THSD4 as a prognostic biomarker of pembrolizumab resistance and a potential therapeutic target to enhance immunotherapy efficacy in breast cancer.

LAY SUMMARY

1. From patient datasets, we identified a novel gene called THSD4 that is associated with poor immune activity in breast tumours and resistance to immunotherapy. This association was strongest in a subtype of breast tumours called hormone receptor-positive (HR+) breast cancers which account for approximately 70 percent of all breast cancer cases in Canada and are typically not eligible for immunotherapy due to poor immune activity.
2. In Nova Scotian breast patient tumour samples, high THSD4 expression was associated with low numbers of a critical type of immune cell called T cells, which are critical for drugs like immunotherapy to work as they can specifically target and kill cancer cells.
3. In a hormone receptor-positive mouse model of breast cancer, reducing THSD4 and treating with immunotherapy resulted in significantly smaller tumours compared to controls with higher immune cell proportions, including activated T cells. These activated T cells are important because immunotherapy resistance occurs when immune cells cannot infiltrate a tumour or when those that do become inactive. This study shows that reducing THSD4 in breast cancer may enhance immunotherapy effectiveness because it allows for the tumours to become infiltrated with cancer-fighting immune cells.

Concurrent: D1-Precision oncology continuum: From molecular markers to therapeutic resistance pathways

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 4/6

Uncovering the Molecular Drivers of Liver Metastasis in Gastroenteropancreatic Neuroendocrine Tumours (GEP-NETs)

Presenter: William You, Sunnybrook Research Institute

Other authors: Zoey Wang, Lufei Liu, Lina Chen, Weei-Yuarn Huang, Chris Lowden, Daniel Schramek, Iacovos Michael

ABSTRACT_ID#2683

ABSTRACT

Gastroenteropancreatic neuroendocrine tumours (GEP-NETs), including pancreatic (PanNETs) and small intestinal NETs (SINETs), are rare malignancies, yet up to 60% of patients present with synchronous liver metastases at diagnosis. While driver mutations have been identified for primary tumour development, the molecular mechanisms underlying metastatic progression remain poorly understood. Building on our previous findings implicating phenotypic plasticity in GEP-NET advancement, we hypothesize that GEP-NET metastasis is driven by distinct molecular alterations acquired during tumour evolution. To identify candidate metastasis regulators, we performed single-cell RNA sequencing on patient-matched primary tumours and liver metastases, revealing 28 differentially expressed genes (DEGs) common to both PanNET and SINET subtypes. To functionally interrogate these candidates, we conducted in vivo CRISPR activation (CRISPRa) and knockout (CRISPRko) enrichment screens targeting upregulated and downregulated DEGs, respectively. Two Cas9-expressing GEP-NET cell lines were transduced with pooled gRNA libraries and introduced into SCID mice via intrasplenic injection to model liver metastasis. Tissues have been harvested, and next-generation sequencing (NGS) is in progress to identify enriched or depleted sgRNAs in metastatic lesions. Concurrently, we are initiating mechanistic studies on the Cocaine and Amphetamine-Regulated Transcript (CART), an upregulated gene in liver metastases in both GEP-NET subtypes, which encodes a neuropeptide. We are generating CART knockout cell lines using the qTAG Cas9 system, and inducible CART loss- and gain-of-function cell lines to modulate its expression and assess its role in metastatic progression. Collectively, this study integrates patient transcriptomics with functional genomics and phenotypic analysis to uncover drivers of GEP-NET liver metastasis and advance therapeutic discovery.

LAY SUMMARY

GEP-NETs often spread to the liver early, but we don't know why. Gastroenteropancreatic neuroendocrine tumours (GEP-NETs) are rare cancers that often spread to the liver (metastasis) by the time they are diagnosed. This makes them much harder to treat, but we still do not fully understand the biological reasons for the metastasis. We use innovative tools to find genes that may drive metastasis. By comparing patients' original tumour and liver metastases samples, we identified a set of genes that change during the spread of cancer. We then used CRISPR gene editing in mouse models to test which of these genes facilitate tumours grow or spread to the liver. We are now studying a gene called CART as a possible new target. One gene, called CART, was consistently more active in liver metastases than in the original tumours. We are now using genetic tools to turn this gene on or off in lab models to see how it affects cancer spread. This could help reveal new ways to block metastasis and improve outcomes for patients with GEP-NETs.

Concurrent: D2-From risk assessment, screening and surveillance to survivorship

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 1/2/3

Establishing an academic hospital based liquid biopsy platform for MRD testing in breast cancer patients

Presenter: Mark Basik, Lady Davis Institute, McGill University

Other authors: Adriana Aguilar-Mahecha, Talia Roseshter, Anna Klemantovich, Josiane Lafleur, Cathy Lan, Luca Cavallone, Jean-François Boileau, Manueal Pelmus

ABSTRACT_ID#3271

ABSTRACT

The detection of minimal residual disease (MRD) requires capturing very minute amounts of circulating tumor DNA in the blood. Tumor informed assays have demonstrated the highest sensitivity for MRD detection. Although commercially available assays for MRD are now available, our objective is to develop a hospital based liquid biopsy platform to reduce costs. Our pipeline includes rapid clinical grade sequencing of FFPE tumor and germline samples to identify 5-6 somatic variants per patient that are used to develop digital droplet PCR assays. ctDNA tests are first validated with the patient tumor's DNA and then used for MRD detection in the patient's plasma. The analytical performance of our assays demonstrate high sensitivity (up to 97% for Variant Allele Frequency $\geq 0.1\%$), and high specificity (99%) across a concentration range (0.005% to 5%). We have analyzed over 500 plasma samples from 118 patients participating in the Q-CROC-03 and TRICIA clinical trials. These studies have confirmed the prognostic value of ctDNA detection in high risk triple negative breast cancer patients (TNBC). Patients with ctDNA negative plasma at the end of neoadjuvant chemotherapy and prior to surgery had a significantly higher relapse free survival than patients with ctDNA positive results ($p = <0.0001$). We were able to detect MRD after surgery with 92% sensitivity with a lead time of 12 months prior to clinical relapse. The median time to develop the ctDNA assays was 49 days, thus enabling the delivery of MRD results in a timely manner for clinical decisions in the adjuvant breast cancer setting.

LAY SUMMARY

Detecting small concentrations of fragments of tumor DNA in the blood require the use of liquid biopsy methods guided by the tumor. We have developed in house liquid biopsy tests guided by the patient's tumor molecular profile and tested hundred of samples from breast cancer patients. Our in house test allows the detection of very small amounts of tumor DNA in the blood with excellent reproducibility. We have shown that at a specific time point, at the end of pre-surgical treatment and before surgery, patients with no tumor DNA in the blood have a much better prognosis than patients with detected tumor DNA. We are able to develop the liquid biopsy tests in an acceptable time to inform clinicians.

Concurrent: D2-From risk assessment, screening and surveillance to survivorship

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 1/2/3

Guidelines for screening for late effects in survivors of young adult cancers: A systematic review

Presenter: Audrey Dong, The Hospital for Sick Children, University of Toronto

Other authors: Maria Locke, Alyssa Chang, Paul Nathan

ABSTRACT_ID#2729

ABSTRACT

Background Survivors of young adult (YA) cancers (ages 18-39) are at increased risk for late effects. Although multiple guidelines exist, recommendations specific to YA populations are not well defined. **Objective** To catalog existing clinical practice guidelines (CPGs) that address screening for late effects in YA survivors of cancer. **Methods** Systematic searches of PubMed, MEDLINE, Web of Science, EMBASE and 146 national/international association websites were conducted through September 2024. Three reviewers independently screened for CPGs, position statements, expert opinions, and scientific statements. The Appraisal of Guidelines for Research and Evaluation (AGREE) II instrument was used to assess the quality of recommendations. **Results** Of 4151 references screened, 61 CPGs were identified and categorized as: single late-effect guidelines (e.g., cardiotoxicity; n=37), survivorship care for specific cancer types (e.g., breast; n=16), and YA-focused guidelines with survivorship recommendations (n=8). Only 8 CPGs explicitly defined the YA population by age; 18 did not specify age criteria; 9 used developmental terms (e.g., “post-pubertal”); and 21 grouped YA with pediatric or adolescent populations. In total, 47 associations and/or consensus groups endorsed recommendations. The most commonly addressed outcomes included reproductive health (n=21) and breast cancer (n=8). **Conclusions** While some CPGs address outcomes relevant to YA survivors, few are YA-specific or comprehensive. Future work should compare guidelines to identify gaps and inform harmonized recommendations. An online repository linking existing CPGs is planned as a next step.

LAY SUMMARY

We looked at existing guidelines that help practitioners monitor and manage long-term health concerns in young adults (ages 18–39) who survived cancer. We found that few guidelines focus specifically on young adults. Often, young adults are grouped together with children or teens, or described using unclear terms, making it difficult for practitioners to provide the best specialized care for this age group. Most guidelines focus on one health problem or a cancer type rather than the overall needs of young adults. Our work shows there is a need for clear, age-specific guidelines to better support young adult survivors. We plan to create a website to help practitioners easily find these guidelines.

Concurrent: D2-From risk assessment, screening and surveillance to survivorship

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 1/2/3

Preliminary findings from the Alberta Cancer Exercise Hybrid Effectiveness-Implementation Study

Presenter: Margaret L. McNeely, Faculty of Rehabilitation Medicine, University of Alberta

Other authors: Tanya Williamson, Christopher Sellar, Shirin Shallwani, Anil Abraham Joy, Harold Lau, Jacob Easaw, Kerry Courneya, Nicole Culos-Reed

ABSTRACT_ID#3224

ABSTRACT

Background: Alberta Cancer Exercise (ACE) is a hybrid effectiveness-implementation study evaluating a cancer-specific community-based exercise program across urban sites in Alberta, Canada. Here we report the 12-week results of the program. **Methods:** Adults with any type and stage of cancer, who were undergoing cancer treatment or up to three years post-treatment completion, were eligible. ACE was delivered in-person at 18 sites across seven cities in Alberta, with video conferencing introduced during the COVID-19 pandemic. Participants took part in 60 minutes of supervised mild-to-moderate intensity exercise twice weekly for a 12-week period. **Results:** From January 2017 to February 2023, 2570 individuals enrolled. Participants were a mean age of 57.8 years, 71.3% were female, 45.4% had breast cancer, 49.4% were undergoing cancer treatment, and 31.7% had a high-risk cancer/metastatic disease. A total of 2330 (90.7%) completed the 12-week program, with a 77.1% attendance rate. Clinically significant targets were met for most objective outcomes including the 30-second sit-to-stand (+2.8 repetitions; 95% CI: 2.6, 2.9), and 6-minute walk test (+39.5 m; 95%CI: 36.3, 42.6). Clinically significant targets for fatigue and quality of life were met only in participants who were not undergoing chemotherapy treatment (n=1904). A total of 6 serious adverse events occurred requiring an ambulance call/emergency visit (4 potentially related and 2 unrelated to exercise). All cases were resolved. **Conclusion:** Results support high interest in, and benefit from ACE among Albertans with cancer. While the risk of a serious adverse event in individuals with cancer is extremely low, it's not negligible.

LAY SUMMARY

- Exercise is an effective intervention that can optimize the health and wellbeing of individuals with cancer and possibly reduce rates of cancer recurrence and secondary cancers.
- In 2017, we launched a hybrid effectiveness-implementation study to evaluate the relative benefit from an Alberta wide clinic-to-community-based cancer and exercise model of care—the Alberta Cancer Exercise (ACE) program. ACE was co-designed by exercise oncology researchers, medical and allied healthcare professionals, and individuals with lived experience of cancer. The program was offered in 7 Alberta urban locations (18 sites), and participants attended twice weekly for 60 minutes per session for a 12-week period. ACE was delivered as circuit-type classes in an in-person studio setting or virtually, or as group-based personal training conducted in the site's fitness centre.
- 2570 Albertans with cancer took part in the supervised 12-week program. We have demonstrated that ACE can: (1) be delivered successfully in-person and virtually across communities in Alberta, (2) satisfy the needs of individuals with cancer, and (3) benefit physical activity behavior, fitness, cancer-related symptoms, and quality of life. ACE is a resource that will help to inform the implementation of exercise programs in real-world settings for diverse groups of individuals with cancer.

Concurrent: D2-From risk assessment, screening and surveillance to survivorship

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 1/2/3

Risk of developing a different-site subsequent primary cancer among cancer survivors in Canada

Presenter: Dylan E. O'Sullivan, Cancer Care Alberta, Alberta Health Services

Other authors: Matthew Warkentin, Darren Brenner, Yibing Ruan

ABSTRACT_ID#2990

ABSTRACT

Background: The number of cancer survivors is rapidly increasing in Canada. The development of subsequent primary cancers (SPCs) is an important contributor to morbidity and mortality. In this study we sought to characterize the risk of SPCs among cancer survivors in Canada. **Methods:** We used data from the Canadian Cancer Registry to identify all first primary cancers (FPCs) occurring between 1992 and 2017. A SPC was considered as the next primary cancer occurring in a different site more than 6 months after initial cancer diagnosis. We estimated cumulative incidence of SPCs, as well as standardized incidence ratios (SIR) and incidence rate differences compared to the cancer-free general population. **Results:** There were 2.5 million cancer survivors in Canada in our analyses. Just over 240,000 different-site SPCs occurred with an average follow-up of 7 years. At 20 years post-diagnosis, the cumulative incidence of SPCs approached 30% for males and 20% for females. Cancer survivors were at an elevated risk of developing a different-site SPC compared to the cancer-free general population developing a first cancer (SIR = 1.25; 95% CI: 1.25-1.26) with nearly all FPC sites (37 of 41) at significantly elevated risk of developing an SPC. The SPCs with the greatest excess risks compared to the cancer-free general population included lung, colorectal, bladder, melanoma, breast, and non-Hodgkin lymphoma. **Conclusions:** Cancer survivors in Canada are at a significantly elevated risk of developing SPCs. Early detection and prevention efforts tailored to cancer survivors will be important to mitigate SPC associated morbidity and mortality.

LAY SUMMARY

There is evidence that cancer survivors are at an elevated risk of developing new cancers compared to the cancer-free general population developing a first cancer and that this risk is increasing over time. The types of subsequent cancers that cancer survivors are at the highest risk for include cancers that can be detected early through formal screening (improves survival and quality of life) and/or that are preventable through modifying lifestyle behaviours. As the number of cancer survivors in Canada continues to increase, screening and prevention efforts tailored to cancer survivors will be important to mitigate morbidity and mortality associated with new primary cancers.

Concurrent: D2-From risk assessment, screening and surveillance to survivorship

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 1/2/3

The feasibility and acceptability of endometrial cancer screening to identify a high-risk postmenopausal population

Presenter: Aline Talhouk, University of British Columbia

Other authors: Andrea Neilson, Arabella Helgason, Dollina Dodani, Rachel Woima, Nimmy Sebastian, Karman Johal, Jessica McAlpine

ABSTRACT_ID#3266

ABSTRACT

Introduction: Endometrioid endometrial cancer (EEC) prevention strategies may not be cost-effective at the population level. The RESToRE study aimed to assess the feasibility and accessibility of a two-step screening approach to identify high-risk individuals from the general population who may benefit from intervention. Methods: The first screening step used an epidemiological risk model and self-reported risk factors in a postmenopausal, asymptomatic population. Those with a 10-year absolute EEC risk $\geq 2\%$ or a body mass index ≥ 34.9 were directed to the second screening step, which involved prescribing the progesterone challenge test (PCT), with 10 mg medroxyprogesterone acetate for 10 days. Withdrawal bleeding, monitored for up to 2 weeks after the completion of the PCT, indicated a positive test and triggered a referral to a gynecologist for an endometrial biopsy. We collected participant and physician feedback to assess acceptability using the Theoretical Framework of Acceptability, and feasibility, as determined by enrollment and retention. Results: RESToRE recruited 713 participants, and 120 were high-risk and eligible (16.8%). Ninety-six of these were eligible for PCT screening (80.0%), 68/96 consented (enrollment=70.8%), and 53/68 completed the PCT (retention=77.9%). Sixteen were PCT-positive (30.2%), 14/16 were biopsied (87.5%), and 1 was diagnosed with endometrial hyperplasia (7.1%). Among those who provided feedback, the majority (76.4%) of participants considered the PCT acceptable, and most (84.2%) physicians perceived the PCT as safe. Conclusions: The feasibility and acceptability of the screening strategies proposed within the RESToRE study could offer possibilities for targeted prevention research in populations at risk of EEC.

LAY SUMMARY

Risk models could be used to find people in the population who are at high risk for endometrial cancer. The progesterone challenge test, a short course of a progestin medication, could be used to screen high-risk patients. Most physicians surveyed found this approach safe, and three-quarters of participants thought it was an acceptable method. Broad screening for endometrial cancer may not be cost-effective for everyone in the general population, but targeted screening based on risk factors may help find and support who is at highest risk, catching cancer earlier and improving health outcomes.

Concurrent: D3-Health Human Resources in Cancer Control

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 5/7/9

Rethinking future workforce planning by developing novel metrics of complexity in cancer care

Presenter: Philip Ding, University of Calgary

Other authors: Matthew Warkentin, Winson Cheung

ABSTRACT_ID#2607

ABSTRACT

The decision to recruit additional oncologists is often based on simple workload measures (e.g., new patient volumes), which may be appropriate previously, when cancer management was less complex and patient attrition was higher. We hypothesized that cancer care complexity has increased over time and that new metrics are needed to optimize workforce planning. We conducted a population-based, retrospective cohort study of adults diagnosed with common solid and blood cancers in Alberta, Canada between 2004 and 2018. We evaluated indicators of complexity including baseline characteristics, clinical course within 2 years of diagnosis, and longevity as measured by overall survival (OS). Logistic and Cox regression models estimated changes over time. A total of 141,040 patients were included (median age 66 years, 51.7% male). Breast cancer was most common (25.6%), followed by prostate, lung, colorectal, and leukemia/lymphoma. Across all sites, annual cancer incidence rate was 249.9 per 100,000 in 2004 and 284.4 per 100,000 in 2018. Specific complexity indicators increased over time, including polypharmacy at diagnosis (OR 1.30, 95%CI 1.28-1.32), multimodality treatment (OR 1.06, 95%CI 1.05-1.08), and hospital admission via the emergency department (OR 1.08, 95%CI 1.07-1.10). These metrics also demonstrated rising complexity in multivariable analyses adjusting for age, sex, and cancer site. Similarly, there was a trend towards greater longevity (HR 0.98, 95%CI 0.98-0.98). Cancer care complexity has increased over time. Workforce planning based solely on incident patient volumes may not reflect the demands of modern cancer care. Recruitment strategies should incorporate multi-faceted indicators of complexity in addition to volume-based metrics.

LAY SUMMARY

We examined the real-world data of over 140,000 cancer patients in Alberta to examine how cancer care complexity has evolved from 2004 to present. Indicators of complexity—such as taking multiple medications at baseline, needing different types of cancer treatments, and emergency hospital admissions—have increased significantly over time, alongside improvements in patient longevity. Oncology workforce planning should be optimized to consider multi-faceted indicators of complexity in addition to traditional volume-based metrics of workload.

Concurrent: D3-Health Human Resources in Cancer Control

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 5/7/9

The Human Crisis in Cancer: A Lancet Oncology Commission

Presenter: Madeline Li, Princess Margaret Cancer Centre, University Health Network

Other authors: Nirmala Bhoo-Pathy, Karla Unger, Mac Skelton, Catherine Walshe, Elizabeth Smyth, Dario Trapani, Naveen Salins, Richard Sullivan, Gary Rodin

ABSTRACT_ID#2846

ABSTRACT

Background Over 19 million cancer cases were diagnosed globally in 2020 and that is projected to increase by 75% over the next three decades. This growth will be most significant in low and middle-income countries, where there is the least health systems capacity. While the human cost is enormous, there is an imbalance in the attention and resources allocated to the physical and psychological well-being, quality of life, and social circumstances of patients and families compared to the biomedical dimensions of the disease. This has resulted in a global crisis of avoidable human suffering that is largely ignored and under-resourced. **Methodology** The Lancet Oncology invited Drs. Gary Rodin and Richard Sullivan to lead a commission on the human crisis in cancer. Evidence for the crisis, factors that are driving it, and global solutions will be elucidated through novel data, meta-analytic reviews, and case studies in the following areas: fragmented care systems, insufficient availability and access to mental health and palliative care, increased financial toxicity, limited training on the human aspects in medical education, and insufficient consideration to the sociocultural factors that influence care, research, and policy. **Impact on Practice** Launching in Fall 2025, the Commission will put a spotlight on this crisis at a critical tipping point, and aims to change the global narrative around person-centred cancer care. Solutions to be discussed include: expand humanistic competencies in oncology curricula, incentivize embedding of psychosocial and palliative care in health systems, and explore innovative health financing models to fund patient-centred interventions.

LAY SUMMARY

1. There is an imbalance of attention and resources between the biomedical and the human dimensions of cancer care.
2. The drivers of this imbalance include fragmented care systems, economic incentives, priorities in medical education and sociocultural factors.
3. Solutions to the human crisis in cancer include expanding humanistic competencies in oncology curricula, incentivizing embedding psychosocial care in health systems, and innovative health financing models.

Concurrent: D3-Health Human Resources in Cancer Control

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 5/7/9

Building a Sustainable Oncology Workforce: A Pan-Canadian Collaborative Call to Action

Presenter: Kristi MacKenzie and Sri Navaratnam, Canadian Association of Provincial Cancer Agencies

Other authors: Tara Sampalli, Stuart Peacock

ABSTRACT_ID#2548

ABSTRACT

Canada's oncology workforce is facing mounting challenges that threaten the accessibility, quality, and sustainability of cancer care. Key issues include staffing shortages, recruitment and retention difficulties, rising burnout, outdated training systems, and increasing healthcare costs. With cancer incidence and mortality projected to rise by 40% and 44% respectively by 2040, a resilient, coordinated, pan-Canadian strategy is urgently needed. In response, the Canadian Association of Provincial Cancer Agencies (CAPCA), with financial support from the Canadian Cancer Society, launched the Pan-Canadian Strategic Oncology Workforce Project. This initiative brought together over 200 system partners—including Provincial Cancer Programs, professional associations, researchers, and workforce experts—to assess the current landscape, highlight successes, and identify opportunities for national collaboration. A central challenge identified is the lack of standardized, pan-Canadian oncology workforce data, which limits pan-Canadian strategic planning and cross-jurisdictional workforce modeling. However, the initiative highlighted strong momentum, collaboration, and innovation in models of care, digital health, AI integration, and workforce wellbeing strategies. Six key opportunities emerged: advancing team-based models of care, leveraging digital innovation, supporting provider well-being, strengthening training and licensure systems, enhancing patient partnership, and improving workforce data and planning. Building on these insights, future efforts will focus on scaling high-impact strategies, strengthening knowledge sharing across jurisdictions, addressing training capacity gaps, and developing standardized, pan-Canadian oncology workforce data. Areas such as AI integration and scalable, innovative models of care are actively being explored through pan-Canadian collaboration to strengthen workforce sustainability and drive coordinated, evidence-informed solutions.

LAY SUMMARY

Canada's cancer care system is under pressure due to a shortage of trained professionals, rising burnout, and growing patient demand. By 2040, cancer cases and deaths are expected to rise significantly, making it urgent to strengthen the healthcare workforce. To address this, the Canadian Association of Provincial Cancer Agencies (CAPCA), with financial support from the Canadian Cancer Society, brought together healthcare leaders from across Canada to explore solutions. Over 200 partners contributed to this project, identifying both the challenges and opportunities in cancer workforce planning. One major gap is the lack of consistent pan-Canadian data on the cancer care workforce. Still, the initiative revealed promising efforts underway—like new care models, use of technology, and programs that support staff well-being and training.

Concurrent: D3-Health Human Resources in Cancer Control

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 5/7/9

How did COVID-19 impact practice of palliative homecare and patient outcomes?

Presenter: Peyman Pakseresht, Surveillance & Reporting, Cancer Research & Analytics, Cancer Care Alberta

Other authors: Jessica Simon, James King, Ingrid DeKock, Andrew Collins, Sukaina Kara, Sandra Peterson, Lyndsay Harrison, Zhiyin Li

ABSTRACT_ID#3241

ABSTRACT

Background: Palliative homecare services are essential for patients with advanced cancers, particularly during times of healthcare system stress. This retrospective cohort study assessed the impact of the COVID-19 pandemic on homecare delivery in Alberta, British Columbia, and Ontario. Methods: Homecare visit data were analyzed for over 100,000 adults enrolled in palliative-focused homecare who died between January 2018, and December 2021. Outcomes included use of emergency departments (ED) and hospitals in the last 90 days of life. Results: During the pandemic, the total number of nurse-provided homecare visits increased across all provinces, while the percentage of patients visiting the ED or being hospitalized decreased. Poisson regression models indicated physician home visits were associated with reduced hospitalization relative rates (RR) in both time periods (RR across provinces: 0.61-0.76 pre-pandemic, 0.51-0.72 Pandemic). Having cancer increased 10% to 38% RR of hospitalization pre-pandemic in BC and ON and 14% during pandemic in ON only. However, the overall volume of homecare activities was not associated with ED use, hospital admissions, or in-hospital death. Discussion: These findings highlight the resilience of homecare teams during COVID-19 in providing increased homecare visits. Physician visits at home play an important role perhaps through urgent symptom management, prescribing and facilitating goals-of-care discussions that impact health service use. ED use is still required in some circumstances, particularly during the pandemic, when other urgent diagnostic avenues were limited. Post-pandemic care models that integrate timely physician home visits with strong homecare support may improve patient outcomes and reduce reliance on acute care services.

LAY SUMMARY

Despite the challenges of COVID-19 pandemic homecare responded with increased home-based visits (mostly in-person) Emergency department visits, hospitalization and death in hospital decreased during pandemic Impact of cancer on health service utilization varies across provinces

Concurrent: D3-Health Human Resources in Cancer Control

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 5/7/9

A Pan-Canadian Approach to Health Human Resources in Cancer Control: Learning from Scalable Innovations and Policy Directions

Presenter: Tara Sampalli and Stuart Peacock, Canadian Association of Provincial Cancer Agencies

Other authors: Kristi MacKenzie, Sri Navaratnam

ABSTRACT_ID#3372

ABSTRACT

The global oncology workforce crisis is straining cancer care systems, with shortages across roles such as oncologists, nurses, and allied health professionals. These deficits are worsening health disparities, particularly in rural, Indigenous, and underserved communities. The COVID-19 pandemic has intensified burnout and systemic pressures, highlighting the urgent need for equitable, sustainable workforce and policy solutions. In response, the Canadian Association of Provincial Cancer Agencies (CAPCA), funded by Health Canada and the Canadian Partnership Against Cancer (CPAC), launched a pan-Canadian strategy to strengthen the oncology workforce. CAPCA's Health Services and Policy Research (HSPR) initiative applies an implementation science and phased approach to identify and support the scaling of innovations that improve access, equity, and sustainability. Phase 1 synthesized evidence from 42 systematic reviews, a national survey (n=72), and 24 key informant interviews, identifying over 30 promising models across five domains: virtual care, artificial intelligence (AI) integration, team-based care, expanded scopes of practice, and home/community-based care. Policy insights emphasize the need for pan-Canadian standards in licensure frameworks, expanded career pathways, digital and AI governance, and infrastructure. Strengthening community-facing roles and care models, alongside sustained funding for implementation and evaluation—with a rural and equity lens—is also critical. The findings highlight the systemic and economic benefits of integrated, technology-enabled, community-embedded care. HSPR findings will be presented from phase 1 and progress on subsequent phases. CAPCA and partners are shaping actionable policy directions to support scalable, equitable innovations in cancer care, highlighting promising models, adoption enablers, and opportunities to strengthen national workforce planning.

LAY SUMMARY

Canada's cancer care system is under pressure due to a shortage of healthcare workers, including doctors, nurses, and support staff. These shortages are causing treatment delays and affecting vulnerable communities the most. The COVID-19 pandemic has made the situation worse, increasing stress and burnout among providers. To tackle this issue, the Canadian Association of Provincial Cancer Agencies (CAPCA), with support from Health Canada and the Canadian Partnership Against Cancer, launched a national research initiative. Working with universities and cancer centres, this project identified over 30 promising ways to improve care such as using virtual care, AI, team-based care, and bringing services into people's homes and communities. These solutions help patients get timely, coordinated care, especially in underserved areas. The research also highlights the need for supportive policies, digital tools, and stable funding. This work will guide future efforts to build a stronger, fairer cancer care system for all Canadians.

Concurrent: D4-Lung Cancer in Canada: Plans and Trends

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 8

Transforming the Future of Lung Cancer in Canada: Co-Creating a Pan-Canadian Action Plan to Reduce Lung Cancer Mortality by 30%

Presenter: Annemarie Edwards, Canadian Cancer Society

Other authors: Christian Finley, Angus Pratt, Julia Nordlund, Anne Mullin

ABSTRACT_ID#2584

ABSTRACT

Lung cancer is the leading cause of cancer death in Canada, claiming more lives annually than breast, colorectal, and prostate cancers combined. Despite advancements in care, inequities in access, diagnosis, and outcomes persist—especially among underserved populations. To address this, the Canadian Cancer Society (CCS), with guidance from a national Steering Committee of diverse stakeholders, developed the 2025-2035 Pan-Canadian Lung Cancer Action Plan. The plan's overarching goal is to reduce lung cancer deaths by 30% over the next decade through coordinated, equity-focused action. The Action Plan was developed through a multi-phase, person-centred and community driven process. Inputs were gathered from environmental scans, two outreach surveys, and a national Lung Cancer Summit held in late 2024. The Summit convened over 120 interest holders representing 9 jurisdictions across the lung cancer ecosystem, including people with lived experience, cancer charities, clinicians, researchers, advocates, policymakers, and representatives from equity-deserving communities. A modified Delphi approach that integrated an adapted form of the James Lind Alliance priority setting partnership was used. This enabled a structured dialogue in plain language and consensus-building to identify and validate key priorities across the continuum of lung cancer care, along with actions and progress indicators. The Action Plan is built around four strategic priorities: 1) Reduce exposure to carcinogenic inhalants; 2) Detect lung cancer earlier; 3) Improve access to timely, best-in-class care; and 4) Accelerate lung cancer research. The 2025-2035 Pan-Canadian Lung Cancer Action Plan outlines a bold, coordinated path forward to reduce lung cancer mortality and address longstanding inequities in care.

LAY SUMMARY

The burden of lung cancer is staggering. In 2024, it's estimated that nearly 21,000 people died from this disease. Too many lives are unnecessarily cut short. Through this plan, we aim to save thousands of lives by reducing lung cancer mortality in Canada by 30% by 2035, an achievement that will reinforce Canada's commitment to the World Health Organization Sustainable Development Goals. The Canadian Cancer Society partnered with a broad range of organizations, patient-based groups, and individuals with living experience to create the Pan-Canadian Lung Cancer Action Plan. This plan reflects the voices of individuals at the frontline of care, including patients, caregivers, clinicians, researchers, health charities, and government representatives. It's built on input from people with expertise in the barriers and the opportunities to improve lung cancer care in Canada. Central to this effort is the recognition of the gross inequities that exist in access to life-saving lung cancer prevention, treatment and care across the country, contributing to disparities in health outcomes for many underserved communities. The stigma surrounding lung cancer further exacerbates these inequities. It's time to break down these barriers to ensure access for all to expanded screening programs, innovative treatments and the highest standard of care.

Concurrent: D4-Lung Cancer in Canada: Plans and Trends

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 8

Impact of PLCOm2012 Ethnicity Model Variations on Eligibility for Lung Cancer Screening

Presenter: Kirk Graff, University of Calgary

Other authors: Matthew Warkentin, Laura Hampton, Huiming Yang, Alain Tremblay

ABSTRACT_ID#2754

ABSTRACT

Background: Screening with low-dose computed tomography reduces lung cancer mortality among individuals at high risk of lung cancer. Three PLCOm2012 model variations predicting lung cancer risk have been developed: the original model with 6 ethnicity/race categories (6R), a model with 3 ethnicity/race categories (3R), and a model with no ethnicity/race parameters (NR). We aimed to determine the impact of model selection on screening eligibility for the Alberta Lung Cancer Screening Program (ALCSP). Methods: Retrospective analysis of 5,961 individuals contacting the ALCSP. For each, we calculated their 6-year risk of lung cancer using the three models and compared their eligibility using a 1.5% 6-year lung cancer risk threshold. Results: Seventy percent of individuals identified as White, 9% Asian, 4% Indigenous, 0.5% Black, and 0.4% Hispanic with 17% not provided. Sixty-seven percent had a risk $\geq 1.5\%$ risk across all 3 models while 30% were $< 1.5\%$ risk across all models. Using the NR model, 31% of Indigenous and 25% of Black individuals were classified below the 1.5% threshold but would have been eligible using 3R or 6R. Using the 6R model, 18% of Asian and 22% of Hispanic individuals were below 1.5%, but were above 1.5% in either the NR or 3R model. Conclusions: Choice of PLCOm2012 model can have a significant impact on screening eligibility, particularly for ethnic minorities. Using the 3R model increases screening eligibility for Indigenous, Black, Hispanic, and Asian individuals. Canadian based data validating and quantifying the impact of race and ethnicity on lung cancer risk are urgently needed.

LAY SUMMARY

The PLCOm2012 models estimate lung cancer risk using smoking history and other important factors, differing in how they account for race or ethnicity. Model choice can affect screening eligibility: Indigenous, Black, Asian, and Hispanic individuals may be excluded by one model but included by another. The three-race model, which classifies participants as Black, Indigenous, or Other, may improve access to life-saving screening for underrepresented groups.

Concurrent: D4-Lung Cancer in Canada: Plans and Trends

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 8

Youth and Nicotine pouches: The concerning new trend examined through TikTok and its implications for cancer prevention

Presenter: Ashmeet Mand, University of British Columbia - Okanagan

Other authors: Laura Struik

ABSTRACT_ID#3357

ABSTRACT

Nicotine pouches have been marketed as a less harmful alternative to smoking. They began to gain popularity, particularly among youth, due to their discrete nature and convenience. However, despite claiming reduced harm, these pouches deliver high nicotine concentrations, posing a risk to oral health and cancer development. Social media platforms, like TikTok, play a major role in promoting and normalizing their use, particularly among youth. Little is known about how nicotine pouch content is presented on platforms like TikTok. This study explores how nicotine pouches (specifically Zyns) are represented on TikTok using a qualitative descriptive design. A total of 200 TikTok posts were screened under the #zyn, #zyns, and #nicotinepouch hashtags, and 132 were analyzed. Posts were analyzed using Braun and Clarke's thematic analysis approach. Collaborative coding ensured reliability and uncovered key themes in the content. Five themes emerged: (1) The Zyn Movement; (2) "Boy Heaven"; (3) "Unintended Negative Consequences"; (4) Product design: "Life doesn't need to stop"; (5) Physical Benefits: "Its like IcyHot for your Mouth." Overall, the content heavily condoned nicotine pouch use and normalized it with males disproportionately represented as the primary users of nicotine pouches. This study highlights TikTok's influence in shaping youth perceptions of nicotine pouches as something trendy and relatively harmless. This underlines the need for targeted interventions and policies to protect youth from the risks of nicotine pouch use, including cancer.

LAY SUMMARY

- Nicotine pouches are shown on TikTok as trendy and harmless. This is concerning because many TikTok users are youth. Videos often describe nicotine pouches as fun, cool, and socially acceptable, with little mention of the health risks.
- Youth are being targeted through product design and messaging. Phrases like "Life doesn't need to stop" and "ZynLifestyle" appear in content from influencers, athletes, celebrities, and everyday TikTok users. This messaging promotes the idea that using nicotine pouches is part of a fun active and carefree lifestyle and that there is a "cool" community to belong to.
- This kind of content can shape youth beliefs and behaviours. When harmful products like nicotine pouches are normalized on social media, young people may underestimate the risk and be more likely to try them.

Concurrent: D4-Lung Cancer in Canada: Plans and Trends

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 8

Trends in adult combustible and non-combustible tobacco product use prevalence in the United States, 2014 to 2023

Presenter: Rafael Meza, BC Cancer Research Institute

Other authors: K. Michael Cummings, Yoonseo Mok

ABSTRACT_ID#3315

ABSTRACT

Background Tobacco use patterns are changing in the US, with some predictions that electronic cigarettes will soon surpass cigarettes as the most prevalent type of tobacco product consumed. Methods We estimated current use prevalence of combustible and non-combustible tobacco products from the National Health and Interview Survey (NHIS) from 2014 to 2023. Combustible products included cigarettes, cigars, pipe and hookah. Non-combustible products included e-cigarettes and oral tobacco. Joinpoint regression was used to characterize trends by age (18+, 18-24, 25-34, 35-54 & 55+) and sex (overall, females, males). Separate analyses were conducted for overall, exclusive and dual use of the two product categories. Results Any tobacco use decreased from 2014 to 2017 at an annual percentage change (APC) rate of -4.83% but has since remain roughly constant. Combustible tobacco decreased at an APC rate of -3.9%, with faster decreases among younger adults (18-24: -11.9%; 25-34: -5.4%; 35-54: -2.9%; 55+: -1.5%). Non-combustible tobacco has been increasing since 2017 at an APC of 8.8%, with faster increases among younger adults (18-24: 10.93%; 25-34: 11.7% since 2016; 35-54: 7.6%; 55+: 3.7% since 2017). Similar trends were observed by sex. Conclusions Overall tobacco use is declining slowly. However, the mix of tobacco products consumed by US adults has shifted. Combustible tobacco use is decreasing, particularly among younger adults. Conversely, non-combustible tobacco use is increasing, at faster rates among younger adults. Continuing monitoring of tobacco use trends is critical to understanding market dynamics and identifying priorities for tobacco control and predicting future tobacco related mortality.

LAY SUMMARY

Tobacco use remains the leading preventable cause of cancer mortality, but use patterns are changing. Combustible tobacco use in the US is decreasing, particularly among younger individuals. However, non-combustible tobacco is increasing, also with faster increases in younger individuals. Fewer combustible tobacco use should reduce cancer risk in the long run. But the implications of the increase in non-combustible product use, particularly of e-cigarettes, on cancer morbidity and mortality is still uncertain as its effects on cancer risk are yet to be determined. Similar studies are needed to characterize trends of combustible and non-combustible tobacco use in Canada.

Concurrent: D4-Lung Cancer in Canada: Plans and Trends

Monday, November 3, 2025, 2:45:00 PM - Hyatt Imperial 8

Increasing Equity in Lung Cancer Prevention and Early Detection: Co-Evaluation of a Patient-Partnered Educational Intervention

Presenter: Ambreen Sayani, Women's College Hospital

Other authors: Zeenat Ladak, salva Niwe, Tatiana Demarco, Aisha Lofters, Angus Pratt, Annemarie Edwards, Bikila Amenu, Christian Finley, Gary Bloch, Howard Freedman, Jackie Manthorne, Janet Parsons, Jean-Claude Camus, Joyce Nyhof-Young, Shari Dworkin, Tara Jeji, Tara Jeji, Vanessa Redditt, Vanessa Wright, Vinesha Ramasamy

ABSTRACT_ID#2738

ABSTRACT

Background. Inequities in smoking rates contribute to a greater risk of lung cancer and mortality. Systemic biases, stigmatizing clinical encounters and a lack of responsive primary-care create further barriers to smoking cessation and lung cancer screening. To fill this gap, we co-created an educational intervention to build Trauma- and Violence-Informed Care (TVIC) skills and competencies in primary care providers (PCPs). We are now co-evaluating the impact of the intervention. **Methods.** This is an equity-oriented, patient-partnered, interdisciplinary, implementation science study. The intervention is an e-learning module for PCPs called “Creating Safe Connections”. Our co-evaluation over a one-year period follows explanatory mixed-methods approaches (surveys and interviews) to assess PCPs participation, satisfaction, and knowledge, guided by the RE-AIM framework (Reach, Effectiveness, Adoption, Implementation, Maintenance). **Results.** Participation & Reach: Preliminary evaluation findings show that enrolled learners (N=23) span across care settings with majority represented in primary care (n=9). Effectiveness & Knowledge: Over half of learners (n=12) have completed the module and agree/strongly agree that they will use learnings in their practice including developing “scripts for staff” about TVIC and using “person first language”. Comparison of pre- and post-evaluation surveys also identify an increase in knowledge, understanding, and confidence of TVIC, by approximately 2-fold. Implementation & Satisfaction: Learners also showed appreciation for the module such as the integrated “first-person experiences”. **Conclusions.** The “Creating Safe Connections” module will equip PCPs to engage in safer, more effective conversations about smoking cessation and lung cancer screening and enable patients across Canada’s healthcare system to achieve better health outcomes.

LAY SUMMARY

“Creating Safe Connections” is a CME-accredited e-learning module that can strengthen provider-patient relationships and enhance preventative care outcomes across Canada. The module has been co-designed with people with lived and living experience of lung cancer risk or smoking cessation. This educational intervention equips primary care providers with Trauma- and Violence-Informed Care approaches to engage in safer, more equitable lung cancer screening and smoking cessation conversations, ultimately promoting better patient health outcomes for at-risk communities.

Concurrent: D5-Cervical Cancer and HPV-related interventions

Monday, November 3, 2025, 2:45:00 PM - Hyatt Doll/Herald

Predictors of participation in cervical cancer screening in Canada: Evidence from the Canadian Partnership for Tomorrow's Health

Presenter: Maryam Darvishian, University of British Columbia

Other authors: Amina Moustaqim-Barrette, Philip Awadalla, Parveen Bhatti, Philippe Broet, Rachel Murphy, Kimberly Skead, Robin Urquhart, Jennifer Vena, Trevor Dummer

ABSTRACT_ID#2817

ABSTRACT

Background Cervical screening is proven to reduce cancer mortality. By identifying disparities in participation, we can design targeted strategies to enhance engagement and ultimately reduce the burden of the disease. Methods Self-reported screening status and demographic, clinical, and lifestyle factors for female participants, were obtained from five regions of the Canadian Partnership for Tomorrow's Health —BC Generations Project, Alberta's Tomorrow Project, Ontario Health Study, Quebec's CARTaGENE, and the Atlantic Partnership for Tomorrow's Health Study (Atlantic PATH). Multivariable logistic regression was used to assess associations with screening status among women with history of screening, defined as having a Pap or smear test <6 months to ≤3 years ago, or >3 years ago. Results In total, 162,592 individuals met the inclusion criteria. Overall, 98% of people had a history of being screened for cervical cancer. The prevalence of screening <6 months to ≤3 years ago ranged from 78% in CARTaGENE to 85% in Atlantic PATH. Having a lower household income, being older (OR 1.35; 95% CI 1.30-1.40), being single, being a current smoker, having poor self-perceived health, having a comorbidity, and it being ≥24 months since a routine medical check-up (OR: 7.79, 95% CI 7.49-8.09). Conclusion Overall uptake of cervical cancer screening was high and exceeded the national target of 80% of eligible people being up to date with screening in this cross-sectional analysis. The observed associations between social, personal and clinical factors with screening status provide insight toward implementing targeted interventions, such as enhanced access to primary care provider services.

LAY SUMMARY

1. By identifying disparities in participation, we can design targeted strategies to enhance engagement and ultimately reduce the burden of the disease.
2. We observed associations between social, personal and clinical factors with screening status. These findings will provide insight toward implementing targeted interventions, such as enhanced access to primary care provider services.

Concurrent: D5-Cervical Cancer and HPV-related interventions

Monday, November 3, 2025, 2:45:00 PM - Hyatt Doll/Herald

Incidence of oncogenic HPV genotypes 16 and 18 by HPV vaccination status, HIV status, and age group among urban gay, bisexual, and other men who have sex with men in Canada, 2017-2024

Presenter: Pranamika Khayargoli, University of Toronto

Other authors: Catharine Chambers, Joseph Cox, Alexandra de Pokomandy, Shelley Deeks, Daniel Grace, Troy Grennan, Trevor Hart, Nathan Lachowsky, David Moore, Rosane Nisenbaum, Gina Ogilvie, Chantal Sauvageau, Darell Tan, Anna Yeung, François Coutlée, Ann Burchell

ABSTRACT_ID#2478

ABSTRACT

Background: Human papillomavirus (HPV) causes over 90% of anal and 73% of oropharyngeal cancers, which disproportionately affect gay, bisexual, and other men who have sex with men (GBM), especially those living with HIV. Prophylactic HPV vaccines have been publicly-funded for Canadian GBM since 2015/16; we evaluated HPV-16/18 incidence following their implementation. Methods: Sexually-active GBM aged ≥ 16 years were recruited from Montreal, Toronto, and Vancouver (2017-2019) via respondent-driven sampling. Participants self-reported HPV vaccination and self-collected anal and oral HPV specimens annually for four years. Incidence rates (IR), defined as new HPV16/18 detections among those negative at baseline, were calculated per 100 person-years using Poisson regression. IRs were stratified by HPV vaccination, HIV status, and age group (16-30; 31-40; 41-50; 51+). Results: 1,836 and 1,926 participants provided ≥ 2 anal or oral specimens, respectively (mean age 39.9 ± 12.8 years, 17% HIV-positive, 42% vaccinated ≥ 1 dose). Anal HPV16/18 IRs were 4.4 [95%CI: 3.6, 5.2] and 1.5 [1.2, 2.1]; oral IRs were 0.5 [0.3, 0.8] and 0.1 [0.1, 0.3]. Anal HPV16 acquisition was higher among unvaccinated (6.3 [5.2, 7.8]) than vaccinated (2.1 [1.4, 3.1]), and among HIV-positive (6.2 [4.0, 9.5]) than HIV-negative (4.1 [3.4, 5.0]) participants; results were similar for HPV18 and oral. Anal HPV16 IRs were lowest in ages 31-40 (3.7 [2.7, 5.0]), and highest in ages 51+ (5.7 [3.7, 8.8]); oral HPV-16 peaked in ages 41-50 (1.6 [0.7-3.8]), similar for HPV18. Conclusions: Following publicly-funded HPV vaccination for GBM in Canada, HPV16/18 acquisition was higher among older, unvaccinated, or those living with HIV.

LAY SUMMARY

Human papillomavirus (HPV) infections that can cause cancers in the anal and oral sites are common among gay, bisexual, and other men who have sex with men (GBM). We found that many Canadian GBM in our study became infected with oncogenic HPV types 16 and 18 in anal and oral regions from 2017-2024, despite publicly-funded HPV vaccination for younger GBM since 2015/16. These infections were more common among men who were older, unvaccinated, or living with HIV. HPV vaccine shows some protection, but more research is needed. We found that among vaccinated participants, there were lower incidence rates of HPV16/18 infections, most likely to cause cancer. However, we still need more research to understand how well the vaccine protects against infections that persist over many years in different risk groups. HPV infections in the oral sites are less common than anal but remain a concern. We found that fewer participants got infected with oral HPV, but those who did were often older, unvaccinated, or living with HIV. Since HPV-related oral cancer is now the most common HPV-associated cancer in Canada, more research is needed to better understand if these infections also last longer (and lead to cancer) in those groups.

Concurrent: D5-Cervical Cancer and HPV-related interventions

Monday, November 3, 2025, 2:45:00 PM - Hyatt Doll/Herald

Vaccination and screening strategies to accelerate cervical cancer elimination in Canada: An OncoSim modeling study

Presenter: Ioana Nicolau, University of Calgary

Other authors: Kirk Graff, Yibing Ruan, Rochelle Garner, Claude Nadeau, Mary Upshall, Jan Hontelez, Inge MCM de Kok, Darren Brenner

ABSTRACT_ID#2934

ABSTRACT

Background and Objective: The World Health Organization launched a global strategy to eliminate cervical cancer as a public health concern. Canada has aligned with this goal, setting a national target of fewer than 4 cases per 100,000 by 2040. Understanding the impacts of various HPV vaccination and cervical screening strategies is critical to achieving this goal.

Methods: We used OncoSim-Cervical (v 3.6.5.7), a microsimulation model, supported by the Canadian Partnership Against Cancer with model development by Statistics Canada, to project cervical cancer outcomes under varying scenarios over time (2025-2050). We varied scenarios by HPV vaccination coverage (70% or 90%), and screening modality (Pap or HPV DNA testing at 70% or 90% participation, 3-year screening intervals for all modalities). Outcomes included age-standardized cervical cancer incidence rates, cumulative cases and deaths prevented, and health-adjusted life years (HALYs) gained out to 2040.

Results: The strategy with 70% vaccination coverage and 90% participation in HPV DNA testing was projected to reduce incidence below the elimination threshold before 2040, preventing approximately 10,000 cases and over 1,000 deaths from 2025-2040 and yielding an additional 22,000 HALY gained. Increasing vaccination uptake to 90% did not change the results until 2040. Scenarios with HPV testing outperformed Pap-based screening alone, even with similar participation rates.

Conclusions: Transitioning to HPV DNA testing with strong participation are important considerations eliminating cervical cancer in Canada. Vaccination impacts are long term and essential to reduce infection prevalence and screening burden.

LAY SUMMARY

The World Health Organization launched a global strategy to eliminate cervical cancer as a public health concern. Transition to HPV DNA testing with high participation is an important strategy to reach Canada's cervical cancer elimination target by 2040. Improving screening participation has a greater short-term impact than increasing vaccination coverage, though both are important.

Concurrent: D5-Cervical Cancer and HPV-related interventions

Monday, November 3, 2025, 2:45:00 PM - Hyatt Doll/Herald

Vaginally delivered therapeutic human papillomavirus (HPV) vaccines in women in rural LMICs

Presenter: Gina Ogilvie, BC Women's Health Research Institute

Other authors: Smritee Dabee, Candice Ruck, Hallie Dau, Amy Booth, Priscilla Naguti, Laurie Smith, Jackson Orem, Carolyn Nakisige

ABSTRACT_ID#2762

ABSTRACT

Background: Therapeutic HPV(T-HPV) vaginal vaccines to treat existing HPV infections are under development. Given their potential to improve treatment coverage, particularly in low-resource settings with high cervical cancer burden, it is critical to better understand T-HPV acceptability among women. **Methods:** Women participating in a cervical cancer screening program from 14 villages in rural Malongo subcounty, Uganda, completed a survey that included questions on vaginal vaccine acceptability and confidence. Village health teams recruited women aged 30-49 years with no cervical cancer or screening history. They used visual info-sheets to demonstrate how vaginal tablet and ring vaccines work. Descriptive statistics and odds ratios were generated. **Results:** Of 2276 recruited women, most were married (78.3%; n=1782) and had ≤ primary education (89%; n = 2026). Self-reported HIV prevalence was 6.7% (n=152). While 99.6% (n=2267) indicated that they would be willing to use a vaginal HPV vaccine, 72.7% (n=1655) were concerned about injuring themselves. Women expressed more confidence in their ability to insert a tablet than ring (96% v 66%; p<0.01), and 61.6% preferred self-inserting than having a health care provider insert the vaccine (n=1403; p<0.0001). Confidence in ability to insert a vaginal ring was associated with cervical cancer awareness (adjusted odds ratio: 1.66, 95% CI 1.34-2.04), previous outreach visit attendance (aOR: 1.43[1.17-1.75]), and not having an STI (aOR: 0.54[0.45-0.64]). **Discussion:** Both vaginal rings and tablets were considered acceptable by the women surveyed, with expressed preference for tablets and self-insertion. These findings offer support for potential T-HPV vaginal vaccine use in low-resource settings.

LAY SUMMARY

Vaccines delivered vaginally to treat existing HPV infections may help reduce gaps in treatment coverage and reach more women who require treatment; thereby preventing progression to cervical cancer. This can be particularly useful in low resource settings like Uganda, where the burden of cervical cancer is high but there is a lack of infrastructure to treat cervical cancer. Our research provides insight into whether therapeutic vaginal vaccines would be acceptable to women in low-resource settings. Overall, women find the use of vaginal vaccines acceptable, indicating the potential for introduction in these settings and the potential to improve access and delivery of cervical pre-cancer treatment. Self-insertion is the preferred option among women. This could help increase the reach of the therapeutic vaccine, particularly in rural areas and for those unable to access a healthcare provider due to barriers like distance and time. Our research also shows that both vaginal rings and tablets are acceptable to women but more feel comfortable using a tablet. This indicates that both options have the potential to be introduced. In a setting where both options are available, careful education and instruction will be necessary.

Concurrent: D6-First Nations, Inuit and Métis Stories

Monday, November 3, 2025, 2:45:00 PM - Hyatt Stephen AB

Telling the stories of Indigenous Cancer Patient Multidisciplinary Rounds - an innovative model of equitable cancer care

Presenter: Veronica Big Plume, University of Calgary

Other authors: Jessica Simon, Patricia Biondo, Arrow Big Smoke, Danielle Petricone-Westwood, Paige Campbell, Sharon Watanabe, Angeline Letendre, Pamela Roach

ABSTRACT_ID#2391

ABSTRACT

Background: "Indigenous Cancer Patient Multidisciplinary Rounds" are novel in bringing together multiple disciplines of healthcare providers and across sectors, including Indigenous Elders, cancer centre and community care providers. Meeting monthly by videoconferencing, they discuss whole person care of Indigenous cancer clients facing complex care barriers. As Indigenous and non-Indigenous researchers and clinicians, we sought to explore the impacts of Rounds from care providers' perspectives. Method: Privileging Indigenous ways of knowing in the design and conduct of this qualitative study, we invited Rounds attendees and others impacted by Rounds to a 1:1 semi-structured interview (based on concepts in the Medicine Wheel) with an Indigenous researcher, to gather their perspectives on Rounds. Transcripts were coded line-by-line by two of our team and emerging themes were discussed with the whole collaborative. Results: To date, emerging themes from 12 interviews include: Gathering and sharing understanding between attendees; Whole person care; Supporting and empowering Indigenous patients; Appreciating Indigenous Ways of Being; Difference in World views, Impact on self; Racism and systemic barriers; and Allyship with Rounds sustaining providers in advocating for clients and system changes. We will share final themes, elements needed to sustain or spread Rounds in other contexts and challenges to anticipate in Rounds function. Conclusion: Rounds bring together and sustain providers from different disciplines, backgrounds and locations, to collectively improve support of individual Indigenous cancer patients and address systemic barriers that can overwhelm individual patients and providers. This model may be useful to other patient groups and health settings facing systemic inequities.

LAY SUMMARY

"Indigenous Cancer Patient Multidisciplinary Rounds" are a novel model of care that improve support for Indigenous cancer clients by bringing together healthcare providers, Indigenous Elders, and community care providers. Learnings from our interviews with Rounds attendees may be useful to others interested in adapting this model of care (e.g., for other illnesses or settings of care) to support people facing complex barriers to care.

Concurrent: D6-First Nations, Inuit and Métis Stories

Monday, November 3, 2025, 2:45:00 PM - Hyatt Stephen AB

Lived experiences of Métis cancer patients, survivors, and caregivers in Saskatchewan: Informing a Métis Cancer Control Strategy

Presenter: Angi Patel, University of Saskatchewan

Other authors: Terri Hansen-Gardiner, Andre Letendre, Rachel Hewitt, Tegan Brock, Marg Friesen, Tracey Carr

ABSTRACT_ID#2672

ABSTRACT

This study explored the lived experiences of Métis cancer patients, survivors, and caregivers in Saskatchewan through a culturally grounded secondary analysis of qualitative interview data using the Collective Consensual Data Analytic Procedure (CCDAP) which was originally collected by the Métis Nation–Saskatchewan. Using an adapted version of the CCDAP, the analysis was conducted collaboratively with Métis Knowledge Keepers, Métis Nation–Saskatchewan research staff, an honours student researcher, and an academic researcher supervising the honours student. The methodological foundation was grounded in Indigenous research paradigms that emphasize relational accountability, respect, reciprocity, and storytelling. Analysis revealed deeply interconnected roles among participants where patients often became caregivers, and survivors shared in caregiving responsibilities which highlighted the importance of kinship, relational care, and community responsibility in Métis cultural contexts. During this presentation, the student researcher, one of the Knowledge Keepers, and academic researcher will describe the challenges Métis face while navigating cancer care systems, including systemic barriers, emotional and financial burdens, and gaps in culturally safe care. The study reflects the importance of community-led analysis and governance in research processes. The findings aim to inform the development of a Métis Cancer Control Strategy and contribute to improved policies, programs, and support tailored to the needs of Métis people in Saskatchewan.

LAY SUMMARY

1. In Métis culture, roles often overlap where cancer patients may also be caregivers, and survivors may help care for others. This highlights the strong emphasis on family, relational care, and shared community responsibility.
2. Métis individuals often have unique needs and face systemic barriers in cancer care. Participants described challenges such as emotional and financial strain, difficulty navigating complex health systems, and a lack of culturally safe care that respects Métis values and traditions.
3. Importance of community-led research matters which is why this study was guided by Métis Knowledge Keepers and community members, showing the importance of Indigenous leadership in research. These insights will help shape a Métis cancer care to improve care and support for Métis people in Saskatchewan.

Concurrent: D6-First Nations, Inuit and Métis Stories

Monday, November 3, 2025, 2:45:00 PM - Hyatt Stephen AB

Engaging with Indigenous Patients with Multiple Myeloma: Lessons Learned and Future Research

Presenter: Julie Stakiw and Terri Hansen-Gardiner, University of Saskatchewan

Other authors: Maryam Jafari, Tracey Carr

ABSTRACT_ID#2953

ABSTRACT

This project explores the development of culturally responsive and community-informed care for Indigenous patients and families with Multiple Myeloma (MM) in Saskatchewan. Initiated in May 2023, the first engagement session was led by First Nations and Métis knowledge keepers and included representation from patients, caregivers, the Métis Nation–Saskatchewan (MN–S), and the Saskatchewan Cancer Agency. It was a moment of recognition for many healthcare professionals, especially clinicians, who realized that Indigenous patients with MM have unique and often unmet needs. Through community consultations and working group meetings, various barriers to care were highlighted such as geographical isolation, fragmented care coordination, limited access to screening and early diagnosis, cultural and language disconnects, historical trauma, and systemic racism. These challenges are made even harder by a deep mistrust of the healthcare system and limited access to understandable health information. Individuals spoke about the difficulties in navigating care that create feelings of disconnectedness such as long travel times, unreliable transportation, nowhere to stay, and a system that often does not reflect or respect their cultural values. These were not just isolated issues, but common struggles shared across communities. The meetings and the working groups, rooted in community engagement and development, aim to improve timely diagnosis, symptom management, and healthcare experiences for Indigenous patients for addressing broader cancer care inequities in Indigenous populations across Canada. In this session, alongside knowledge keepers, we will share our lessons learned and future research directions.

LAY SUMMARY

1. Indigenous patients with Multiple Myeloma face unique barriers to care and through community engagement sessions, we learned that many First Nations and Métis patients experience delayed diagnoses and fragmented care. Challenges like long travel distances, limited access to specialists, and lack of culturally safe support often leads to poorer outcomes.
2. Trust and communication matters deeply in healthcare because historical trauma and cultural disconnection make it hard for some patients to feel safe or understood in the healthcare system. Many shared stories of not being listened to, struggling with paperwork, or not understanding medical language.
3. Community-led solutions are key which is why we are working with Indigenous leaders, patients, and healthcare providers to create a new care model that's respectful, supportive, and rooted in community priorities. This includes better care coordination, early diagnosis, access to palliative care, and culturally appropriate navigation, all delivered as close to home as possible.

Concurrent: D6-First Nations, Inuit and Métis Stories

Monday, November 3, 2025, 2:45:00 PM - Hyatt Stephen AB

Mawlugutinej: Towards self-determined cancer care in Mi'gmaq territory - When ancestral knowledge and cultural safety save lives

Presenter: Tanya Webster, CISSS de la Gaspésie - Centre de santé Listuguj

Other authors: Catherine Leblanc, Donna Metallic

ABSTRACT_ID#2410

ABSTRACT

Funding from the Canadian Partnership Against Cancer (CPAC) has made possible a collaborative project between Listuguj Mi'gmaq Health Services and Santé Québec - CISSS de la Gaspésie. This partnership aims to encourage citizen participation in cancer screening and investigation in a community where these services were virtually absent, despite high cancer rates. In six months, more than 150 people have benefited from screening for cervical, breast and lung cancer, thanks to the establishment of a dedicated local counter. This initiative stands out for the transformation it has initiated: a local team has been formed to offer services in a culturally secure environment, while accessing CISSS clinical and IT infrastructures, in a spirit of collaboration. The clinical partnership between professionals from Listuguj and CISSS de la Gaspésie fosters mutual sharing of knowledge while respecting cultural realities. Based on Mi'gmaq oral tradition, screening algorithms have been adapted, and awareness-raising activities (testimonials, art, video, radio) have been implemented. The project has fostered the emergence of a screening culture in Listuguj, strengthened community autonomy and paved the way for respectful and equitable collaboration between the Mi'gmaq and CISSS- Santé Québec. Through this project, the CISSS de la Gaspésie has moved from a logic of providing services to a logic of promoting co-development, the transfer of knowledge and self determination for First Persons Communities.

LAY SUMMARY

First structured access to cancer screening in Listuguj: Thanks to the partnership between Listuguj Mi'gmaq Health Services and CISSS de la Gaspésie, over 150 people have been able to access cervical, breast and lung cancer screening, which was previously almost non-existent in the community.

A culturally adapted and local approach: A local team has been trained to offer services in a safe environment that respects Mi'gmaq traditions. The project integrates oral tradition, art, radio and testimonials to raise awareness of the importance of screening, adapting the tools of the Quebec health network.

A partnership model aimed at health equity and self-determination: The CISSS, in partnership with the Listuguj team, has embarked on a transformation of its role, moving from a model centered on the provision of care to a partnership approach based on self-determination. Professionals share their clinical expertise and tools with local teams, who adapt them to their own context. This model has fostered the emergence of a genuine screening culture in Listuguj, while strengthening the community's ability to take charge of its own health in a sustainable and autonomous way.

Concurrent: D6-First Nations, Inuit and Métis Stories

Monday, November 3, 2025, 2:45:00 PM - Hyatt Stephen AB

Li Kawnsayr: Stories of Métis Cancer Journeys

Presenter: Celine Wick, Métis National Council

Other authors: Breane Mahlitz, Carolyn Lacka, Stephanie Thevarajah

ABSTRACT_ID#2384

ABSTRACT

The Métis National Council Health Team worked to produce a compelling and strengths-based documentary series shedding light on the health inequities faced by Métis Nation citizens. The pilot episode, Li Kawnsayr, aims to address the current data gap while privileging Métis ways of knowing, being and doing. While Indigenous health remains a rapidly growing body of research, significant gaps remain in relation to Métis health research. Métis have been historically underserved, misrepresented & underrepresented within health research and data collection. The docuseries will focus citizens' stories of their experience and challenges with their health. Ultimately, this series aims to amplify the diverse voices and scope of knowledge within the Métis community around experiences and perspectives on addressing Métis-specific needs. Métis health research and data collection must be done by Métis communities in ways that honour & empower Métis worldviews and value systems. Through storytelling, the docuseries episode is helping to raise awareness, foster understanding, provide needed data, and advocate for addressing the health gaps faced by the Métis Nation. Our team, in partnership with an Indigenous film crew, travelled across the homelands, visiting with Métis citizens and families to amplify their experiences of cancer. On filming days, our participants welcomed us into their home where we shared a meal and visited. Visiting (keeoukaywin) is "vital, as it richly textures everyday life [and] ... is how we come to know, and to re-know" (Gaudet, 2019); "visiting is memory work" (Tuck et al, 2022). Centering visiting (keeoukaywin) helps us to honour reciprocity & relationality. Maarsii

LAY SUMMARY

The docuseries is an opportunity to collect much needed Métis-specific qualitative data in ways that align with Métis values, uplift our Nation and demonstrate the strength of our people. This docuseries pilot episode has been an opportunity to showcase stories of resiliency but also to advocate for the need for sustainable long-term funding for Métis citizens across the Homeland. Li Kawnsayr (as well as the future episodes) will add to the growing body of Métis research to showcase Métis ways of knowing and research methodology.

LIGHTNING PRESENTATIONS

Organized by Session and Last Name of Presenting Author

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Determining kinase activation states in chronic lymphocytic leukemia (CLL) through multiplexed targeted mass spectrometry - Poster #775

Presenter: Havva Afshari, University of Manitoba

Other Authors: Victor Spicer, Prasanta Paul, Oleg Krokhin, Thomas Klonisch, Versha Banerji, Sachin Katyal, René P Zahedi

ABSTRACT_ID#2653

ABSTRACT

Introduction: Dysregulated kinases represent a common and effective target in cancer treatment. Phosphoproteomics can decipher signaling networks, but determining specific kinase dysregulation remains challenging. Many kinases are activated by phosphorylation of their 'T-loop', which can be sensitively measured using targeted mass spectrometry (parallel reaction monitoring, PRM). Chronic lymphocytic leukemia (CLL) is incurable even with treatment. BTK inhibitors are dosed daily and costly, often-times have off-target effects, whereby many patients develop resistance. We aim to identify novel and more specific CLL drug targets by profiling kinase activation using T-loop phosphorylation PRM assays. **Methods:** In-house proteome data on 57 CLL patient samples and MEC1 cells was used to develop PRM assays for kinases, T-loop phosphopeptides, and relevant signaling proteins on an Orbitrap Exploris 480. To identify critical kinases and signaling molecules in complementary discovery data, we developed a novel machine learning-based symbolic architecture that reveals causal structures across 'omics data through deformation-aware convergence. We evaluated our two-pronged approach using MEC1 cells treated with BTK inhibitors ibrutinib and acalabrutinib, which differ in specificity and on/off-target profiles. **Results:** We identified 317 protein kinases and 92 phosphatases. PRM assays were developed for phosphorylated/non-phosphorylated T-loop peptides representing >100 kinases, and >20 adaptor proteins and phosphatases. Compared to controls, our assay confirmed known and revealed novel off-target effects of these drugs. **Conclusion:** Our approach using a translational CLL model enables multi-pronged phenotyping of drug responses in a variety of cancers. Integration of PRM with discovery/machine learning approaches may personalize profiling of signaling pathways in patient samples.

LAY SUMMARY

Many cancers, like chronic lymphocytic leukemia (CLL), happen because a communication signal within a cell gets stuck in the "on" position. These signals are controlled by proteins called 'kinases', that act like on/off switches of many processes inside cells. If kinases themselves are too active, they can cause cancer. Because of this, many cancer drugs target kinases – our body has over 500 of them. CLL is the most common blood cancer in the elderly. Current treatments for CLL block one key kinase, but these drugs are expensive and in many patients they stop working over time. We want to find out which other kinases are too active in individual patients, to improve current therapies. Unfortunately, there is no simple and reliable test to check which kinase switches are wrongfully flipped on. We are developing a lab test using a 'mass spectrometry', a technology that lets us measure the activity of over 100 kinases are directly in patients samples. We use artificial intelligence to look for complex patterns in our results. The goal is that our lab test will help doctors to match patients with the best treatments and track how well those treatments are working.

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Blood-based epigenetic instability linked to cancer, aging, and cardiovascular disease - Poster #259

Presenter: Salman Basrai, Ontario Institute for Cancer Research

Other Authors: Ido Nofech-Mozes, Rajesh Detroja, Fernando Scolari, Mehran Bakhtiari, Andrea Arruda, Tracy Murphy, Scott Bratman, Steven Chan, Mark Minden, Jae-Sook Ahn, Dennis Kim, Robert Kridel, Filio Billia, Sagi Abelson

ABSTRACT_ID#2624

ABSTRACT

Rationale: The abundance, dynamics, and context-dependent heterogeneity of DNA methylation—where a pattern considered abnormal in one cell type may be normal in another—complicate the identification of early methylation changes that drive or signal disease development. This complexity can obscure early markers of increased cancer risk, making it challenging to detect and intervene in carcinogenic processes at their inception. **Approach:** We studied DNA methylation instability by conducting genome-wide analyses to identify CpG sites with highly consistent methylation profiles in healthy blood cells. We examined alterations at these stable loci in a wide variety of hematological cancer cohorts. **Results:** Our findings reveal that hypermethylation of these CpG sites is ubiquitous across myeloid and lymphoid malignancies, correlates with clonal burden and mutation frequency dynamics throughout treatment, and exhibits unique patterns that reflect the cell of origin. In non-cancer cohorts, we observed that methylation levels at epigenetically stable loci become increasingly variable with age—a shift also linked to higher cardiovascular disease risk and lower survival rates. Additionally, our results indicate that highly stable CpGs are enriched in the promoters of age-related genes, frequently overlapping key transcription factor binding sites, suggesting that their disruption contributes to the altered gene expression observed with aging. **Impact:** This study highlights DNA methylation instability as a blood-based biomarker for both cancer and cardiovascular disease and uncovers a mechanistic link between methylation dynamics and the expansion of maladaptive hematopoietic clones. These results have significant implications for early cancer detection and estimating disease risk in healthy individuals.

LAY SUMMARY

DNA methylation – chemical modifications of the nucleotides in DNA – represents a valuable layer of information that can be used to study diseases. However, methylation profiles are highly variable across different cell types and they can evolve over time, making it difficult to identify informative markers of increased cancer risk that are broadly applicable. We studied genome-wide DNA methylation profiles in thousands of blood samples derived from healthy individuals and patients with a wide variety of blood cancers. We identified genomic loci which exhibit consistently low methylation levels in healthy individuals and become abnormally hypermethylated in cancer, aging, and cardiovascular disease. Our findings reveal that this phenomenon of DNA methylation instability is correlated with disease progress in leukemia patients and can be used to track changes in the size of cancer cell populations, similar to somatic mutations. Additionally, DNA methylation instability increases gradually even in healthy aging individuals and is a risk factor for various cardiovascular complications. These results have significant implications for early cancer detection, monitoring residual disease in patients undergoing treatment, and estimating disease risk in healthy individuals.

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Development of a Lipid Nanoparticle based Anti-BCMA CAR-NK Therapy for Multiple Myeloma - Poster #375

Presenter: Angela Hamie, University of Alberta

Other Authors: Bee Tan, Justine Lai, Carina Debes Marun, Xiuying Hu, Pankaj Tailore, Michael Chu

ABSTRACT_ID#3109

ABSTRACT

Introduction: Chimeric Antigen Receptor (CAR)-T cell immunotherapy has demonstrated promise in treating multiple myeloma (MM), however it carries severe risks such as cytokine release syndrome (CRS), neurotoxicity, and T cell malignancies. Additionally, viral vector based CAR delivery poses concerns of immunogenicity and insertional mutagenesis. CAR-natural killer (NK) cells offer a safer alternative, reduced toxicity and allogeneic "off-the-shelf" use. Furthermore, lipid nanoparticles (LNPs) present a non-viral and cost-effective method for delivering mRNAs, enabling safe and scalable CAR therapy production. **Methodology:** mRNA CAR constructs, consisting of an IgH-recognizing BCMA, were encapsulated in LNPs and transfected into NK-92 cells or primary NK cells isolated from patient and healthy donors. mRNA LNP delivery efficiency was assessed using fluorescent microscopy, flow cytometry and Western blotting. Functional assays will evaluate the cytotoxicity of the modified NK cells against BCMA-expressing MM cell lines. **Results:** A formulation containing lipid 5, β -sitosterol, DSPC, and PEG 2000 (50:38:10:2) demonstrated superior transfection efficiency in NK cells compared to formulations containing SM-102, MC-3, or cholesterol. 70-90% mRNA transfection efficiencies were achieved in NK cells expanded in vitro over 7-10 days. Anti-BCMA CAR expression was confirmed through fluorescent microscopy, flow cytometry, and Western blot. Current studies are evaluating the cytotoxicity of the modified NK cells against MM cell lines. **Conclusions:** Optimized mRNA LNP encapsulation allowed for high transfection efficiencies in primary NK cells, confirming the feasibility of this non-viral approach for CAR engineering. This safer alternative to viral vector-based CAR-T therapy supports the development of allogeneic CAR-NK therapies for MM treatment.

LAY SUMMARY

We are developing a safer and more accessible type of cancer immunotherapy called CAR (Chimeric Antigen Receptor)-T therapy, where a type of cancer fighting immune cells, called T cells, are taken from patients and engineered in the lab with a "CAR" gene that helps them better target and destroy cancer cells. Rather than using traditional T cells, which may cause serious side effects, we are using natural killer (NK) cells—a safer alternative with similar cancer-fighting abilities. Because NK cells can be collected from healthy donors and stored, they offer the potential for a readily available, off-the-shelf therapy that improves patient access. Furthermore, instead of relying on the traditional viruses to deliver the "CAR" gene—an approach that is both costly and potentially risky—we use lipid nanoparticles, which are tiny particles that offer a safer and more scalable delivery method. So far, we have successfully engineered these modified NK cells in the lab and are now testing their ability to target and kill cancer cells in laboratory models, with plans to expand to animal studies. This work brings us closer to developing safer, scalable, and more accessible cancer therapies.

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Single cell profiling of PR subclones defines high-risk disease in multiple myeloma - Poster #781

Presenter: Noemie Leblay, University of Calgary

Other Authors: Sungwoo Ahn, Alexandra Poos, Mehmet Samur, Sheri Skerget, Holly Lee, Mansour Poorebrahim, David Jung, Sacha Benaoudia, Elie Barakat, Daniel Penaherrera, Herve Avet Loiseau, Jonathan J. Keats, Nikhil Munshi, Niels Weinhold, Nizar J. Bahlis, Paola Neri

ABSTRACT_ID#3076

ABSTRACT

Multiple myeloma (MM) is the second most common hematologic malignancy characterized by the expansion of malignant plasma cells in the bone marrow (BM). Despite therapeutic advances, MM remains incurable, with a subset of patients defined as high-risk (HR) showing poor outcomes. Identification of HR MM remains challenging due to the lack of prognostic and predictive biomarkers. We here evaluated a new method for identifying HR MM based on the presence of highly proliferative (PR) cells and characterized their transcriptomic signature for potential therapeutic targets. Using the 10x GemCode system, we analyzed the single-cell transcriptomics profile of BM plasma cells obtained from 58 newly diagnosed (ND) and 78 relapse/refractory (RR) MM patients. A PR signature ($\geq 5\%$ of MM cells) was found in 27% of ND and 50% of RR cases, including in standard-risk patients. A low PFS was observed in all patients carrying PR subclones (ND: 30 mos vs 43 mos, $p=0.004$, RR: 63 mos vs 105 mos, $p=0.009$). Of note, PR subclones persisted overtime, and in 44% of patients PR cells enrichment was observed at progression, consistent with Darwinian clonal evolution and therapeutic pressure. Pathway enrichment analysis of PR cells revealed enrichment of MTORC1, MYC targets, and DNA repair pathways. Among the DEGs identified, high ENO1 (alpha-enolase) expression was associated with poor PFS, suggesting its role in HR. The presence of PR cells identifies HR disease and correlates with poor survival at diagnosis and progression. Their identification can improve disease prognosis and characterizing their molecular signature may offer therapeutic possibilities.

LAY SUMMARY

Multiple myeloma (MM) is the second most common blood cancer characterized by the presence of cancerous plasma cells in the bone marrow. It is an incurable disease, and some patients have a more aggressive form of MM, called high risk, that responds poorly to treatment and is associated with shorter survival. In this study we have developed a method that can identify these high-risk cancer cells in newly diagnosed and relapsed MM patients. We have shown that these cells persist overtime and their presence is associated with poor outcomes. Furthermore, we have investigated the unique features of these high-risk cancer cells and identified specific signals that can be potentially targeted to generate more effective treatments for these patients.

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Identifying new metabolic vulnerabilities in CBFA2T3::GLIS2+ acute megakaryoblastic leukemia - Poster #581

Presenter: Sarah St-Amand, Université de Montréal

Other Authors: Caroline Capdevielle, Mathieu Neault, Alejandro Schcolnik-Cabrera, Zaynab Nouhi, Laura Hulea, Frédérick A. Mallette

ABSTRACT_ID#2875

ABSTRACT

The CBFA2T3::GLIS2 (CG2) chimera is seen in aggressive pediatric acute megakaryoblastic leukemia (AMKL). CBFA2T3 is normally expressed in hematopoietic cells and plays a role in stem cell maintenance and self-renewal. On the other hand, GLIS2, a transcription factor, is not expressed in normal hematopoietic cells. Recently, metabolic adaptations have gathered attention for their therapeutic potential. However, the metabolic reprogramming caused by CG2 and the role of CG2 and its interactors in this rewiring remains unexplored. RNA sequencing of CG2-driven tumors in a mouse model of AMKL revealed a dysregulation of several genes related to pyruvate metabolism. Steady-state analysis of metabolite levels by GC-MS showed an accumulation of pyruvate, aspartate and GABA. Deeper investigations in AMKL human cell lines showed that mitochondrial respiration and glycolysis were affected by the CRISPR-Cas9-mediated knockdown of GLIS2. Based on the transcriptomic data, we investigate the role of PDK2 in AMKL. Using PDK2 shRNA or AZD7545, a PDK inhibitor, we observed a decrease in cell proliferation 7 days post-infection or treatment. To understand how CG2 affects metabolism, we characterized the CG2 interactome by BioID. We discovered numerous transcription factors and chromatin modifiers, including lysine demethylase KDM4A. Targeting by shRNA or QC6352, a KDM4 inhibitor, has a marked negative effect on cell proliferation and synergizes with Navitoclax, a drug already validated as effective in our AMKL models. We propose that CG2 induces metabolic rewiring of hematopoietic cells by a transcriptional role to promote leukemogenesis. These vulnerabilities, undescribed in AMKL, represent potential therapeutic targets to treat this dreadful disease.

LAY SUMMARY

Leukemic cells adapt their metabolism to sustain rapid growth. Directly targeting some of these changes can be a potential therapy. Targeting the metabolic changes makes cells more sensitive to other drugs.

Lightning: A1-Blood Cancers

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 4/6

Delineating the roles of integrin $\alpha 6$ in central nervous system involved acute lymphoblastic leukemia - Poster #567

Presenter: Ami (T.T.Chau) Truong, University of British Columbia

Other Authors: Pascal Leclair, Ling Vicky Li, Gregor Reid, Chinten James Lim

ABSTRACT_ID#3072

ABSTRACT

Acute Lymphoblastic Leukemia (ALL) is the most common pediatric cancer, marked by the uncontrolled growth of immature lymphoblasts in the bone marrow. Despite advances in treatment, the 5-year survival drops from >85% to below 50% when leukemic cells invade the central nervous system (CNS). CNS-directed prophylactic chemotherapy administered to newly diagnosed ALL patients is effective at reducing rates of CNS-ALL, but often results in neurotoxicity, affecting quality of life. Integrins, particularly integrin $\alpha 6$ (CD49f), were shown to play crucial roles in cancer by regulating tumor cell trafficking and promoting drug resistance. While the correlation of $\alpha 6$ with leukemia persistence to chemotherapy is known, its specific roles in leukemia progression and CNS invasion are not fully characterized. We investigated the roles of $\alpha 6$ -integrins in CNS trafficking of NALM6, a neurotropic B-precursor ALL cell line. CRISPR-mediated ablation of $\alpha 6$ reduced broad-spectrum cell adhesion and SDF-1-directed chemotactic migration. In vivo studies in NSG mice revealed that loss of $\alpha 6$ significantly delayed systemic leukemia progression and CNS burden involving various CNS entry pathways. Furthermore, longitudinal monitoring and postmortem analysis suggested that CNS invasion by NALM6 ALL is initiated by early stage leukemic accumulation in the spinal and calvarial bone marrow. These findings elucidate mechanisms driving CNS invasion by leukemic cells, highlighting the importance of integrin $\alpha 6$ in promoting this phenomenon and the potential of utilizing integrin $\alpha 6$ as a target for diagnostic and prognostic strategies in CNS-ALL.

LAY SUMMARY

Acute Lymphoblastic Leukemia (ALL) is the most common cancer diagnosed in children. It arises from uncontrolled growth of immature white blood cells in the bone marrow. When leukemic cells are found to invade the central nervous system (CNS), patients' overall survival rate is reduced from 85-90% to below 50%. It is still unknown what properties of a leukemic cell predispose it to invade the CNS, and treatment options are limited and prone to brain damage. $\alpha 6$ -integrins are specialized adhesion proteins that facilitate cell binding to laminin, a matrix component that is abundant in CNS tissues. It is known to be correlated with leukemia chemotherapy persistence, and it is suspected that high levels of $\alpha 6$ expression promote leukemic cells to invade the CNS. However, direct evidence linking $\alpha 6$ -integrins to CNS-ALL is still lacking. My study found that: Integrin $\alpha 6$ plays crucial roles in facilitating cell adhesion and cell migration of CNS-invasive leukemic cells. Animals bearing cells that lack integrin $\alpha 6$ developed leukemia at a slower pace and had fewer leukemic cells in the brain and spinal cord. The CNS can be breached at early disease stages of leukemia, long before it can be detected in the patient's cerebral spinal fluid.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

Interplay between DOT1L and the Mediator complex regulates glioblastoma stem cell growth - Poster #261

Presenter: Samir Assaf, University of Calgary

Other Authors: Danielle Bozek, Kyle Heemskerk, Amy Banks, Graham MacLeod, Ravinder Bahia, Emilie Cutts, Michael Johnston, Orsolya Cseh, Stephane Angers, Ana Nikolic, Marco Gallo, H. Artee Luchman, Samuel Weiss

ABSTRACT_ID#3044

ABSTRACT

Epigenetic mechanisms that govern normal stem cell fate are dysregulated in glioblastoma stem cells (GSCs). Unraveling the complexity and plasticity of these mechanisms provides an opportunity to harness epigenetics as a tool to reprogram GSC fate and modulate sensitivity to therapeutics. Here, we demonstrate that the histone methyltransferase Disruptor of Telomeric Silencing 1-Like (DOT1L) is a key regulator of GSC stemness and growth through modulation of chromatin accessibility at enhancer elements, notably the cell cycle gene *CyclinD2*. To unravel the underlying epigenetic and transcriptional factors linked to DOT1L regulation in GSCs, we performed a chemogenomic genome-wide dropout screen in the context of DOT1L inhibition. Interestingly, the top hits from this screen were the MED23 and MED24 subunits from the Mediator transcriptional complex. Further investigation identified MED23 as the main driver of GSC growth arrest following DOT1L inhibition by repressing *CyclinD2*, whereas MED24 solely stabilized MED23 incorporation into the Mediator complex tail module. ATAC-seq performed on MED23-knockout GSCs in the context of DOT1L inhibition revealed that MED23 deficiency resists the chromatin accessibility changes induced by the loss of DOT1L epigenetic activity at enhancer elements. Consequently, this enables MED23-knockout GSCs to rescue their growth following DOT1L inhibition by restoring expression of the cell cycle regulator *CyclinD2*. These findings provide critical mechanistic insights into the cooperation between DOT1L and the Mediator complex to regulate GSC growth. Thus, targeting DOT1L in glioblastoma tumors expressing high levels of *CyclinD2* may prove to be beneficial.

LAY SUMMARY

Inhibition of the epigenetic regulator DOT1L promotes glioblastoma stem cell differentiation and growth arrest by restricting chromatin accessibility at enhancer elements corresponding to stemness and cell cycle genes, notably *CyclinD2*. MED23 deficiency resists the chromatin accessibility changes induced by DOT1L inhibition. MED23 is essential for glioblastoma stem cell growth arrest following DOT1L inhibition.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

The proteasome activator, PA28ab, interacts with KIF11 to promote stemness and therapeutic resistance in glioblastoma - Poster #779

Presenter: Kyle Heemskerk, Charbonneau Cancer Institute, University of Calgary

Other Authors: Ravinder Bahia, Samir Assaf, Orsolya Cseh, Fatima Hamood, Gayatri Dronamraju, Xiaoguang Hao, Rozina Hassam, Artee Luchman, Samuel Weiss

ABSTRACT_ID#2789

ABSTRACT

Proteasome activators canonically bind and regulate the proteasome core particle. However, efforts to inhibit the core particle have failed in clinical trials for glioblastoma (GBM). PA28ab is known to bind the core particle and process peptides for MHC class I antigen presentation. Here, we report a non-canonical role for PA28ab in regulating the proteome of GBM stem cells (GSCs). We developed PA28ab knockout (KO) and overexpressing GSCs and found that PA28ab promoted stem cell features of GSCs without altering in vitro growth. PA28ab KO led to decreased sphere formation and improved overall survival in orthotopic xenografts. Transcriptomic and proteomic characterization of PA28ab KO GSCs revealed alterations of endomembrane system and synaptic trafficking machinery. PA28ab pull-down followed by mass spectrometry revealed undescribed interactors of PA28ab in GSCs. We found that PA28ab interacted with KIF11, a kinesin motor protein known to regulate GSC function and normal neuronal growth and migration. Interaction of PA28ab with KIF11 stabilized KIF11 and promoted resistance to KIF11 inhibitors in GSCs, suggesting that PA28ab can non-canonically regulate the stability of specific proteins. Immuno-peptidomic characterization of PA28ab KO GSCs revealed that its role in peptide processing is conserved in GSCs. However, PA28ab knockdown in a syngeneic immunocompetent model of GBM showed a survival benefit, highlighting that its role in peptide processing may be secondary to tumor promotion in GBM. Our findings suggest that PA28ab has cellular roles beyond peptide processing for MHC class I antigen presentation and has potential as a therapeutic target in GBM.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

D-serine facilitates aggressive migration and stemness of recurrent glioblastoma cells by interacting with host endothelial cells - Poster #833

Presenter: Ryan Mota, University of Manitoba

Other Authors: Ping Lu, Emma Martell, Alejandra Llanes Cuesta, Lisa Liang, Sheila K. Singh, Tanveer Sharif, Christopher M. Anderson

ABSTRACT_ID#2919

ABSTRACT

Introduction: Glioblastoma (GBM) cells infiltrate deep brain structures by exploiting perivascular pathways, utilizing stem-like properties and malignant traits to access nutrient-rich environments that support tumor expansion. Our findings suggest that N-methyl-D-aspartate receptors (NMDARs) in brain Endothelial Cells (ECs) play a key role in transducing signals initiated by parenchymal cells, facilitating recurrent GBM progression. **Hypothesis:** D-serine enhances GBM migration and stemness by interacting with host endothelial cells, thereby promoting recurrent tumor aggressiveness. **Methods:** Patient-derived recurrent GBM cells and human cerebral microvascular ECs were co-cultured in a transwell system to assess the role of D-serine in GBM migration and stemness. Pharmacological inhibitors, CRISPR/Cas9-mediated gene silencing, and enzymatic modulation of D-serine were employed to determine the contribution of GBM-derived D-serine and endothelial NMDARs (eNMDARs) to tumorigenesis. Xenograft experiments employed gene silencing or pharmacological inhibitors of Serine Racemase (SRR) to test the effects of recurrent tumorigenesis. **Results:** In silico analysis of patient datasets showed increased SRR transcripts in recurrent GBM tissue which led to a poorer patient outcome. In vitro, ECs potentiate GBM migration and stemness in co-culture. A low D-serine microenvironment was established and led to a significant reduction in GBM migration and stemness markers. NMDAR antagonism and eNMDAR silencing further mitigated these effects. In vivo, SRR silencing, or inhibition, reduced tumor burden at 4 weeks post-injection and extended survival in mice. **Conclusion:** Our findings demonstrate that brain ECs enhance GBM malignancy through D-serine-mediated activation of eNMDARs. This pathway supports GBM migration and stemness, presenting a novel therapeutic target for recurrent GBM treatment.

LAY SUMMARY

Brain blood vessel cells help drive glioblastoma (GBM) spread. We found that endothelial cells (which line blood vessels in the brain) play an active role in helping GBM cells migrate and grow by a D-serine-mediated signalling loop. D-serine boosts tumor stem-like behavior by interacting with endothelial cells. D-serine, produced by GBM cells, makes the tumors more aggressive by enhancing their ability to migrate and act like stem cells through the release of pro-tumor molecules from endothelial cells. These aggressive properties are known to cause the cancerous cells to recur and be resistant to therapy. Blocking D-serine or its targets slows tumor growth. When we reduced D-serine levels or blocked its receptor (NMDAR) in endothelial cells using drugs or gene editing, GBM cells became less invasive in lab and animal models. These approaches also reduced tumor size and improved survival in mice.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

Dual functions of IL-33 orchestrate tumour-associated macrophages in glioblastoma - Poster #859

Presenter: Tala-Maria Mouannes, McGill University

Other Authors: Shyam Menon, Xueqing Lun, Peipei Zeng, Jianbo Zhang, Isabelle Carrier, Eduardo Diez, Alisha Poole, Donna Senger, Stephen Robbins

ABSTRACT_ID#3135

ABSTRACT

Glioblastoma (GBM) is an aggressive brain tumour characterized by a profoundly immunosuppressive microenvironment dominated by tumour-supportive myeloid cells. Our group has identified interleukin-33 (IL-33), a dual-function cytokine with nuclear and extracellular roles, as a key modulator of this innate immune landscape. While full-length IL-33 promotes glioma progression by recruiting immunosuppressive glioma-associated macrophages (GAMs), a nuclear-deficient variant lacking the nuclear localization sequence (IL-33 Δ NLS) halts tumour growth and prolongs survival. This shift is characterized by the emergence of glioma-inhibiting macrophages (GIMs), a pro-inflammatory population with anti-tumour activity. Preliminary cytokine profiling reveals that IL-33 Δ NLS-expressing glioma cells secrete a distinct repertoire of immune-stimulatory factors, including IL-12p40 and TNF α . These cytokines are known to enhance innate immune activation, suggesting that the loss of IL-33's nuclear localization reshapes tumour-intrinsic signaling to drive myeloid reprogramming. Together, these findings position IL-33 as a critical regulator of macrophage polarization in GBM, where the balance between its nuclear and extracellular functions may be key in determining the shift between tumour-promoting and tumour-suppressive immune states. Ongoing studies aim to define the transcriptional programs that distinguish glioma-inhibiting macrophages from their tumour-promoting counterparts using bulk and single-cell RNA sequencing. By integrating molecular profiling with functional co-culture assays, this work seeks to uncover the mechanisms by which IL-33 shapes the tumour immune landscape and to identify targetable pathways for reprogramming tumour-associated macrophages (TAMs) toward a tumour-suppressive phenotype. This approach offers a promising avenue for leveraging myeloid plasticity to improve therapeutic outcomes in glioblastoma.

LAY SUMMARY

Glioblastoma, the most aggressive form of brain cancer, creates a tumour environment that attracts and reprograms immune cells to support tumour growth instead of fighting it. These immune cells—macrophages and microglia—are part of the body's innate immune system and are collectively known as tumour-associated macrophages (TAMs). We discovered that a protein called IL-33, produced by glioma cells, plays a key role in shaping this immune environment. IL-33 has two functions: one inside the cell and one outside. When IL-33 enters the cell nucleus, it promotes tumour growth by converting immune cells into tumour-supporting cells. However, when IL-33 is prevented from entering the nucleus, the tumour shrinks and immune cells become more active in attacking the cancer. Using gene analysis and laboratory models, we are investigating how IL-33 changes the behaviour of immune cells. This research could lead to new strategies to “reprogram” these cells to fight glioblastoma, offering new hope for therapies that target the tumour's immune environment.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

Understanding the Interaction Between Macrophages and Tumor Cells in the Brain Tumor Microenvironment to Enhance Anti-Tumor Immune Responses - Poster #835

Presenter: Mallak Salla, University of Alberta, Cross Cancer Institute

ABSTRACT_ID#2521

ABSTRACT

Mallak Salla, Reza Mirzaei Department Of Oncology, Cross Cancer Institute, University of Alberta Glioblastoma (GBM) is an aggressive primary brain tumor characterized by profound immune suppression within the tumor microenvironment. Among immune cells, macrophages are the most abundant in the GBM microenvironment and are generally expected to possess intrinsic anti-tumor functions. However, these functions are often suppressed by tumor cells. The extensive intratumoral heterogeneity of both macrophages and GBM cells—at single-cell and spatial levels—makes understanding the mechanisms driving their communication a significant challenge. To address this limitation, we developed a novel platform that integrates single-cell RNA sequencing, spatial transcriptomics, and spatial CRISPR screening to investigate macrophage–GBM interactions while preserving the single-cell spatial heterogeneity of the GBM microenvironment. Using published single-cell and spatial transcriptomics datasets, we first identified distinct subsets of macrophages and GBM cells, as well as their anatomical locations. We then analyzed interactions between different macrophage subsets—including monocyte-derived macrophages—and GBM cells, identifying key signaling networks that mediate their communication. Building on these observations, we designed a spatial CRISPR library targeting 50 genes that are highly expressed in tumor cells and have known or potential druggable properties. We aim to use this CRISPR screen to identify genes whose inhibition can reprogram macrophages toward an anti-tumor phenotype and suppress tumor growth in a rodent GBM model.

LAY SUMMARY

Glioblastoma (GBM) suppresses the immune system: We found that glioblastoma, an aggressive brain cancer, creates a complex tumor microenvironment that weakens the body's natural immune response. GBM has developed ways to suppress the anti-tumor functions of the immune cell macrophages. Advanced technology reveals how immune and tumor cells communicate: By combining single-cell RNA sequencing, spatial transcriptomics, and a new CRISPR screening method, we were able to map where different tumor and immune cells are located within the microenvironment, and how they talk to each other to suppress immune responses. Targeting tumor genes to reprogram immune cells: Through the advanced technology techniques utilized in this research, we were able to identify tumor genes that seem to influence immune suppression. We are now testing whether blocking these genes can reprogram macrophages activating them to fight the tumor cells. This discovery could lead to more effective treatments for GBM.

Lightning: A2-Brain Tumours

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 1/2/3

Chimeric antigen receptor macrophages (CAR-M) sensitize glioblastoma models to temozolomide and immune checkpoint blockade combination therapy - Poster #391

Presenter: Shannon Snelling, University of Calgary

Other Authors: Joanna Pyczek, Xueqing Lun, Kristofor Ellestad, Franz Zemp, Laura Mah, Sorana Morrissy, Douglas Mahoney, Jennifer Chan

ABSTRACT_ID#2800

ABSTRACT

Glioblastoma (GBM) is fatal brain cancer in which immunotherapeutic approaches, including immune checkpoint blockade (ICB), have demonstrated little efficacy. Among myeloid phenotypes in GBM, the presence of inflammatory myeloid cells is associated with improved prognosis. Emerging evidence suggests that chimeric antigen receptor macrophages (CAR-M) reverse immunosuppression and sensitize previously resistant peripheral solid tumours to anti-PD-1 ICB. Whether CAR-M can sensitize GBM to ICB, however, has not been explored and is uncertain given the distinct immunologic features of the brain. We have developed syngeneic CAR-M carrying a CAR with novel signaling domains and a scFV against the GBM neoantigen EGFRvIII and have tested the CAR-M in vitro and in vivo. anti-EGFRvIII CAR-M selectively kill EGFRvIII expressing murine GBM cells and secrete inflammatory chemokines and cytokines in vitro. To test CAR-M in vivo, we developed a syngeneic model of GBM (EGFRvIII-OE/CDKN2A-KO/PTEN-KO) which is sensitive to but not cured by temozolomide (TMZ) chemotherapy and is resistant to ICB therapy (anti-PD1 + anti-CTLA4). To test whether CAR-M sensitize GBM to ICB, we delivered intracranial CAR-M or control macrophages (1x10⁶ cells total) on day 14 post-tumour cell implant on the background of TMZ (50mg/kg oral gavage daily day 7-12) and ICB (200ug anti-PD-1 and 200ug anti-CTLA4 intraperitoneally day 7,10,13,16,19). Control macrophage group mice all died by day 40, whereas two CAR-M treated mice survived to day 300 (N=5/group). These results demonstrate that our CAR-M are functionally active in the presence of EGFRvIII expressing cells and have the potential to sensitize GBM to TMZ/ICB therapy.

LAY SUMMARY

Glioblastoma (GBM) is a devastating brain cancer that has no cure. Large phase 3 clinical trials testing single agent immunotherapy for GBM have been disappointing and the field has concluded that combination therapy including immunotherapy will likely be needed to treat GBM. We wanted to know whether a new immunotherapy called chimeric antigen receptor macrophages (CAR-M) can be combined with standard chemotherapy and immune checkpoint blockade immunotherapy to improve GBM treatment. This work discovered that CAR-M therapy in combination with standard chemotherapy and immune checkpoint blockade can prolong survival in mouse models of GBM compared to standard chemotherapy and immune checkpoint blockade alone. We hope in the future that this work will contribute to people living well for longer with brain cancer.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

Exploring Kaiso's role in Androgen Receptor expression in triple-negative breast cancer - Poster #429

Presenter: Stephanie Ali Fairbairn, McMaster University

Other Authors: Kyle Kim, Robert Cowan, Akudo Eze-Onurah, Lindyann Lessey, Ryan Rattan, Salija Golamari, Juliet Daniel

ABSTRACT_ID#3138

ABSTRACT

Triple-negative breast cancer (TNBC) disproportionately affects women of African ancestry (WAA), who experience higher mortality rates compared to white women, despite lower incidence rates. Emerging evidence suggests that an ancestral genetic susceptibility may contribute to this disparity. Our lab identified the transcription factor Kaiso as a potential driver of increased metastasis and mortality in WAA TNBC patients. Notably, 60–80% of TNBC cases also exhibit low androgen receptor (AR) expression, correlating with poor response to AR inhibitors and reduced overall survival. This finding highlights a distinct TNBC subtype—quadruple-negative breast cancer (QNBC). Since *in silico* analyses revealed that high Kaiso and low AR expression are associated with worse survival outcomes in breast cancer patients, we hypothesized that this expression pattern contributes to the elevated mortality observed in WAA with TNBC. Immunohistochemistry of TNBC tissues from WAA and white patients confirmed significantly lower AR expression in WAA and a higher prevalence of AR-negative (QNBC) cases. Chromatin immunoprecipitation indicated that Kaiso associates with the AR promoter, suggesting transcriptional regulation. CRISPR-Cas9-mediated Kaiso knockout in TNBC cells increased AR mRNA and protein levels while reducing expression of the AR target gene ACSL4. Preliminary data revealed that Kaiso knockout TNBC cells are more sensitive to the AR inhibitor enzalutamide and to the chemotherapeutics, cisplatin and doxorubicin. Our findings suggest that Kaiso negatively regulates AR expression and may serve as a therapeutic target. Targeting Kaiso in combination with AR inhibitors could offer a novel strategy to improve outcomes for TNBC patients, particularly among WAA.

LAY SUMMARY

Black women are 40 % more likely to die from breast cancer and this may be due to an ancestral genetic susceptibility and a higher prevalence of the aggressive, triple-negative breast cancer subtype (TNBC). There is a high expression of the protein, Kaiso in TNBC tissues and past research from our lab and others showed that this correlates with a higher likelihood of a poor outcome. Our research shows that Kaiso may reduce androgen receptor (AR) expression in TNBC and make it less responsive to drugs such as enzalutamide that target AR. Clinical trials showed that enzalutamide improves the survival rates in patients who express AR. We used a technique called CRISPR-Cas9 to decrease Kaiso expression in TNBC cells and this resulted in the cells being more responsive to the AR inhibitor enzalutamide and to the chemotherapeutics, cisplatin and doxorubicin. Thus, highlighting the potential to target Kaiso in combination with AR to improve outcomes for TNBC patients.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

Investigation of IDH2 inhibition-associated vulnerabilities in triple negative breast cancer cells - Poster #437

Presenter: Bahareh Heidari, Institute for Research in Immunology and Cancer, Université de Montréal

Other Authors: Nariman Gharari, Roberto Piva, Sylvie Mader

ABSTRACT_ID#3279

ABSTRACT

Introduction: Isocitrate dehydrogenase 2 (IDH2) is a key mitochondrial enzyme in the tricarboxylic acid cycle, catalyzing the oxidative conversion of isocitrate to alpha-ketoglutarate (α -KG) in a reversible manner. WT-IDH2 inhibition was shown to suppress the migration and invasion of mesenchymal triple negative breast cancer (TNBC) cells through accumulation of α -KG, which decreases ATP synthesis and HIF1 α levels, in turn inhibiting glycolysis. We hypothesize that targeting WT-IDH2 can be part of a therapeutic regimen for mesenchymal TNBC tumors. Methods/Results: We used a genome-wide CRISPR knockout lentiviral library to decipher the mechanism of mesenchymal TNBC MDA-MB-231 cell killing by the IDH2 inhibitor, AGI6780. Gene set enrichment analysis revealed that deletion of genes related to the oxidative phosphorylation (OXPHOS) pathway, particularly complex I and V, were synthetic lethal with AGI6780 treatment. Validation experiments confirmed that genetically knocking out of the NDUF51 subunit of complex I or the ATP5O subunit of complex V decreased proliferation of cells in the presence of AGI6780. In addition, pharmacological inhibition of complex V by Bedaquiline increased sensitivity to AGI6780 treatment, evidenced by a drastic reduction in ATP level and increased cell death. Interestingly, although TNBC tumors tend to favor glycolysis, viability of MDA-MB-468 cells, another TNBC model, was also strongly reduced when OXPHOS was disrupted by combination of IDH2 and complex V inhibitors. Impact: Our study contributes to the identification of targets that can synergize for the killing of TNBC breast cancer cells and may lead to novel therapeutic approaches for suppressing metastatic progression of TNBC tumors.

LAY SUMMARY

Targeting TNBC (triple negative breast cancer) energy metabolism: We studied TNBC as an aggressive form of breast cancer that lacks hormone receptors and HER2 expression, limiting the availability of targeted therapeutic options. We focused on a mitochondrial enzyme called IDH2 (isocitrate dehydrogenase 2), which helps cells produce energy. Inhibition of this enzyme reduced the energy supply, and increased cell death. Genome-wide screening identified weak points: Using a genome-wide CRISPR knock out screening in MDA-MB-231 cells in the presence of IDH2 inhibitor, AGI6780, we found that cells became especially vulnerable when we inhibited both IDH2 and parts of the electron transport chain, Complex I and/or Complex V. Therapeutic potential beyond one cell type: This combination approach worked not just in one, but in two different TNBC cell models—suggesting that many TNBC tumors which are typically relying on glycolysis, may still be sensitive to therapies that disrupt mitochondrial function.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

Breast Cancer System Performance: Wait Times, Stage at Diagnosis, and Survival Rates for First Nations and All Other Women in BC - Poster #237

Presenter: Kevin Linn, University of British Columbia

Other Authors: Nadine Caron

ABSTRACT_ID#3190

ABSTRACT

Objectives: This presentation will focus on breast cancer wait times for resolution after abnormal screening mammography, stage at diagnosis, and breast cancer survival, and will discuss potential policy options. **Methods:** A population-based retrospective cohort methodology was utilized using linked administrative health data. Wait time was defined as the percentage of women with an abnormal program screening mammogram result who received their diagnosis (benign or cancer) within the recommended time interval from the screening date (≤ 5 weeks without tissue biopsy, OR ≤ 7 weeks with tissue biopsy). The percentage of incident stage I-IV and stage unknown breast cancers was calculated as a proportion of the total incidence count of all five stages. For survival, age-stratified hazard ratios of the cancer-specific survival rate in First Nations women relative to the survival rate in all other women in the province were calculated. **Results:** A higher proportion of breast cancers among First Nations women were found to be diagnosed at Stage II, while most breast cancers are diagnosed at Stage I among all other women in BC. Delays are seen in the time to resolution of abnormal screens, with First Nations women waiting longer for resolution. Lastly, breast cancer survival rates among First Nations were lower than those of All Other BC residents in the period of 2008-2017, with an age-stratified hazard ratio of 1.36 (95% CI: 1.14-1.62). **Conclusion:** Our findings show disparities in breast cancer care in the province. The cumulative impact of delays and disparities in care may be contributing to lower survival.

LAY SUMMARY

Disparities in breast cancer care exist in British Columbia Targeted interventions and system improvements are needed.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

CD13 Loss Disrupts Tissue Architecture to Promote Cancer Progression - Poster #489

Presenter: Marie-Ève Proulx, McGill University

Other Authors: Rachel Catterall, Shruti Rughani, Luke McCaffrey

ABSTRACT_ID#2420

ABSTRACT

Breast cancer is the most diagnosed cancer among women in Canada. A requirement for breast cancer initiation is the disruption of epithelial tissue architecture. In healthy breast tissue, luminal epithelial cells are highly organized, forming ducts with hollow lumens and well-defined apical-basal polarity. The apical membrane faces the central lumen, and the basolateral membrane faces the extracellular matrix and neighbouring cells. The development of breast cancer is characterized by filling of the lumen and subsequent dispersion of malignant cells from the duct. Despite the knowledge that disrupted tissue architecture is permissive for cancer initiation and progression, our understanding of the mechanisms contributing to this tissue disruption in early cancer remains largely unknown. Our previous research identified CD13, a transmembrane metalloprotease expressed on the surface of epithelial cells, as a pioneer factor essential for lumen formation and maintenance. Notably, CD13 protein levels are reduced in breast cancer. Here, we demonstrate that the loss of CD13 is sufficient to recapitulate early architectural changes observed in breast cancer using a 3D mammary organoid model. In this model, CD13 depletion leads to increased epithelial stratification and loss of lumen integrity. Strikingly, while many oncogenes require long latency to develop cancer, we observed that loss of CD13 dramatically accelerates these steps in a 3D organoid model. These findings uncover a previously unknown role for CD13 as a potent tumour suppressor in the mammary epithelium.

LAY SUMMARY

We demonstrate that the loss of a protein, CD13, can recapitulate the architectural changes observed in early breast cancer in a spheroid model. We found that loss of CD13 dramatically accelerates breast cancer progression by disrupting the structure in a spheroid model.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

Mast cells as biomarkers for capecitabine benefit in triple negative breast cancer - Poster #629

Presenter: Katherine Rich, University of British Columbia

Other Authors: Elahe Shenasa, Dongxia Gao, Ali Bashashati, Torsten Nielsen

ABSTRACT_ID#3054

ABSTRACT

Background: Triple negative breast cancer (TNBC), accounting for 10-15% of cases, carries the worst prognosis and does not benefit from endocrine or HER2 targeted therapies. Clinical trials such as FinXX and CIBOMA found some TNBC patients do benefit from adjuvant capecitabine. RNA expression profiling found mast cell genes predicted capecitabine benefit in these trials. We explore whether deep learning can detect mast cells on hematoxylin & eosin (H&E) stained pathology slides, as an inexpensive predictive biomarker. Design: Using a contrastive learning framework, we trained a deep learning model that can identify mast cells from H&E-stained tissue sections. The model was trained on an internal UBC breast cancer series (n=283) and rigorously evaluated against CD117 positive controls, as well as serial breast cancer tissue microarrays stained with CD117 and tryptase. The model was then applied to tissue sections from the FinXX trial and compared with gene expression and outcome data. Results: The model achieved a balanced accuracy of 89.6%. In the UBC series tissue microarray, model predictions significantly correlated with CD117 and tryptase-stained cores ($R=0.43$, 0.41 respectively, $P<0.0001$). Mast cell predictions were significantly correlated to the average gene expression of mast cell markers ($R=0.34$, $P<0.001$). Comparison with outcome data from the FinXX trial will be presented. Conclusion: We have developed and validated a robust algorithm to detect mast cells from H&E stained tissue to help determine if mast cells are a predictive biomarker for capecitabine benefit in TNBC.

LAY SUMMARY

We found that machine learning can reliably detect mast cell presence from standard tissue images. This gives us a quick and cost-effective way of detecting and evaluating potential biomarkers. Our mast cell classifier was thoroughly validated. It outperformed a pathologist's annotations when compared to gold-standard immunohistochemistry (IHC) stains, showing stronger correlation with the true presence of mast cells. Mast cells may help identify which triple negative breast cancer patients benefit from capecitabine. While past studies relied on gene expression data, our model brings us closer to confirming mast cells as a biomarker for guiding treatment decisions.

Lightning: A3-Breast Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 5/7/9

ER+ signaling in breast cancer: KO by SUMOs? - Poster #197

Presenter: Anaïs Vivet, Immunology and Cancerology Research Institute, Université de Montréal

Other Authors: Antoine Bouchard, Laurent Cappadocia, Sylvie Mader

ABSTRACT_ID#2493

ABSTRACT

Estrogen receptor alpha (ERα) is a key oncogenic transcription factor in breast cancer, expressed in approximately 70% of tumors. Its hormone-driven activation promotes cell proliferation and survival, making ERα a major therapeutic target. Fulvestrant, a clinically relevant pure anti-estrogen, inhibits ERα by inducing its ubiquitination and multi-SUMOylation. While ubiquitination promotes receptor degradation, SUMOylation represses ERα transcriptional activity through destabilization of DNA binding and induction of chromatin closure. Our recent findings further suggest that the E3 SUMO ligases PIAS1 and PIAS2 contribute to ERα SUMOylation in the presence of fulvestrant2, and that the StUBLs RNF4 and RNF111 can induce ubiquitination of ERα in a SUMO-dependent manner. We now hypothesize that the recruitment of a SUMO E3 ligase would be sufficient to induce ERα multi-SUMOylation and downstream transcriptional inactivation. To test this hypothesis, fusion proteins were then generated by linking ERα to either PIAS1 or the minimal catalytic domain of ZNF451 (30 amino acids). SUMOylation of the fusion proteins irrespective of the presence of antiestrogens was confirmed by western analysis in HEK293 and MCF-7 cell lines, and their nuclear localization was validated by immunofluorescence. Luciferase reporter assays showed that both ERα-PIAS1 and ERα-ZNF451 fusion proteins were transcriptionally repressed and exerted dominant-negative effects on estrogen signalling. This inhibition was partially reversed by expression of the deSUMOylase SENP1 or by addition of SUMOylation inhibitors. Together, these results support the suggestion that targeted SUMOylation of transcription factors represents a potential therapeutic approach to suppress the activity of oncogenic transcription factors, including ERα.

LAY SUMMARY

This study shows that fusing ERα to SUMO E3 ligases (PIAS1 or ZNF451) induces its SUMOylation and suppresses its transcriptional activity, even without anti-estrogens. The resulting repression was partially reversible, suggesting SUMOylation directly contributes to ERα inactivation. These findings support targeted SUMOylation as a potential therapeutic strategy to inhibit oncogenic transcription factors like ERα.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

Effect of mtDNA and gDNA on Immune Cell Activation in Colorectal Cancer Subtypes - Poster #791

Presenter: Angie Chen, University of Alberta

Other Authors: David Doell, Siwon Choi, Shayla Mosley, Courtney Mowat, Kristi Baker

ABSTRACT_ID#2333

ABSTRACT

There are two subtypes of colorectal cancer (CRC) known as microsatellite instable (MSI) and chromosome instable (CIN). Patients with MSI CRC have greater survival compared to patients with CIN CRC due to increased activation of cGAS/STING, which is a cytosolic DNA sensing pair that detects the presence of leaked mitochondrial DNA (mtDNA) or genomic DNA (gDNA). MSI CRCs commonly have a mutation in an important mitochondrial protein which induces mtDNA leakage upon dysfunction, therefore we hypothesize that MSI mtDNA will have a more potent cGAS/STING response leading to greater immune activation. We tested this using dendritic cells (DCs) and T cells isolated from C57BL/6 mice along with mtDNA and gDNA isolated from MSI and CIN variants of the mouse MC38 CRC cell line. DCs were treated with varying ratios of mtDNA:gDNA, resulting in increased mRNA expression of cGAS/STING response genes after MSI mtDNA treatment. This was suppressed in STING knock-out DCs as seen with flow cytometry measurements of immune factor expression. Cocultures of mtDNA or gDNA treated DCs with either CD8+ or CD4+ T cells showed varying levels of T cell activation with flow cytometry. CD8+ T cells had greater activation during coculture with mtDNA-treated DCs, indicating a DNA type specific response, while CD4+ T cells had greater activation during cocultures with MSI DNA-treated DCs, indicating a CRC subtype specific response. These results show the importance of mtDNA in the activation of immune cells in response to CRC and present new potential adjuvant therapies for patients with CIN CRC.

LAY SUMMARY

Colorectal cancer (CRC) contains two subtypes called MSI and CIN. MSI patients have a better prognosis compared to CIN patients due to stronger natural immunity potentially caused by the activation of an immune pathway. This immune pathway can be activated by DNA which normally resides in specific cell compartments such as the nucleus or mitochondria, the energy producing component of the cell. This DNA is called mitochondrial DNA and has a bacterial origin. We hypothesize that MSI mitochondrial DNA will trigger the immune pathway more strongly compared to DNA from the nucleus. Results show that treating dendritic cells, which are immune cells vital for triggering the anti-tumour response, with mitochondrial DNA originating from MSI colorectal cancer subtypes causes a greater immune pathway activation compared to treatment with nuclear DNA. Dendritic cells treated with mitochondrial DNA from MSI CRC can strengthen T cell response, which are responsible for killing tumour cells. Currently, we are investigating how mitochondrial DNA enhances immune activation through these immune pathways. Our goal is to better understand why MSI CRC have a stronger immunity than patients with CIN CRC so that we can one day utilize these mechanisms to develop new immunotherapy treatments for CIN CRC patients.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

Incidence and Mortality Impact of New-Onset Depression Among Patients with Colorectal Cancer: A Population-Based Cohort Study - Poster #767

Presenter: Vicki Cheng, University of British Columbia

Other Authors: Eric C. Sayre, Jonathan M. Loree, Sharlene Gill, Rachel A. Murphy, Mary A. De Vera

ABSTRACT_ID#2971

ABSTRACT

Background: The relationship between colorectal cancer (CRC) and depression remains poorly understood. We examined incidence and predictors of new-onset depression following CRC diagnosis and evaluated associations between post-CRC depression onset and all-cause and cancer-specific mortality. **Methods:** Using administrative health data from British Columbia, we conducted a population-based cohort study of individuals diagnosed with CRC from 2012–2018. Depression was identified using ICD-9-codes (296.2, 296.3, 296.5, 300.4, 309.x, 311). We calculated rates of new-onset depression post-CRC diagnosis and used Cox models to estimate hazard ratios (HR) and 95% confidence intervals for depression and mortality outcomes, in CRC patients without history of depression, and with+without history of depression (“full cohort”). **Results:** Among 13,986 CRC patients (44.8% female, mean age 66.0), depression incidence peaked in the first-year post-CRC diagnosis (3.54/100 PY without history of depression; 6.43/100 PY full cohort). In CRC patients without history of depression, predictors of new-onset depression included female sex (HR 1.40; 1.22–1.61) and stage IV (HR 1.77; 1.34–2.33). In the full cohort, predictors included female sex (HR 1.27; 1.14–1.42), stage IV (HR 1.56; 1.26–1.92), and history of depression (HR 8.06; 7.21–9.00). Among CRC patients without history of depression, new-onset depression after CRC diagnosis was associated with 97% higher all-cause (HR 1.97; 1.74–2.23) and 73% higher cancer-specific mortality (HR 1.73; 1.48–2.03), with similar associations observed in the full cohort (all-cause: HR 1.63; 1.46–1.81; cancer-specific: HR 1.51; 1.31–1.73). **Conclusion:** Our findings underscore the importance of identifying and managing depression in individuals with CRC throughout and beyond their care.

LAY SUMMARY

- The link between colorectal cancer (CRC) and depression is not yet well understood. We studied how often people develop depression after being diagnosed with CRC, what factors may increase their risk of depression, and how depression after a CRC diagnosis might affect risk of death.
- In this study of nearly 14,000 people in British Columbia diagnosed with CRC, new cases of depression were highest in the first year after CRC diagnosis—especially among those with a history of depression. We found that certain groups are more likely to develop depression after CRC, including females and individuals with stage IV CRC.
- Depression after CRC diagnosis was also linked to a higher risk of death. For example, in CRC individuals without a history of depression, those who developed depression after their CRC diagnosis had a 97% higher risk of dying from any cause and a 73% higher risk of dying from cancer specifically. These findings highlight the importance of recognizing and addressing depression in people with CRC.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

NOTUM Depletion Restores CD8+ T Cell-Mediated Removal of Colorectal Cancer Cells - Poster #819

Presenter: Julian Chua, University of Calgary

Other Authors: Parnika Sakthivel, Arshdeep Kaur, Elleine Allapitan, Oliver Bathe, Parham Minoo, Arshad Ayyaz

ABSTRACT_ID#2540

ABSTRACT

Colorectal cancer (CRC) is expected to be the fourth most diagnosed cancer and is currently the 2nd leading cause of cancer related deaths in men and 3rd in women in Canada. While immunotherapy has presented promising results against microsatellite high (MSI-H) CRC, the more common subtype, microsatellite stable (MSS) CRC which represents nearly 85% of all CRC cases, is unresponsive to immunotherapy. In this study (Chua et al., 2025 bioRxiv) we aim to elucidate mechanisms contributing to this immunotherapy resistant phenotype. Through comparative single-cell genomic analysis of 160 primary human CRC tumors across 5 datasets I identified a novel cell type unique to MSS CRCs. These cells, which we named WNT inhibitory cancer cells (WICs), evade cytotoxic T cell-mediated recognition by expressing immunotherapy resistant genes such as Notum and Apccdd1. To test the impact of these cells, WICs were ablated using an inducible iCaspase9 system under the control of the Apccdd1 promotor in AKP cOVA mouse derived organoids. This was followed with an adoptive cell therapy model using OT-1 T cells, primed for cOVA. Ablation of this WICs with the addition of adoptive cell therapy treatment significantly increased cancer cell death in AKP cOVA organoids in comparison to organoids containing WICs. Importantly, we have identified NOTUM as a key modulator which inhibits CD8+ T cell-mediated elimination of CRC cells. This project thus reveals a novel cell type unique to immunotherapy resistant type of bowel cancers, identifying new therapeutic targets to improve immunotherapy outcomes for the majority of CRC patients.

LAY SUMMARY

1. Identification of a previously unknown cancer cell type called WNT inhibitory cancer cells (WICs), that is only found in immunotherapy-resistant MSS CRC tumors. These cells enhance tumor immunotherapy resistance by expressing WNT inhibitory genes like Notum and Apccdd1.
2. Selective ablation of WICs with adoptive cell therapy in intestinal cancer organoids significantly increased cell death in comparison. This suggests that removing these resistant cells could make immunotherapies more effective against MSS CRC.
3. NOTUM is a key modulator, which inhibits CD8+ T cell-mediated elimination of colorectal cancer cells.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

Development of a blood-based colorectal cancer screening test using targeted metabolomics - Poster #687

Presenter: Liam Fitzgerald, University of Calgary

Other Authors: Dennis Orton, Karen Kopciuk, Hans Vogel, Robert Hilsden, Oliver Bathe

ABSTRACT_ID#2482

ABSTRACT

Introduction. Colorectal cancer (CRC) screening reduces both disease incidence and mortality. Unfortunately, screening compliance remains low, partly due to the limitations of currently available screening modalities. A potential solution to this problem is a screening blood test based on metabolomics. **Methods.** The Biocrates MxP® Quant 500 targeted metabolomics kit was used to quantify 628 metabolites in sera from a discovery cohort of patients with CRC (n=111), adenomatous polyps (AP; n=63), serrated polyps (SP; n=62), and disease-free controls (DFC; n=154). Multivariate statistical analysis was completed to derive meta-biomarkers capable of predicting the presence and absence of CRC, AP, or SP. A large external screening set (n=838) was used to validate our signatures. **Results.** Discovery meta-biomarkers were derived for CRC, AP, and SP comprised of 31, 32, and 62 metabolites, respectively. Accuracies were >84% and receiver operating characteristic analysis demonstrated areas under the curve (AUROC) >0.85 for all three conditions based on 7-fold internal cross-validation. Performance was preserved on external validation, with AUROCs of 0.96, 0.73, and 0.72 for CRC, AP, and SP, respectively. **Conclusion.** A metabolomics-based blood test designed to screen for CRC and premalignant polyps is promising. Future studies will be directed to further refinement of the biomarkers for AP and SP to enhance specificity and positive predictive value.

LAY SUMMARY

Our metabolomics-based blood biomarkers are uniquely designed to detect premalignant polyps as well as cancer. The signature has significant clinical potential, although further studies are needed to optimize the biomarkers for premalignant polyps.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

Systemic CD4+-Dominant Immune Response Following Liver Resection in Patients with Colorectal Cancer Liver Metastases - Poster #395

Presenter: Shuo Ren, McGill University Health Centre

Other Authors: Oran Zlotnik, Migmar Tsamchoe, Jessica Bloom, Anastasia Tsatoumas, Vered Domankevich, Stephanie K Petrillo, Anthoula Lazaris, Peter Metrakos

ABSTRACT_ID#2688

ABSTRACT

Purpose: Colorectal cancer liver metastases (CRLMs) are a key driver of systemic immunosuppression and a critical determinant of poor prognosis in affected patients. While surgical resection remains the mainstay of treatment, the intricate dynamics of the immune landscape post-resection remain insufficiently characterized. This study aims to delineate the immune reprogramming that occurs following liver resection in CRLMs patients, offering insights into potential therapeutic strategies. **Experimental Design:** Peripheral blood samples were collected from CRLMs patients prior to and following liver resection, once disease-free status was confirmed post-surgery. Immune populations from both adaptive and innate immunity were evaluated via multiparameter flow cytometry, using FlowJo and FlowAI software for analysis. **Results:** Post-surgery, circulating CD4+ T cells, CD28+ CD4+ T cells, and the CD4+/CD8+ T cell ratio were significantly increased. TIM3+ CD4+ T cell levels significantly decreased, while PD-1+ CD4+ T cells exhibited a downward trend, though the change was not statistically significant. No significant changes were observed in the myeloid cell subsets examined pre- and post-surgery; however, elevated levels of S100A9+ M-MDSCs were found in recurrent patients post-surgery. **Conclusions:** Immune reprogramming, characterized by a CD4+ T cell-dominant response and a reduction in the exhausted phenotype, reveals potential mechanisms underlying the reversal of systemic immune suppression following liver metastasis resection. These findings highlight the promise of adoptive CD4+ T cell therapy, particularly when combined with immune checkpoint inhibitors, as a compelling therapeutic strategy to enhance antitumor immunity.

LAY SUMMARY

1. We identify the potential mechanisms driving the reversal of systemic immune suppression following liver metastasis resection. Given the observed systemic immune remodeling, characterized by an increase in CD4+ T cells and alterations in immune checkpoint expression after liver resection, adoptive CD4+ T cell therapy—especially in combination with immune checkpoint blockade—emerges as a promising strategy to enhance immune responses and reduce recurrence in CRCLM patients. This approach underscores the pivotal role of CD4+ T cells, not only as helpers but also in directly driving cytotoxic responses, thereby enhancing anti-tumor immunity after surgery. However, larger-scale clinical trial with extended follow-up is essential to validate these findings and optimize treatment protocols for personalized patient care.
2. In this study, we found that recurrent patients exhibited higher S100A9+M-MDSCs expression following liver resection. This finding aligns with our earlier work, which demonstrated that elevated levels of circulating extracellular vesicle (EV)-associated S100A9, originating from MDSCs, were linked to the poorest prognosis in patients with colorectal cancer liver metastases.

Lightning: A4-Colorectal Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Imperial 8

TGF- β Inhibition as a Strategy to Target Vessel Co-option and Immune Evasion in Colorectal Cancer Liver Metastases - Poster #797

Presenter: Anastasia Tsatoumas, McGill University

Other Authors: Audrey Kapelanski-Lamoureux, Amatzia Gantz, Stephanie K Petrillo, Anthoula Lazaris, Peter Metrakos

ABSTRACT_ID#2488

ABSTRACT

Colorectal cancer (CRC) is the third most commonly diagnosed cancer and a leading cause of cancer-related death, primarily due to liver metastases (LM), which affect over 50% of patients and account for 90% of CRC-related mortality. Surgical resection offers the only potential cure, but only 15–20% of patients are eligible. Current therapies are non-specific and overlook the biological heterogeneity of colorectal cancer liver metastases (CRCLM). Two major CRCLM subtypes exist: desmoplastic lesions, which rely on angiogenesis, and replacement lesions, which co-opt liver vasculature, resist anti-angiogenic therapy, and correlate with poor prognosis. TGF- β signaling promotes vessel co-option via epithelial-to-mesenchymal transition (EMT), the TGF-RUNX1-TSP1 loop, and by fostering immune exclusion. We hypothesize that TGF- β signaling from tumor-associated macrophages (TAMs), hepatocytes, and tumor cells cooperatively drives replacement-type CRCLM by promoting vessel co-option and immune suppression. To test this, we targeted TGF- β signaling from all three sources and selectively depleted TAMs, key TGF- β sources. CRCLM was modeled in C57BL/6 mice via intrasplenic injection of MC38 cells. Mice were randomized into control, TGF- β inhibitor, standard-of-care (SOC: chemotherapy + anti-VEGF), and SOC + TGF- β inhibitor. A separate cohort was treated with anti-CSF1R to deplete TAMs or IgG control. Tumor histology, survival, and immune infiltration were analyzed. Both SOC and TGF- β inhibition modestly improved survival, but only TGF β -treated tumors showed necrosis and stromal remodeling. Anti-CSF1R did not improve survival. Notably, anti-TGF- β or anti-CSF1R increased CD8+ T-cell infiltration, supporting TGF- β 's role in vessel co-option and immune exclusion. Targeting this pathway may enhance subtype-specific immunotherapy in replacement-type CRCLM.

LAY SUMMARY

1. **Liver Metastases Differ in How They Grow and Respond to Treatment:** Two major subtypes of CRCLM exist. Desmoplastic lesions grow by forming new blood vessels (angiogenesis) and can respond to anti-angiogenic therapies. In contrast, replacement-type lesions co-opt existing liver blood vessels, making them resistant to current treatments and linked to worse outcomes. Recognizing these differences is crucial for developing more precise therapies.
2. **TGF- β Signaling Drives Aggressive Tumor Behavior and Immune Suppression:** Our research shows that TGF- β , produced by tumor-associated macrophages (TAMs), liver cells, and cancer cells, plays a key role in enabling replacement-type lesions to hijack liver vessels and avoid immune detection. This signaling supports vessel co-option and keeps immune cells, especially CD8+ T cells, out of the tumor, helping it grow unchecked.
3. **Blocking TGF- β Reactivates Immune Attack in Resistant Tumors:** In our mouse model, TGF- β inhibition led to tumor damage, structural remodeling, and increased CD8+ T-cell infiltration—effects not seen with standard treatment alone. Depleting TAMs also boosted immune infiltration but didn't improve survival on its own. These findings suggest that targeting TGF- β could enhance immunotherapy specifically for replacement-type CRCLM.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

AI for Predicting Pembrolizumab Monotherapy Response in High PDL1 Advanced NSCLC - Poster #65

Presenter: Liton Devnath, BC Cancer Research Centre (BCCRC)

Other Authors: Ian Janzen, Cheryl Ho, Barbara Melosky, Stephen Lam, Ren Yuan, Calum MacAulay

ABSTRACT_ID#2591

ABSTRACT

Pembrolizumab monotherapy is an anti-PD-L1 immunotherapy that is approved as a first-line treatment for non-small-cell lung cancer (NSCLC) patients with high PD-L1 expression ($\geq 50\%$). However, approximately 55% of these patients do not respond. Early identification of likely non-responders is critical to enable timely transition to alternative treatments. This pilot study analyzed a retrospective cohort of NSCLC patients treated with first-line PD-L1 monotherapy, divided into a discovery set (n: 97; 27 non-responders) and an external test set (n: 17; 9 non-responders). Treatment response was assessed using baseline and follow-up CT scans in accordance with RECIST v1.1 criteria. A transfer learning approach was applied to extract deep learning (DL) features from CT images to differentiate responders from progressive disease patients. A log-rank test ranked the discriminative power of the 128 DL features found for the discovery set. Two types of support vector machine classifiers, an SVM with radial basis function (SVM-RBF) and polynomial kernel (SVM-Poly), were evaluated using the top-ranked features. Classifier performance was assessed at three scales: slice, lesion, and patient, and two approaches: with and without clinical descriptors. The SVM-RBF model performed best when trained on the top 10 features combined with clinical data, achieving an AUC of 0.75 (sensitivity: 81.5%, specificity: 81.4%) and an external test set F1 score of 72.9%. Integrating CT-based DL features with clinical descriptors demonstrated more balanced and robust performance, offering a promising tool to identify patients at risk of progression on pembrolizumab monotherapy to support first-line treatment decisions in high PD-L1 NSCLC.

LAY SUMMARY

A pilot study analyzed a retrospective cohort of NSCLC patients treated with first-line PD-L1 monotherapy. Transfer learning approach used to extract deep learning features from CT images. Integrating CT-based DL features with clinical descriptors demonstrated a balanced and robust performance.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

Characterization of the tumor microenvironment in pulmonary adenocarcinoma: a new insight into the biology of Spread Through the Alveolar Space - Poster #803

Presenter: Arnaud Driussi, Université Laval

Other Authors: Fabien C. Lamaze, Mark Sorin, Sébastien Renaut, Victoria Saavedra Armeno, Nathalie Gaudreault, Andréanne Gagné, Dominique Boudreau, William Enlow, Yohan Bossé, Logan Walsh, Philippe Joubert

ABSTRACT_ID#3151

ABSTRACT

Introduction: In lung adenocarcinoma (LUAD), Spread Through Air Spaces (STAS) corresponds to the presence of cancer cells within the lung airways beyond the boundaries of the tumor, and is associated with local recurrence and poor prognosis. However, the mechanisms underlying this dissemination remain unknown. Since STAS may represent a mode of tumor spread, understanding its biology is crucial. We hypothesize that STAS results from an alteration in cellular interactions and signaling within the tumor microenvironment (TME). Our objective is to identify TME and transcriptomic features associated with STAS. **Method:** Tissue microarrays comprising 398 tumors were constructed and the TME was characterized using a 35-antibody panel on the Hyperion mass cytometry imaging platform. Following cell type identification, cellular interaction parameters (cellular neighborhoods, CNs) were evaluated and compared between STAS(-) and STAS(+) groups. In parallel, tumor mRNA from 487 patients was sequenced and gene set enrichment analyses (GSEA) were performed to compare the expression of canonical signaling pathways between the groups. Finally, spatial transcriptomics was conducted on 49 tumor samples to localize STAS-associated signatures within the tissue. **Results:** CN analysis indicated a higher frequency of tumor cell clusters and a lower frequency of neutrophil cluster. GSEA identified alterations suggesting extracellular matrix remodeling. This signature was primarily localized to epithelial regions. **Conclusion:** These results highlight a distinct TME and cellular signaling profile in STAS(+) LUAD. Since STAS represents a form of tumor dissemination, identifying the characteristics associated with STAS will enhance our understanding of the biological mechanisms underlying tumor spread in LUAD.

LAY SUMMARY

In some lung cancers, cancer cells can spread beyond the main tumor into nearby air spaces, a pattern called Spread Through Air Spaces (STAS). STAS is linked to higher risk of recurrence and worse outcomes. By analyzing the tumor microenvironment (the surrounding cells and molecules), we found that STAS-positive tumors have more clusters of cancer cells and fewer clusters of immune cells called neutrophils. This suggests changes in how cells interact within these tumors. We also discovered changes indicating a remodeling of the tissue structure around the tumor. These changes were mainly found in areas of the tumor that are in contact with lung airways.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

Unveiling Molecular Signatures of Arsenic-Induced Lung Damage: A Multi-Omics Approach Using Lung Models and Human Tissues - Poster #159

Presenter: Melis Erkan, Dalhousie University

Other Authors: Kennedy Whelan, Jayme Salsman, Alison Wallace, Graham Dellaire, Victor Martinez

ABSTRACT_ID#2633

ABSTRACT

Introduction: Arsenic, a natural carcinogen, contributes to lung cancer through chronic low-dose exposure. In Nova Scotia, arsenic in drinking water may underlie lung cancer rates, even among non-smokers. Arsenic induces gene expression changes associated with methylation alterations resulting from its biotransformation. We aim to identify biomarkers of arsenic-induced damage using lung models exposed to physiologically relevant arsenic concentrations. **Methodology:** We exposed human bronchial epithelial cells (HBECs) to varying concentrations of sodium arsenite. Cells were treated over 6 passages of 48–96-hour exposure cycles, with one group having arsenic removed after the final passage. DNA and total RNA were isolated from treated cells and sequenced using Illumina's platforms. Bioinformatics analysis will be performed via DRAGEN BioIT (DNA/RNA) and miRMaster (miRNA). **Results:** Coding and non-coding RNAs have been sequenced and processed. Methylation and DNA sequencing are in progress. Data quality assessment and normalization are ongoing for differential expression analysis with DESeq2. Our goal is to identify persistent arsenic-induced gene expression changes maintained during exposure, with a focus on regions exhibiting differential methylation. Next, we will examine irreversible molecular changes by removing arsenic post-exposure. Molecular signatures will be validated using samples from the Nova Scotia Lung Tumour Biobank and lung organoids to model chronic exposure. **Conclusion:** Our multi-omics approach will allow us to detect and correlate the relationship between methylation, transcription expression, and miRNA quantification. With this, we will create the scaffold for the clinically relevant genetic components of arsenic-specific damage and help develop prevention and early detection strategies for arsenic-induced lung cancer.

LAY SUMMARY

Chronic exposure to arsenic, a known human carcinogen, is known to cause cumulative damage to our cells and genetic material. This is thought to be contributing to the rising lung cancer incidences across Canada, especially Nova Scotia. We are hoping to use lab-made human tissues to determine the changes in human genetic material (DNA and RNA) over time, to understand how the damage accumulates. Our DNA and RNA are made of four fundamental building blocks (called “bases”), and we use “sequencing technologies” to determine the exact order of these bases in our samples. Then, we can use computational programs to detect any changes in the sequence of our bases and the genes they encompass, as genes control the creation, function, and quantity of significant molecules in our body. This is because specific changes to our genes can lead to cancer by altering or shutting down protective molecules and systems in our body. By identifying these changes and damages, we can pinpoint the “biomarkers” of arsenic-induced lung cancer. Biomarkers are the distinct signs of molecular damage, which can help us contribute to the development of effective strategies for prevention, intervention, and early detection for this devastating disease.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

Translating Trials to Practice: A Real World Analysis of Effectiveness and Safety of Neoadjuvant Chemoimmunotherapy in Resectable Non-Small Cell Lung Cancer - Poster #101

Presenter: Siddhartha Goutam, University of Calgary

Other Authors: Michelle Dean, Amanda JW Gibson, Vishal Navani

ABSTRACT_ID#2832

ABSTRACT

Introduction: Neoadjuvant chemoimmunotherapy (NCIT) has proven effective in improving overall survival (OS) in patients with resectable non-small cell lung cancer (NSCLC) in pivotal phase III clinical trials. The extent to which trial outcomes reflect real world experience is unknown. We aimed to characterize the complete (PCR) and major pathologic response (MPR) rates of patients treated with NCIT, quantify NCIT completion, adverse events and surgical outcomes of patients treated in routine practice. **Methods:** We utilized the Glans-Look Lung Cancer Research Repository (GLR), a database of all patients with lung cancer in Alberta, Canada. Demographic, clinical, treatment and outcomes data were extracted from the GLR and characterized using univariate methods. **Results:** 64 patients initiated NCIT, in whom median age was 64. 36(56%) were of male sex and 61(95%) were ECOG ≤ 1 . 25(43%) had PD-L1 $> 50\%$ and 18(28.1%) had a tier-1 mutation. 57 (89.0%) completed all prescribed cycles, and 55 (85.9%) proceeded to surgical completion. The objective response rate at pre-operative CT was 70.3%. 54 (98.1%) patients had R0 margins. 19 (34.5%) patients had PCR, and 28 (50.9%) of patients had MPR. 23 (35.9%) of patients had serious adverse events ($\geq$ Grade 3, or requiring ED visit/hospitalization/treatment delay). Operative complications occurred in 20 patients, most commonly air leak/pneumothorax (6), atrial dysrhythmia (4), and kidney injury (2), with median hospital stay of 6 days. **Conclusion:** NCIT is similarly effective and safe in a real world cohort of patients compared to the population in Checkmate 816. Incidence of surgical complications and complications mimicked trial data.

LAY SUMMARY

1. The combination of chemo- and immunotherapy administered preoperatively has proven effective in improving surgical outcomes and overall survival in patients with surgically resectable non-small cell lung cancer. Our study showed that 1/4 patients achieved complete response (i.e. no visible cancer cells in resected tumor specimen) after chemo-immunotherapy prior to surgery.
2. Patients developed toxicities of systemic therapy and surgical complications, however the rate of these were similar to what was described in the trial population.
3. Our findings suggest that the combination chemoimmunotherapy in a real cohort of patients in Alberta, Canada has similar efficacy in achieving tumor killing, and safety compared to the pivotal clinical trials. However we have yet to characterize how long our patients survive, which will require a longer duration of follow up.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

Identifying Novel Modifiers of EGFR Induced Tumourigenesis - Poster #467

Presenter: Jana Jajarmi, BC Cancer Research Centre

Other Authors: Amy Nagelberg, Raj Chari, Lok-In Josephine Ma, William Lockwood

ABSTRACT_ID#2893

ABSTRACT

Lung cancer is the deadliest form of cancer worldwide, and accounts for 24% of all cancer deaths in Canada. The most common subtype of lung cancer is lung adenocarcinoma (LUAD) which is frequently driven by activating mutations in epidermal growth factor receptor (EGFR). While the development of targeted therapies against mutant EGFR (mEGFR) such as tyrosine kinase inhibitors (TKIs) has significantly improved patient outcomes, they are not curative and acquired resistance remains a major challenge. It is therefore essential to identify other genes that cooperate with mEGFR in driving LUAD tumourigenesis so new therapies can be developed. Our lab previously utilized an in vivo Sleeping Beauty (SB) insertional mutagenesis screen to identify such genes, and comparison with human LUAD data from TCGA produced a list of 385 candidate genes mutated in both mouse and human tumours. We then validated these using a targeted CRISPR screen in mEGFR NIH-3T3 cells under multiple conditions including in the presence of clinically used TKI. After stringent analysis we have identified 5 candidate oncogenes with available small molecule inhibitors for further study implicated in driving tumourigenesis, TKI resistance, or both. The results of this work will greatly expand our understanding of mEGFR driven LUAD tumourigenesis, progression, and drug resistance and lay the groundwork for future therapies against this devastating disease.

LAY SUMMARY

- Mutations in the gene epidermal growth factor receptor (EGFR) are very common in lung cancer. This type of cancer is treated using drugs called tyrosine kinase inhibitors (TKIs), but patients become resistant very quickly so we need to find other ways to treat them.
- Our lab has used a combination of genetic approaches to identify other genes that when mutated, can work with mutant EGFR to drive lung cancer formation and progression. We have narrowed down our target list to 5 potential genes that may be related to tumour formation, TKI resistance, or both. Importantly these targets have drugs that shut them down already developed, so the timeframe from research to clinical use is shorter.
- This work will enhance our understanding of mutant EGFR driven lung cancer and TKI resistance, and lay an important foundation for future therapies.

Lightning: A5-Lung Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Doll/Herald

Drug tolerance and persistence to EGFR inhibitor treatment are mediated by an ILK-SFK-YAP signaling axis in lung adenocarcinoma - Poster #231

Presenter: Rocky Shi, BC Cancer Research Institute

Other Authors: Dylan Farnsworth, Christopher Febres-Aldana, Justine Chow, Ravinder Sheena, Tejveer Atwal, Juan Lui Gomez Marti, Samantha Li, Kiersten Thomas, Che-Min Lee, Shannon Awrey, Paul McDonald, Romel Somwar, Shoukat Dedhar, Marc Ladanyi, Kevin Bennewith, William

ABSTRACT_ID#2904

ABSTRACT

Combating resistance to targeted therapy remains a major challenge to improving lung cancer care. Epithelial-mesenchymal transition (EMT) in tumour cells is an established non-genetic resistance mechanism to EGFR tyrosine kinase inhibitors (TKI) that is also associated with worse outcome in patients. Here we demonstrate that integrin-linked kinase (ILK) is an important driver of EMT-mediated TKI resistance in lung adenocarcinoma (LUAD) by promoting a drug-tolerant persister (DTP) cell phenotype. Our results indicate that high ILK expression is associated with EMT in LUAD patients and that genetic suppression of ILK can limit EMT progression and reduce the viability of DTP cells by impairing YAP activation, ultimately improving osimertinib (Osi) sensitivity in LUAD cells. In addition, we found that LUAD cells in extracellular-matrix rich microenvironments have increased dependences on ILK-mediated cytoprotective effects during Osi treatment. Importantly, LUAD cells with high ILK expression are able to persist during EGFR-TKI treatment, progressing through EMT to develop EGFR-TKI resistance. To improve clinical translatability of our findings, we showed that pharmacological inhibition of ILK can suppress EMT and improve Osi response in LUAD cells. Lastly, we found that strong immunohistochemistry staining of ILK in patient biopsies was significantly associated with, and may be used to predict, receptor tyrosine kinase-independent mechanisms of EGFR-TKI resistance. Overall, our results suggest that ILK is an important regulator of EGFR-TKI response and may be exploited as a predictor for acquired resistance, providing evidence for co-targeting ILK with EGFR to better control minimal residual disease and EGFR-TKI resistance in lung cancer.

LAY SUMMARY

Integrin-linked kinase (ILK) is associated with tumour progression and therapy resistance in lung cancer. ILK promotes lung cancer cell survival during initial stages of treatment by activating SFKs and YAP, providing a window of opportunity for lung cancer cells to evolve and become fully drug resistant. Suppression of ILK improves response to therapy and delays the onset of drug resistance in lung cancer.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Revolutionizing Ovarian Cancer Research: Unveiling the Power of 3D Microtissues - Poster #181

Presenter: Breanne Bevelander, University of Saskatchewan

Other Authors: Reza Javan, Parnaz Soori, Taylor Dzikowski, Devrie Johnston, Nima Daneshvar Baghbadorani, Lacey Winstone, M. Dean Chamberlain

ABSTRACT_ID#2401

ABSTRACT

Ovarian cancer is among the most common and deadly cancers in those assigned female at birth. It is complex and heterogeneous, yet primarily studied using simplistic 2D cell culture methods. This oversimplification limits our understanding of chemoresistance, and the tumour microenvironment. To address this, we have developed a novel 3D model, termed “microtissues,” that incorporate spatial complexities often overlooked in 2D cell culture. Preliminary data indicate that microtissues facilitate cell migration and preserve characteristics of tissue architecture that typically are not found in traditional culturing systems. Histological analyses illustrate this in that cells within the microtissues spontaneously organize into structures representative of their histotype. Additionally, we have conducted in vivo testing of microtissues. When ovarian cancer cells are embedded in microtissues prior to injection, they develop tumours more rapidly compared to cells injected with Geltrex. Furthermore, we have focused on cryopreserving our microtissues while retaining their morphology and functionality, allowing us to share our innovative model with collaborators and enhancing the sustainability of our research. Thus far, our microtissues have proven to be readily cryopreservable, maintaining viability upon thawing. Further, within 48 hours of thaw, microtissues appear to normalize their proliferation rate to that of microtissues grown under normothermic conditions. Once confirmed across additional cell lines, cryopreserved microtissues will undergo chemotherapeutic testing to assess whether the cryopreservation process impacts experimental outcomes. With these applications and many more, microtissues have the potential to be incredibly valuable in cancer research and beyond.

LAY SUMMARY

Ovarian cancer is an extremely complex and devastating disease that affects thousands of Canadians each year. This complexity, and the chemoresistance that results from it, must be appropriately modeled in vitro to improve patient outcomes. While simplistic 2D models have been crucial in building our current understanding of cancer biology, a more complex understanding of tumour microenvironment is needed. We have worked to generate a new 3D model, termed “microtissues”, to mimic the complexities of cancers like ovarian cancer. This model has proven useful in mice studies and has shown varied gene expression and chemotherapy response from other cancer models, suggesting microtissues may provide novel insights into cancer biology that other systems fail to capture. The most exciting difference our model has shown is that when ovarian cancers of different types are placed in our microtissues, the cells spontaneously move and form the structures that are characteristic of that ovarian cancer type, making our model a good candidate in the push towards precision medicine. This cell-directed behaviour highlights our model’s potential as a pre-clinical tool towards precision medicine.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Exploiting Multidimensional Synthetic Lethalities to Overcome PARPi Resistance in Ovarian Cancer - Poster #487

Presenter: Mandana Bigdeli, Centre de recherche du Centre hospitalier de l'Université de Montréal (CRCHUM)

Other Authors: Skye Sauriol, Euridice Carmona, Diane Provencher, Anne-Marie Mes-Masson, Francis Rodier

ABSTRACT_ID#3085

ABSTRACT

Introduction: High-grade serous ovarian cancer (HGSOC) is the most common and lethal subtype of ovarian cancer. Despite therapeutic advances, most patients relapse. PARP inhibitors (PARPi) are initially effective in homologous recombination-deficient HGSOCs by exploiting synthetic lethality, but resistance frequently develops due to therapy-induced adaptations. We previously demonstrated that NAMPT inhibitors (NAMPTi) can re-sensitize PARPi-resistant cells, though the underlying mechanism remains unclear. We also showed that PARPi induces a reversible senescence state, which can be exploited with senolytics in a “one-two punch” strategy, currently being tested in a phase I clinical trial (NCT05358639). Here, we hypothesize that the new NAMPTi-PARPi combination triggers therapy-induced cell fate decisions, particularly senescence, leading to enhanced cell death via a temporally coordinated synthetic lethal mechanism. Methods: We evaluate cell fate dynamics in response to NAMPTi-PARPi using isogenic sensitive and resistant HGSOC cell lines, assessing proliferation, apoptosis, mitotic catastrophe, senescence, and the secretome via live-cell imaging, biomarker assays, and mass spectrometry. We identify altered pathways and markers using a multiomics approach (transcriptomic and metabolomic profiling). Resistance markers and therapeutic targets will be validated using multiplexed antibody imaging and patient-derived xenografts. Results: The NAMPTi-PARPi combination re-sensitizes resistant HGSOC cells, induces extensive DNA damage, and triggers senescence-to-death transitions. Transcriptomic data reveal dysregulation of senescence, OXPHOS, cholesterol biosynthesis, and metabolic pathways, which are under validation. Conclusion and Impact: Targeting therapy-induced cell fate adaptations offers a rational strategy to overcome PARPi resistance. This approach supports precision treatment design and may inform therapeutic strategies in other cancers with similar resistance mechanisms.

LAY SUMMARY

Ovarian cancer often becomes resistant to treatment, especially to a class of drugs called PARP inhibitors (PARPi), which are designed to target tumors with specific DNA repair defects. Once resistance develops, treatment options are limited. Our research explores a new two-step strategy to target and eliminate resistant cancer cells. We combine PARP inhibitors with another drug that blocks cancer cell metabolism (called a NAMPT inhibitor). This combination pushes cancer cells into a vulnerable state (senescence), which ultimately leads to their death. This approach re-sensitizes tumors to treatment and could improve outcomes for patients with ovarian cancer that no longer responds to standard therapies. By understanding how cancer cells adapt to therapy at the molecular level, we aim to design smarter, more precise and effective treatment combinations.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Towards in vivo optical fallopian imaging for tubo-ovarian cancer detection - Poster #693

Presenter: Jeanie Malone, BC Cancer Research Institute

Other Authors: Adrian Tanskanen, Lien Hoang, Jessica McAlpine, Pierre Lane, Calum MacAulay, Andrea Neilson

ABSTRACT_ID#3194

ABSTRACT

MOTIVATION: Tubo-ovarian cancers are the most lethal gynecologic cancer; there are currently no effective screening measures. The most common and aggressive tubo-ovarian cancer subtype, high grade serous carcinoma (HGSC) originates in the fallopian tubes before seeding to the ovaries and peritoneal cavity. This work explores the feasibility of endoscopic fallopian imaging for the early-stage detection of tubo-ovarian cancers. **BACKGROUND:** We have previously demonstrated localization of HGSC in resected fallopian tubes with optical imaging catheters. These catheters use optical coherence tomography (OCT) to capture high-resolution (~30µm) three-dimensional imaging of the fallopian lumen. **METHODS:** A pilot study (n=40) is underway to image fallopian tubes in advanced stage tubo-ovarian cancer patients undergoing salpingo-oophorectomy. Under general anaesthesia, the imaging catheter is inserted into each fallopian tube through hysteroscopic guidance and OCT is collected. Feasibility is assessed through cannulation time/success and image quality. Diagnostic potential is assessed through comparison of imaging features with histologic findings mapped along the length of the fallopian tube. **RESULTS:** Preliminary work has demonstrated successful ex vivo cannulation of the fallopian tubes through the uterus. The mucosa is well visualized after a saline flush which separates overlapping plicae. Circumferential stratifications suggest visualization of the myosalpinx. From preclinical studies, we expect HGSC to appear more homogenous and less attenuating compared to surrounding tissue. Recruitment is anticipated to begin summer 2025. **IMPACT:** This study is the first step towards translation of an optical imaging device for the detection of tubo-ovarian cancers, which may ultimately support early detection or new care pathways for patients.

LAY SUMMARY

Ovarian cancer is one of the most lethal gynecological conditions in Canada, owing to a lack of early-stage detection methods. The most common ovarian cancers begin in the fallopian tubes, so we aim to develop tools to examine the fallopian tubes for early signs of cancer. We are conducting a trial imaging the fallopian tubes in tubo-ovarian cancer patients with a small imaging catheter immediately before surgical removal of their fallopian tubes as part of their normal treatment. From looking at resected tissue, we have demonstrated that tubo-ovarian cancers can be seen with this device but need to assess whether this can be achieved in patients to see if this approach could be used to detect early cancers.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Epitype-immune interactions in high-grade serous ovarian cancer - Poster #853

Presenter: Sarah Nersesian, Ottawa Hospital Research Institute

Other Authors: John Abou-Hamad, Emma Durocher, Barbara Vanderhyden, David Cook

ABSTRACT_ID#3358

ABSTRACT

Treatment options for high-grade serous ovarian cancer (HGSC) are limited, and immunotherapy is only effective in ~10% of patients. There is a critical need to better understand the dynamic mechanisms of immune evasion in HGSC, which may be linked to local epithelial cells within tumours. We identified three epithelial phenotypes (“epitypes”: SecA, SecB, Cil) consistently across HGSC tumour samples within an integrated single-cell atlas. Understanding how each epitype interacts with immune cells can inform immunotherapy strategies applicable across HGSC tumours. To explore these interactions, we analyzed gene expression and cell communication using a single-cell RNA sequencing atlas (n=471 patients, ~2 million cells). Predicted co-localization and intercellular signaling were validated using targeted spatial transcriptomics on whole tissues (n=9 tumours) and a tissue microarray (n=101 tumours, n=15 normal). The three transcriptionally distinct epitypes show clinical relevance: SecB cells are present in nearly all samples but enriched in metastatic and chemo-treated tumours. SecB’s gene expression and communication patterns support an immune-modulating role, including association with IFN signaling and class I HLA pathways. Spatial mapping shows SecB often surrounds luminal regions and co-localizes with diverse immune cell types. To functionally validate these findings, co-culture assays are underway using HGSC cells and immune populations isolated from patient ascites, analyzed by flow cytometry. These preliminary findings establish a framework for designing targeted immunotherapies based on epithelial state and suggest that the SecB epitype plays a key role in immune evasion. Targeting SecB-specific mechanisms may improve immunotherapy response in HGSC.

LAY SUMMARY

High-grade serous ovarian cancer (HGSC) often avoids detection by the immune system, and current immunotherapy only works for about 10% of patients. Our research focuses on how different types of epithelial cancer cells may help tumours escape immune attack. We identified three main types of epithelial cancer cells—called epitypes (SecA, SecB, Cil)—across hundreds of HGSC tumours. These epitypes behave differently and interact with immune cells in unique ways. One type, SecB, is especially common in tumours that have spread or been treated with chemotherapy. SecB appears to play a key role in helping tumours evade the immune system. It shows high activity in immune-related pathways and is often found near immune cells in tumour tissue. These findings suggest that targeting the SecB epitype could lead to better immunotherapy options for more patients with HGSC. Co-culture experiments using real patient cells are underway to test this approach further.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Combining novel Ran GTPase inhibitors with PARP inhibitors as a therapeutic strategy in ovarian cancer -

Poster #233

Presenter: Sabrina Ritch, Centre de recherche du Centre hospitalier de l'Université de Montréal (CRCHUM)

Other Authors: Euridice Carmona, Ryhem Gam, Wenying Piao, Isaline Bontemps, Benjamin Péant, Xiaohong Tian, Jian Hui Wu, Diane Provencher, Anne-Marie Mes-Masson

ABSTRACT_ID#2818

ABSTRACT

High grade serous ovarian cancer (HGSC) is characterized by high degrees of aneuploidy. We have shown that downregulation of the small GTPase Ran decreases cell proliferation and promotes apoptosis of aneuploid cells, with little effect on normal diploid cells, making RAN a promising therapeutic target for HGSC. We have developed a series of novel small-molecule inhibitors of Ran (RANi) that disrupt aneuploid HGSC cell line proliferation without affecting normal diploid cells, while also inhibiting the homologous repair (HR) pathway. Resistance to PARP inhibitors (PARPi) is thought to involve the reactivation of the HR pathway in cancer cells. Thus, combining PARPi with drugs that inhibit the HR pathway offers a potential strategy to overcome this resistance. This study aims to test various RANi alone or in combination with Olaparib (PARPi) on ex vivo live human ovarian cancer (OC) samples using microfluidic models. Specifically, micro-dissected tumors (MDTs) of OC xenograft and patient samples were loaded into microfluidic devices and treated with selected RANi inhibitors and/or Olaparib. MDTs were then formalin-fixed, paraffin embedded and analyzed via immunofluorescence for proliferation (KI67) and apoptosis (cleaved-caspase 3 (CC3)). Preliminary results demonstrated a dose-dependent decrease in KI67+ cells, followed by an increase in CC3+ cells within the tumour epithelium of OC MDTs treated with RANi alone compared to controls. MDTs treated with a combination of Olaparib and RANi demonstrated higher levels of apoptosis than with each inhibitor alone. While a larger sample cohort needs to be studied, our data suggest a potential therapeutic efficiency of this combination.

LAY SUMMARY

A distinct feature of ovarian cancer cells is their abnormal number of chromosomes. We have shown that blocking the protein Ran causes death in cells with an abnormal number of chromosomes but not in normal cells. Therefore, we are developing new drugs to target the Ran protein in ovarian cancer cells, to kill tumours without affecting normal tissue. Resistance to PARP inhibitors, a common treatment used against ovarian cancer, involves the reactivation of a DNA repair pathway that is blocked by our Ran inhibitors. Therefore, we aim to test whether combining these two inhibitors can make the treatment more effective. Inhibitors were selected for this project based on their ability to slow the growth of ovarian cancer cells with little effect on normal cells. Our goal is to test the effects of these Ran inhibitors alone or in combination with PARP inhibitors, using tumour samples grown in mice or generously donated by ovarian cancer patients. Using miniature lab models, we have observed that RANi alone caused cancer cells to stop growing and begin dying. Even better, combining RANi with Olaparib led to even more cancer cell death, suggesting this combination could be a powerful new treatment option, pending further testing.

Lightning: A6-Ovarian Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Stephen AB

Driving innovation in ovarian cancer survivorship research through national patient and caregiver engagement: an Ovarian Cancer Canada and Cancer Research Society joint initiative - Poster #733

Presenter: Alicia Tone, Ovarian Cancer Canada

Other Authors: Jessica Lawson, Irina Navarrete, Patient Partners in Research (PPiR), Leslie Born, Dena Edwards, Dajan O'Donnell

ABSTRACT_ID#2795

ABSTRACT

Purpose To develop and implement a national ovarian cancer survivorship research competition driven by the greatest needs of patients and caregivers, while building a foundation for long-term impact on survivorship care and policy. **Methods** Twenty-six patients and caregivers from 9 provinces/territories worked with Ovarian Cancer Canada (OCC) and Cancer Research Society to co-develop a competition framework and 15-question bilingual, anonymous survey to define national survivorship priorities. Results were summarized by counts/percentages or thematic analysis of open-text responses and communicated to researchers as part of the RISE (Research Impact in Survivorship and Engagement) competition launch in April 2025. Proposals will be screened by academic experts (pass/fail), with patients and caregivers driving final rankings and funding recommendations. **Results** We received 134 survey responses (82% EN/18% FR), including from 118 (88%) people with ovarian cancer and 16 (12%) caregivers. 58% of participants were diagnosed between 2020-2024, 35% were diagnosed before age 50, 4% were First Nations, Métis or Inuk (Inuit), 31% had a household income less than \$75,000, 76% were married and 70% were from urban communities. The most disruptive challenges for patient respondents were related to physical (90%), emotional/mental (86%) and sexual (48%) health, navigating healthcare systems (49%) and changes in family dynamics (48%). In response to published competition guidelines based on our findings, we received 25 Expressions of Interest from 5 provinces; 19 (76%) applicants were new to OCC. **Conclusions** This unique competition driven by individuals directly impacted by ovarian cancer has garnered significant interest from patients, caregivers and multidisciplinary researchers.

LAY SUMMARY

People directly affected by ovarian cancer (as a patient or caregiver) are working with Ovarian Cancer Canada and Cancer Research Society to develop and implement a national funding competition on ovarian cancer survivorship, that is driven by the greatest needs of the community. A national survey of patients and caregivers revealed the most disruptive challenges faced following an ovarian cancer diagnosis, such as impacts on physical, mental/emotional and sexual health, changes in family dynamics and navigating the healthcare system. The open funding call resulting from this survey has attracted researchers from outside of Ovarian Cancer Canada's typical biomedical applicant pool, indicating wide interest in this topic and the value of a patient- and caregiver-driven, multidisciplinary approach.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

Harnessing a marine worm hemoglobin potential to enhance tumor biobanks preservation and 3D living avatars viability for personalized cancer research - Poster #481

Presenter: Maëlle Cottin, Centre de recherche du Centre hospitalier de l'Université de Montréal (CRCHUM)

Other Authors: Samuel Bourdages, Alexandre Bernèche, Guillaume Cardin, Apostolos Christopoulos, Thomas Gervais, Franck Zal, François Yu, Francis Rodier

ABSTRACT_ID#2547

ABSTRACT

Tumour biobanks have been used for decades and play a crucial role in translational biomedical research. However, their quality can be compromised by conditions of ischaemia and hypoxia, affecting tissue integrity, representativeness and viability. Hemarina, a pharmaceutical laboratory, is studying the oxygenating properties of haemoglobin from a marine worm (M101). This non-immunogenic extracellular haemoglobin can transport up to 154 oxygen molecules. Our hypothesis is that the addition of M101 during standardized bio-banking protocols could improve the representativeness, conservation and viability of samples. The first objective is to optimise hypoxic culture conditions in order to assess the oxygenating properties of M101 in vitro. The second objective is to analyse the properties of M101 on the conservation and stabilisation of non-living bio-banks. In the final objective, we will analyse the properties of M101 on increasing the viability and representativeness of living 3D avatars using micro-fluidic devices. We have demonstrated that M101 has significant oxygenating and anti-inflammatory properties in vitro on Pc3 cells. Preservation of PC3 xenografts in a solution supplemented with M101 significantly reduced the expansion of hypoxia and tissue metabolic stress without causing oxidative stress. Finally, the addition of M101 into MDTs culture chips improved their structure, stabilize their size and increase their proliferation over-time. In conclusion, M101 demonstrates significant oxygenating potential in vitro and ex-vivo, supporting its strong potential for the preservation of tumour samples. Its widespread use could revolutionize sample preservation and live avatar models, opening up new avenues for oncology research and personalized medicine.

LAY SUMMARY

M101, a special haemoglobin from marine worms, can deliver much more oxygen than human haemoglobin, and adding it to tumour biobanking protocols helps keep tissue samples healthier and more representative during the pre-analytical period. During our experiments we showed that M101 not only significantly reduce hypoxia and metabolic stress in preserved tumor samples, but also improves 3D tumor models grown on microfluidic chips structural stability and increases cell proliferation over time, making these models more reliable for research. By enhancing both preserved tissue samples and live tumor avatars, the use of M101 could greatly improve how tumour samples are preserved and studied, making research more reliable and opening up new possibilities for personalized cancer treatments.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

Investigating the role of CHD4 in prostate cancer - Poster #255

Presenter: Lingwei (Stephanie) Han, Goodman Cancer Institute, McGill University

Other Authors: Catherine R. Dufour, Anthony Alfonso, Megan Katz, Annabelle Beaumier, Anna Dolgova, Vincent Giguère

ABSTRACT_ID#2511

ABSTRACT

Prostate cancer (PCa) progression is largely regulated through androgen acting on the androgen receptor (AR), leading to a global change in gene transcription. Mammalian target of rapamycin (mTOR) is another key factor in PCa progression and emerging studies revealed that mTOR directly regulates gene expression by associating with chromatin in the nucleus (nmTOR). However, the detailed mechanism on how nmTOR regulates gene expression with AR in PCa remains unclear. Recently our group uncovered a nmTOR-AR functional crosstalk in PCa cells where upon androgen stimulation, nmTOR associates with chromatin and activates an oncogenic metabolic gene program in an AR-dependent manner. Using a proteomic approach referred to as rapid immunoprecipitation mass spectrometry of endogenous protein (RIME), we previously identified the nucleosome remodeling and deacetylase (NuRD) complex as a partner of nmTOR-AR bound on chromatin in PCa cells. Through ChIP-seq, RNA-seq, and ATAC-seq approaches, our investigation reveals a dynamic transcriptional complex formed by CHD4, the catalytic subunit of NuRD, nmTOR and AR on the chromatin in a time-dependent manner. While CHD4's role in PCa's role remains to be investigated, our results revealed depletion of CHD4 triggers a widespread transcriptional rewiring in PCa cells, impacting a significant portion of androgen-response genes and neuroendocrine PCa markers. This underscores the novel function of CHD4 in coordinating with AR and mTOR to regulate gene expression and potentially drive PCa progression and therapeutic resistance.

LAY SUMMARY

- Prostate cancer (PCa) often grows because of signals from a hormone called androgen and its receptor androgen receptor (AR). Treatment often target AR but PCa progresses eventually develop resistance though AR gene mutation or deletion. Currently we don't fully understand mechanisms underlying PCa progression and how PCa can survive without AR.
- Our lab discovered another important factor in cell growth, mTOR, functions along AR in PCa. It's understudied role in the nucleus of cells potentially explains PCa's treatment resistance and mechanism of progression.
- We also discovered CHD4, a protein that controls DNA wrapping and expression, as a player within the AR-mTOR crosstalk. Our results suggest potential crucial role of CHD4 in regulating AR activity and PCa progression toward very aggressive PCa subtypes.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

Androgenic Effects of 11-oxyandrogens in Castration-Resistant Prostate Cancer - Poster #591

Presenter: Ariane Lapointe-Belleau, Centre hospitalier universitaire de Québec-Université Laval

Other Authors: Michèle Rouleau, Lyne Villeneuve, Joseph Descarreaux, Patrick Caron, Caroline Beaudoin, Ashwini Uchil, David Simonyan, Cylia Dahmani, Charles Joly-Beauparlant, Vincent Gélinas, Arnaud Droit, Nicolas Marcoux, Vincent Castonguay, Louis Lacombe, Éric Lévesqu

ABSTRACT_ID#2450

ABSTRACT

Background and Objective: Adrenal-derived 11-oxygenated androgens (11-oxyandrogens) emerged as potential key contributors to prostate cancer (PCa) progression through their biotransformation into potent androgen receptor (AR) agonists. The objective was to obtain clinical and mechanistic evidence of 11-oxyandrogens contribution in patients with metastatic castration-resistant prostate cancer (mCRPC) initiating AR signaling pathway inhibitors (ARPI). **Methods:** A pilot study of 35 mCRPC evaluated serum steroids via mass spectrometry and their association with survival using Kaplan-Meier curves. The capacity of 11-oxyandrogens in driving PCa cell proliferation and its reversal by enzalutamide was assessed, supported by comprehensive transcriptomic and proteomic analyses in CRPC cells. **Key Findings and Limitations:** We highlight 11-ketotestosterone (11KT) and its hydroxylated derivative, 11-hydroxytestosterone (11OHT) as the predominant potent androgens, accounting for 81% of circulating total androgens. Despite the limited sample size, higher baseline levels of 11KT, 11OHT, the abundant precursor 11 β -hydroxyandrostenedione (11OHA4) and the downstream metabolite 11-hydroxyandrosterone (11OHA4), are associated with prolonged progression-free survival (PFS) (hazard ratios (HRs) ranging from 0.56 to 0.69; $P < 0.05$), suggesting an enhanced treatment response. 11KT and 11OHT drive robust AR-mediated proliferative responses of CRPC cells and modulate expression profiles, paralleling canonical androgens. The AR antagonist effectively inhibits their androgenic actions, suggesting they are primarily AR-mediated. Additionally, 11-oxyandrogens display distinct effects, including the activation of potential AR-independent pathways. **Conclusions and Clinical Implications:** These findings show that 11-oxyandrogens are drivers of AR activation and support the clinical relevance of 11-oxyandrogen profiling to help predict response to ARPI in mCRPC, underlining the need for further investigation.

LAY SUMMARY

We studied the contribution of non-canonical androgens originating from the adrenal gland, named 11-oxygenated androgens, in patients with metastatic castration-resistant prostate cancer (mCRPC) initiating treatment. We found that circulating non-canonical 11-oxygenated androgens predominate in mCRPC patients and that patients with higher circulating levels of 11-oxygenated androgens exhibit a better response to treatment and demonstrate improved survival outcomes. We also observed that 11-oxygenated androgens display a strong androgenic influence on cancer cells, by stimulating proliferation and by inducing a shift in gene and protein expression, with some distinctive effects from classical androgens. Profiling androgens including 11-oxygenated androgens may help predict how patients respond to therapies, highlighting their clinical relevance and the need for further study.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

Impact of MAG-EPA supplementation on the prostate tumor microenvironment - Poster #827

Presenter: Lucie Leclair, Université Laval

Other Authors: Gabriel Lachance, Oscar Molina, H       Hovington, Yves Fradet, Karine Robitaille, Vincent Fradet

ABSTRACT_ID#2747

ABSTRACT

Chronic inflammation is increasingly recognized as a significant factor in the development and progression of prostate cancer. In a randomized, controlled phase IIb clinical trial we evaluated the effect of supplementation with eicosapentaenoic acid (MAG-EPA), an anti-inflammatory omega (w)-3, in prostate cancer patients undergoing radical prostatectomy. A total of 130 patients received 3 g/day of MAG-EPA or a placebo for 6–8 weeks before surgery and were followed for one year. MAG-EPA supplementation led to a significant reduction in cancer aggressiveness between biopsy and surgery (termed downgrade) and a lower rate of biochemical recurrence. This study explores the impact of MAG-EPA on the prostate tumor microenvironment and its association with observed clinical outcomes. Fatty acids were profiled in prostate tissue collected during prostatectomy. Tissue microarrays were constructed from tumor and adjacent normal tissues. Immune cell populations were characterized using immunohistochemistry and multiplex immunofluorescence. Fatty acid profiling revealed increased levels of w3-EPA and -DPA, and decreased levels of w6-adrenic and -Osbond acids in the prostate of MAG-EPA supplemented patients. Spatial analyses revealed a greater number and larger lymphocyte aggregates in MAG-EPA-supplemented patients who experienced downgrade ($p=0.03$). These aggregates were enriched in FoxP3+CD8+ lymphocytes ($p<0.001$). These results suggest that MAG-EPA induces a favorable reorganization of immune cells within the microenvironment. Our findings support the potential of MAG-EPA as a complementary therapeutic approach that could delay or avoid invasive standard treatments affecting patients' quality of life. Further analyses of interactions between lipid metabolism and immune modulation will help uncover the antitumor mechanisms involved.

LAY SUMMARY

Omega-3 supplementation could reduce cancer aggressiveness: MAG-EPA, an anti-inflammatory omega-3, taken before prostate surgery was linked to a reduction in cancer aggressiveness between biopsy and surgery. Improved immune response: patients who took daily supplement of MAG-EPA had more and larger clusters of immune cells in their tumors, which can be associated with better cancer control by the immune system. Delay invasive treatments: this approach could help reduce the need for aggressive treatments, improving quality of life for prostate cancer patients.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

Rising Circulating Cell-Free Tumour DNA During the Trajectory of Prostate Cancer Patients Reveals Genomic Alterations Related to Progression - Poster #783

Presenter: Samira Rahimirad, McGill University

Other Authors: Eleonora Scarlata, Lucie Hamel, Seta Derderian, Ginette McKercher, Fadi Brimo, Fred Saad, Armen Aprikian, Simone Chevalier

ABSTRACT_ID#2663

ABSTRACT

Background: Although prostate cancer (PCa) has a high survival rate, several patients progress to late stages. Indicators of progression and therapy response/resistance are key for evaluating treatment efficacy. Longitudinal liquid biopsies (blood) are non-invasive and may be meaningful to monitor progression and reveal underlying mechanisms. We aimed to identify genomic alterations in aggressive PCa primary tumours and track them in patients' blood to capture changes emerging during progression. Methods: Banked fresh frozen prostate tissues from six radical prostatectomy cases with lethal PCa were processed to identify tumour foci and macro-dissect cores of high cellularity (>75%) for DNA extraction. Their serial plasma collections (<4 mL) were used to isolate cell-free DNA (cfDNA), next sequenced along with tumour and matched blood DNAs. Results: Already established and novel copy number variations (CNVs), mostly deletions, were found in primary tumours of the six patients. Longitudinal cfDNA analysis revealed rising circulating tumour DNA (ctDNA) fractions when patients reached the metastatic and late stages. Alterations in ctDNAs were associated with emerging neuroendocrine and stem cell subtypes. Although genomic amplifications were rare in primary tumours, they were common during progression. Furthermore, the clonality of ctDNA alterations were related to treatment resistance over time. Conclusion: The identification of novel genomic changes in tumour DNA and ctDNAs in advanced disease is enhancing our knowledge of PCa severity. Findings on ctDNA underscore the significance of longitudinal liquid biopsy for monitoring disease progression and could contribute to the development of novel diagnostic tests and more effective treatments impacting on PCa lethality.

LAY SUMMARY

The sequencing of serial cell-free DNA from blood of prostate cancer patients is worth adding to clinical tests (PSA) and imaging to monitor disease progression. The genomic profile of each patient's primary tumour is unique at time of radical prostatectomy and remains patient's specific in their liquid biopsies, even if modified later during progression. These findings highlight the importance of integrating blood tumour DNA into Precision Oncology.

Lightning: A7-Prostate Cancer

Sunday, November 2, 2025, 4:30:00 PM - Hyatt Walker/Bannerman

The Discovery of Novel Neuroendocrine Prostate Cancer Biomarkers Utilizing a Proteomic Approach - Poster #537

Presenter: Unnati Yagnik, University of British Columbia

Other Authors: Syam Somasekharan

ABSTRACT_ID#3071

ABSTRACT

Neuroendocrine prostate cancer (NEPC) is an aggressive form of prostate cancer (PCa) with a poor survival rate of 14.3%. NEPC is marked by a cellular transformation to neuroendocrine phenotype and an increased expression of neuroendocrine markers. An earlier detection of NEPC is crucial for its timely intervention and prolong patient life; however, this is hampered by the limited number of biomarkers available to differentiate NEPC from other PCa types. We hypothesize that molecular profiling of PCa patient specimens at the proteomic level will uncover novel NEPC biomarkers. We performed Tandem Mass Tag (TMT)-labelled mass spectrometry to profile the proteome of nine PCa patient specimens. We discovered that one of the nine specimens expresses a significantly higher level of known neuroendocrine markers, CgA, CgB, and DDC; its NEPC features were further validated through immunohistochemistry. We identified a unique set of 39 proteins that are upregulated in this specimen by >2.5-fold change compared to all other specimens. We conducted pathway analysis and a database search which unveiled a set of novel proteins from this subset, including GRN, GNS, KCTD12, and THSD7A, that are associated with neuroendocrine phenotypes and have not been previously studied in NEPC. We intend to further validate these proteins by performing immunohistochemistry on a larger tissue microarray of NEPC and non-NEPC tumors to assess their sensitivity and specificity, thereby further characterizing them as potential biomarkers of NEPC.

LAY SUMMARY

Neuroendocrine prostate cancer (NEPC) is an aggressive form of prostate cancer with a 14.3% survival rate. Unfortunately, there are very few molecular markers that can be used in the clinic to detect the presence of NEPC, which has made it difficult to identify and treat this deadly disease in its early stages. By analyzing the molecular makeup of nine different prostate cancer samples, including one NEPC sample, we found 39 proteins that were unique to NEPC. From this group of 39, we identified four proteins in particular that are linked to neuroendocrine traits and processes; further, none of these four proteins have been previously studied in the context of NEPC. These four proteins hold potential to be utilized to identify the presence of NEPC. This could allow for earlier diagnosis and treatment, prolonging patient life and survival.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

Ash2L: an epigenetic regulator of tumor invasion - Poster #416

Presenter: Arielle Beaudoin, Université Laval

Other Authors: Arianne Gervais, Nafiseh Sadat Mousavi Maleki, Emeline Lelong, Samer Hussein, François Bordeleau, Darren E. Richard, Josée N. Lavoie

ABSTRACT_ID#3062

ABSTRACT

The metastatic migration of tumour cells requires large cellular deformations, which are limited by the size and rigidity of the nucleus. Alterations in chromatin organization allow tumour cells to adjust nuclear mechanical resilience and increase their invasive potential. Metastatic migration also involves the formation of invadopodia, invasive structures that secrete matrix proteases. The formation of invadopodia is coordinated with nuclear changes that promote metastatic migration, through mechanisms that remain poorly understood. We discovered that the phosphorylation of the epigenetic factor Ash2L is involved in the regulation of nuclear mechanical resilience by remodeling chromatin. Moreover, Ash2L is phosphorylated by the oncogene Src, a kinase that regulates invadopodia formation. Our hypothesis is that Ash2L phosphorylation coordinates changes in nuclear organization and invadopodia formation to regulate tumour invasion. The objectives of this study are to analyze the impact of Ash2L and its phosphorylation on invadopodia formation and to investigate the regulation mechanisms by Ash2L. Using genetically modified MDA-MB-231 metastatic breast cancer cell lines, we showed that either the depletion of Ash2L or the expression of a phosphomimetic Ash2LY489E stimulated invadopodia formation. In contrast, the expression of non-phosphorylated Ash2LY489F inhibited these structures. Furthermore, Ash2L regulation of cell mechanics was impaired by epigenetic drugs or the disruption of LINC complexes, molecular connectors for nucleocytoplasmic interactions. These results indicate that Ash2L phosphorylation coordinates nuclear mechanics and invadopodia formation by a mechanism that requires nuclear and cytoskeleton connections. Future transcriptomic analyses will help define novel intermediates involved in Ash2L's role in promoting metastatic migration.

LAY SUMMARY

Deaths associated to solid cancers are primarily due to the spread of malignant cells to distant organs, where they form metastases. In order to succeed, metastatic cells acquire new properties that promote their migration through the confined spaces of surrounding tissues. This metastatic migration requires extreme cellular deformations, which are limited by the stiffness of the nucleus—the organelle that safeguards the genetic material. To navigate these narrow spaces, metastatic cancer cells must adopt various migratory strategies, including altering nuclear stiffness. They also employ specialized structures known as invadopodia, which enable them to widen tissue gaps and create migration tracks. However, the molecular mechanisms regulating these processes remain poorly understood. In our lab, we are working to define a new mechanism involved in metastatic migration. This signaling mechanism involves the modification of a protein that regulates nuclear organization. We discovered that this pathway enables metastatic cancer cells to alter nuclear stiffness and increase their ability to form invadopodia. Ultimately, we hope this study will contribute to a better understanding of metastatic migration and lead to new therapeutic avenues for treating metastatic cancers that develop resistance to commonly used treatments.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

Identification of Molecular Targets Regulating the Entry of Platelet Derived Microparticles - Poster #436

Presenter: Félix Després, Université de Moncton

Other Authors: Gilles A. Robichaud

ABSTRACT_ID#2435

ABSTRACT

Cancer metastasis is responsible for 90% of deaths associated to the disease. Understanding the mechanisms driving metastasis is vital to mitigate mortality rates and improve patient outcome. Our research team has recently reported that cancer cells have the ability to internalize platelet-derived microparticles (PMPs) and gain bioactive content transferred by PMP cargo. As a result, cancer cells gain malignant features (ex: migration and invasion) and impart drug resistance thus contributing to disease progression. Given that the mechanisms enabling cancer cells to internalize PMPs are unfortunately not well understood, we set to identify key surface receptors mediating PMP entry into target cells. Using a combined approach of bioinformatics (artificial intelligence) and mass spectrometry on a series on PMP-permissive and non-permissive target cells, we generated a list of surface receptors as potential PMP internalization mediators. Current validation studies are under way using RNAi silencing approaches on selected receptors to further identify specific mediators of PMP cancer cell entry. Our ultimate goal is to characterize PMP internalization mechanisms to develop new strategic inhibitors against PMP cancer cell internalization processes. As a result, extracellular vesicle (EV) blockade would amputate the cancer cells' ability to benefit from EV transfer of bioactive cargo and their functions driving cancer malignancy.

LAY SUMMARY

Cancer cells have the ability to internalize platelet-derived microparticles (PMPs) and gain bioactive content transferred by PMP cargo. The mechanisms enabling cancer cells to internalize PMPs are unfortunately not well understood. Using a combined approach of bioinformatics (artificial intelligence) and mass spectrometry on a series on PMP-permissive and non-permissive target cells, we generated a list of surface receptors as potential PMP internalization mediators. Our ultimate goal is to characterize PMP internalization mechanisms to develop new strategic inhibitors against PMP cancer cell internalization processes. As a result, extracellular vesicle (EV) blockade would amputate the cancer cells' ability to benefit from EV transfer of bioactive cargo and their functions driving cancer malignancy.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

Soluble CD109 antagonizes EGFR-mediated pro-tumorigenic signaling and functional responses in head and neck squamous cell carcinoma - Poster #528

Presenter: Varsha Reddy Durgempudi, McGill University

Other Authors: Tenzin Kungyal, Anie Philip

ABSTRACT_ID#2677

ABSTRACT

Introduction: Multiple cell-signaling pathways have been dysregulated in SCC, including that of epidermal growth factor receptor (EGFR). Our group has shown that CD109 (a membrane-anchored protein) promotes tumor growth and metastasis in vivo, by activating EGFR signaling. Interestingly, we also showed previously that membrane CD109 (mCD109) is endogenously released from the cell surface; however, the function of released/soluble form of CD109 (sCD109) remains poorly understood. In the current study, we examined whether sCD109 regulates mCD109/EGFR-mediated pro-tumorigenic signaling in HNSCC progression. Objectives: (1) Does sCD109 regulates mCD109-EGFR interactions? (2) Does sCD109 modulates in vitro tumorigenicity? (3) Does sCD109 regulates angiogenesis tube formation? (4) Does sCD109 alter phospho-proteome? Results: (1) HNSCC cells treated with/without sCD109 recombinant protein in the presence/absence of EGF for 15 mins were processed using proximity ligation assay which showed that sCD109 binds with EGFR thereby inhibiting mCD109-EGFR interactions. (2) HNSCC cells allowed to form 3D spheroids in ultra-low attachment plates were treated accordingly for six days wherein we observed that sCD109 inhibits in vitro tumorigenicity. (3) HUVEC cells cultured on solidified Matrigel, were analyzed after 18 hours of treatment, and we observed that sCD109 inhibits angiogenesis tube formation. (4) Phospho-proteome array was used as per the Biotechne-protocol and our findings demonstrated that sCD109 inhibited EGF-induced AKT, ERK, p53 and GSK3 α/β activation. Conclusions: While our team has previously shown that mCD109 is pro-tumorigenic, our current findings suggest that sCD109 opposes pro-tumorigenic effects of mCD109. Together, our findings provide critical insights into understanding of molecular processes driving HNSCC progression.

LAY SUMMARY

Head and Neck Squamous cell carcinoma (HNSCC) is a type of cancer that originates from squamous cells in various organs, particularly in the head and neck region, such as the mouth, throat, and voice box. A protein called CD109, found on the surface of cancer cells, has been found more abundantly in HNSCCs and its increased presence in early abnormal growths is associated with a higher risk of cancer spreading to other parts of the body. Interestingly, CD109 can be released from the cell surface into the surrounding environment. The released form of CD109, referred to as soluble CD109 (sCD109), has been found (in our current study) to inhibit the tumor-promoting effects of its membrane counterpart, mCD109. In laboratory experiments, soluble CD109 blocked the signaling of a protein called EGFR, which is often overexpressed in cancer growth, by binding to EGFR with high affinity. Intriguingly, soluble CD109 could block the cancer cell's ability to form 3D tumor spheroids and nutrients-supplying new microvascular blood vessels formation. Additionally, soluble CD109 also inhibited key markers of cancer progression (such as AKT, ERK). Overall, our findings suggest sCD109 provide critical insights into understanding of molecular processes driving HNSCC progression, potentially improving patient outcomes.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

FLT4, a novel regulator of Renal Cell Carcinoma progression - Poster #434

Presenter: Djazia Haferssas, Université de Montréal

Other Authors: Jonatan Barrera Chimal, Nathalie Henley, Casimiro Gerarduzzi

ABSTRACT_ID#3255

ABSTRACT

Background: Renal Cell Carcinoma (RCC) is among Canada's 10 most frequent cancers. Disruption of p53 function, a critical tumor suppressor, plays a pivotal role in cancer development. While p53 mutations are frequent, these occur at low incidence in RCC. Reactivating this pathway presents a potential therapeutic strategy as MDM2 and MDMX, the major negative regulators of p53, are frequently elevated in RCC patients. However, the molecular mechanisms underlying MDM2/MDMX-mediated p53 regulation in RCC are unclear. Our study identified FLT4 as a novel regulator of MDM2/MDMX and p53 activity. Notably, both FLT4 and its ligand, VEGF-C, are overexpressed in RCC patients. We propose that FLT4 activation enhances the stability of the MDM2/MDMX complex, resulting in p53 inactivation and promoting RCC tumorigenesis. Objective: Determine the molecular mechanisms of FLT4-mediated p53 inactivation in RCC. Methods: 1) Bioinformatic analysis of RNAseq data of FLT4 and VEGFC expression in RCC patients. 2) RCC cell lines (ACHN and Caki-2) treatment with VEGFC and/or FLT4 inhibitor for functional assays, as well as p53 analysis. 3) Transfection with combination of FLT4, MDM2 and MDMX plasmids for p53 axis analysis. 4) RCC cells transduction with Luciferase and FLT4 and injection in immunodeficient mice to study the tumor development and metastatic potential. Results: FLT4 activation stabilizes MDM2/MDMX and degrades p53. This p53 degradation lowers the sensitivity to genotoxic treatments and promotes RCC cell proliferation and migration in vitro and in vivo. Conclusion: Aberrant FLT4 signaling contributes to p53 suppression through the modulation of MDM2/MDMX promoting RCC proliferation and survival.

LAY SUMMARY

- Our study has identified FLT4, a Receptor Tyrosine Kinase frequently deregulated in Renal Cell Carcinoma, as a key player in cancer progression.
- FLT4 and its ligand, VEGF-C, are both overexpressed in Renal Cell Carcinoma, leading to aberrant activation of FLT4.
- The aberrant activation of FLT4 in Renal Cell Carcinoma is observed in tumor epithelial cells and plays an oncogenic role. Specifically, FLT4 activation increases the levels of MDM2 and MDMX, which are negative regulators of p53. This results in enhanced degradation of p53. Since p53 is a crucial tumor suppressor that regulates cell proliferation and survival, its degradation leads to increased proliferation and survival of tumor cells.
- FLT4 was previously known for its role in controlling vascularization. Our study reveals an additional role in promoting cancer progression, specifically in Renal Cell Carcinoma, through the modulation of the MDM2/MDMX/p53 signaling axis.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

Novel Insights into the Clinical Relevance of Perineural Invasion - Poster #448

Presenter: Nickson Joseph, University of Saskatchewan

Other Authors: Paulos Chumala, George Katselis, Anand Krishnan

ABSTRACT_ID#2903

ABSTRACT

Introduction The invasion of cancer cells into peripheral nerves, particularly into the perineural space is called perineural invasion (PNI). Although identified as a major risk factor for cancer progression, the molecular drivers of PNI and its direct clinical relevance are poorly understood. Using our in-vitro PNI model, we characterised the molecular profile of PNI competent breast cancer (BC) cells to reveal the insights into its clinical relevance. **Methods** Dorsal Root Ganglion (DRG)-nerve explants were embedded in Cultrex into which eGFP+ MDA MB 231 cells were seeded to create a 3D co-culture. After 14 days, the explants were isolated and used either for proteomic profiling experiment or immunostaining. Single cell suspension was made from enzymatic digestion of the 14-day old explants. The eGFP+ PNI BC cells were then sorted from the suspension using a cell sorter and proteins isolated from these purified cells were used for LC-MS/MS analysis. **Results** The 2-fold change list of proteins revealed downregulation of major metabolic pathway proteins from glycolysis, pentose phosphate pathway etc. The PNI cells appeared to have unique morphology and did not give positive staining for proliferation marker Ki67. The upregulated proteins like CPLANE-1 and hemopexin may be novel targets for theranostics. **Conclusion** Our findings indicate that BC PNI cells reach a state similar to dormancy where these cells shut down metabolic pathways suggesting the clinical relevance of PNI. Upregulated proteins - CPLANE-1 and hemopexin are potential candidates to target dormant PNI cells in in-vivo experiments or even clinically.

LAY SUMMARY

1. We used an in-vitro model which can better recapitulate perineural invasion (PNI) to study its biology. Previous in-vitro models in the literature lacks intact nerve which the cancer cells could use as a scaffold for PNI.
2. A novel understanding of the underlying cellular and molecular interplay was observed. An insight into the dormancy-like state of breast cancer PNI cells was observed which is novel.
3. We also showed the capacity of the model to screen potential targets of PNI for theranostics.

Lightning: B1-Cancer Biology

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 4/6

Real-Time Imaging and Humanized Models Illuminate gamma delta T Cell Dynamics, Sphingolipid Metabolism, and AML Immunogenicity - Poster #844

Presenter: Raheleh Niavarani, Princess Margaret Cancer Centre, University Health Network

Other Authors: Xi Lei, Sadaf Mazhab Jafari, Lyndsey De Guzman, Shirley Wang, Ralph DaCosta

ABSTRACT_ID#3355

ABSTRACT

Acute myeloid leukemia (AML) remains a clinically challenging malignancy, in part due to the emergence of chemoresistance. To better understand the immunological dynamics underlying AML progression and treatment resistance, we employ intravital microscopy in conjunction with humanized mouse models, allowing real-time visualization of human immune cell behavior within the leukemic bone marrow microenvironment. Our study focuses on the role of gamma delta (gd) T cells, a unique subset of lymphocytes with emerging relevance in cancer immunotherapy. We reveal that $\gamma\delta$ T cells engage in dynamic interactions with AML, exhibiting migratory patterns and cytotoxic activity. Notably, we examine the role of gd T cell function in relation to chemoresistance in AML. Additionally, we show that modulation of sphingolipid ceramide levels in different biological conditions reshapes the immunogenic landscape of AML cells. These findings underscore the importance of gd T cells in the AML microenvironment and suggest new avenues for targeting immune-metabolic pathways to overcome resistance and enhance immunotherapy efficacy.

LAY SUMMARY

gd T cell cytotoxicity and the resulting AML tumor cell death have been visualized for the first time in a humanized mouse model of AML. The bone marrow niche regulates the metabolism of gd T cells and tumor cells, impacting the effector function of gd T cells.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Integrative Transcriptomic Profiling of Metastatic Medulloblastoma using Patient-Derived Xenograft Models - Poster #568

Presenter: Ana Isabel Castillo Orozco, Research Institute of the McGill University of Health Center (RI-MUHC)

Other Authors: Geoffroy Danieau, Livia Garzia

ABSTRACT_ID#3080

ABSTRACT

Medulloblastoma (MB) is a highly aggressive and the most common brain tumor in childhood. MB presents a high intertumoral heterogeneity, with at least four molecular subgroups identified (SHH, WNT, Group 3, and Group 4). Metastatic MB, or Leptomeningeal Disease (LMD), is predominantly found in the MB Group 3 type. Although LMD represents a main clinical challenge, its molecular mechanisms remain poorly characterized. Recent research has shown that primary and MB metastasis diverge dramatically. Our work has focused on establishing therapy naïve Group 3 Patient-Derived Xenografts models of primary and metastatic Medulloblastoma to conduct transcriptomic profiling at the bulk and single-nuclei RNAseq levels to identify genetic drivers/pathways that sustain leptomeningeal disease compartment. Our results show various signaling pathways enriched across LMD models, such as MYC targets, unfolded protein response, and fatty acid metabolism. Using single-sample GSEA analysis (ssGSEA) and deconvolution approaches, we have also identified that our PDXes models retain neoplastic subpopulations previously identified in MB single-cell sequencing studies. Similarly, we have identified slight differences in cell subpopulation proportions between primary and leptomeningeal compartments. Our single-nuclei studies have confirmed these results and differentially expressed genes previously found in bulk RNAseq analyses. These results suggest the presence of cell populations enriched in the metastatic compartment with an aberrant transcription phenotype and adaptations in metabolism to survive the leptomeningeal space. Our recent findings suggest that LMD should be treated differently from primary brain tumors and that identified metabolic pathways may be potential targets for targeted therapeutics to treat or prevent this devastating disease.

LAY SUMMARY

Medulloblastoma is an aggressive pediatric brain tumor that originates mainly from the cerebellum. Unfortunately, when it metastasizes, cancer cells disseminate along the brain coverings (meninges). These tumors spread much like butter on bread. Accordingly, these tumors cannot be surgically removed, which makes it very hard to obtain samples for research. Samples from autopsies are equally complex due to ethical and logistical challenges. Unfortunately, metastatic medulloblastoma is fatal, and no progress has been made in the last 30 years. In our lab, we have developed mice models that can faithfully recapitulate the behavior of metastatic medulloblastoma, allowing us to study it and compare it against primary brain tumors (before it metastasizes). We have demonstrated that primary and metastatic medulloblastoma exhibit significant differences in gene expression, which can provide insights into how to treat metastasis more effectively and design targeted therapies to prevent it. We have utilized state-of-the-art technologies, such as single-cell transcriptomics, to make robust comparisons between primary and metastatic tumors and reveal molecular differences at the cellular level. We have identified molecular patterns and populations enriched in the metastatic compartment, suggesting an aggressive behaviour characterized by aberrant hyper-transcription and exacerbated protein production.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Bioresponsive Immunomodulator Nanocomplex for Selective Immunoengineering in Metastatic Lymph Node

- Poster #398

Presenter: Yueyang Deng, McGill University

Other Authors: Mo Chen, Tianxu Fang, Tianwen Luo, Xiaona Cao, Guojun Chen

ABSTRACT_ID#2586

ABSTRACT

Lymph node (LN) metastasis (LNM) frequently occurs in various cancer types and is associated with high aggressiveness, poor prognosis, and low survival rates. However, effective clinical interventions remain limited primarily due to the distinctive immunosuppressive microenvironment found in metastatic LNs. Targeted delivery of immunomodulators and selective immunoengineering in metastatic LNs offers a promising avenue for repurposing these LNs as an effective anti-tumor system while mitigating the risk of unwanted immune activation elsewhere. Here, we develop a bioresponsive LN-targeted immunomodulator nanocomplex designed to selectively reprogram the immune microenvironment in metastatic LNs for LNM inhibition. The immunomodulator nanocomplex can target LNs due to specific chemokine receptor 7 (CCR7) modification and selectively release anti-PD-1 antibodies (aPD-1) in response to glutathione (GSH) that is found elevated in the extracellular matrix of metastatic LNs. In two mouse models, our data suggested that the immunomodulator nanocomplex can selectively activate T-cell mediated antitumor immune responses in metastatic LNs and thus effectively inhibit tumor growth and prolong the survival of animals. This approach holds significant promises for reducing the necessity for complete LN dissection, thereby presenting a valuable therapeutic option for a broad spectrum of cancer patients.

LAY SUMMARY

1. We created an intelligent protein delivery system that specifically targets lymph nodes. This system carries immune-boosting protein and releases it in response to certain chemical signals found in cancerous lymph nodes.
2. The treatment reactivates the body's immune system within these metastatic lymph nodes, helping T cells recognize and fight the cancer more effectively—without triggering widespread immune responses that could harm healthy tissues.
3. In mouse models, this approach slowed tumor growth and extended survival, showing promise as a less invasive alternative to completely removing lymph nodes during cancer treatment in clinic.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Discovery of Adrenomedullin 2 as a driver of intratumoral cancer cell invasion, intravasation, and metastasis

- Poster #780

Presenter: Seyedeh Sara Karimian, University of Alberta

Other Authors: Konstantin Stoletov, Alice Amitrano, Bishwa Ranjan Si, Varinder Madhav Verma, Gane Ka-Shu Wong, Konstantinos Konstantopoulos, John D Lewis

ABSTRACT_ID#2313

ABSTRACT

Cancer cell intravasation is the key step of metastasis, and the factors that drive this process are poorly known. Intravasation requires both the metastatic cancer cells capable of entering the vasculature and weakened intratumoral blood vessels that serve as “intravasation ports.” Using high-resolution intravital confocal time-lapse microscopy, we discovered that within the primary tumour mass, cancer cells that display increased ATF2 (ATF2^{high}) activity localize to the perivascular regions where they robustly invade and intravasate. ATF2^{high} cells preferably intravasate into the large, torturous tumour-draining veins that serve as preferable ports of cancer cell intravasation. Comparative RNAseq analysis of ATF2^{high} and ATF2^{low} cancer cells revealed that the human Adrenomedullin 2 peptide hormone (ADM2) is significantly upregulated in highly invasive ATF2^{high} cell population. shRNA-mediated knockdown of ADM2 expression drastically abrogated intratumoral cancer cell invasion and intravasation. Importantly, ADM2-deficient tumours showed significantly diminished vasculature size, depriving the metastatic cancer cells of intravasation-competent tumour vasculature. Moreover, ADM2 knockdown inhibited the formation of invasive metastatic lesions, establishing the requirement of ADM2 peptide hormone throughout the metastatic cascade. Our findings identify Adrenomedullin 2 as a novel pluripotent anti-metastasis target that can simultaneously block tumour angiogenesis and cancer cell invasion, intravasation and invasive lesion formation.

LAY SUMMARY

Cancer intravasation—the entry of cancer cells into blood vessels—is a key step in cancer spreading to other parts of the body. Currently, there are no treatments that specifically target this process. Our team developed a cutting-edge imaging system that lets us watch cancer cells invade and enter blood vessels in real time and with high resolution. Using this system and fluorescent markers, we found that cancer cells with high activity of the ATF2 protein cluster around blood vessels and are solely responsible for all intravasation events. These events mostly occur in large (~50 µm) veins deep within the tumour, which we call “intravasation ports.” This pattern was seen in several human cancers, including fibrosarcoma, head and neck, and prostate cancer. To better understand these ATF2-positive cells, we separated them from ATF2-negative ones and compared their gene expression. We discovered that the ADM2 gene is expressed three times more in ATF2-positive cells. When we knock down ADM2 levels, the cancer cells can no longer invade tissue or form intravasation ports. These findings suggest that ADM2 plays a critical role in cancer cell intravasation, and we propose to further study ADM2 as a potential new target for anti-metastatic cancer therapies.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Prolactin and Cholesterol Signaling Crosstalk: a therapeutic target in breast cancer associated bone metastasis - Poster #200

Presenter: Isbel Lopetegui Gonzalez, University of Calgary

Other Authors: Odul Karayazi-Atici, Juan Arteaga, Arvind Singla, Destiny Francis, Frank R Jirik, Sarah Manske, Carrie S Shemanko

ABSTRACT_ID#2674

ABSTRACT

Breast cancer is the most diagnosed cancer among women worldwide and is often associated with osteolytic bone metastasis, characterized by bone destruction. This research explores the connection between prolactin (PRL) signaling and cholesterol metabolism in breast cancer associated bone metastasis. We found that breast cancer cells engineered to secrete human PRL are more susceptible in vitro to the cholesterol-lowering drug, simvastatin (ST), than wild-type or empty vector controls, through a mechanism independent of hydroxy methyl glutaryl Coenzyme A reductase (HMGCR) levels. This sensitivity appears unrelated to breast cancer subtype, as it was observed in both estrogen receptor-positive MCF7 and Her2-positive SKBR3 cells. For in vivo analysis, MCF7 cells secreting PRL (MCF7_LGB_hPRL) and control cells (MCF7_LGB_EV) were injected into the tibias of immunodeficient mice pre-treated with ST for nine days. The treatment continued until day 42. ST significantly increased tissue mineral density (TMD) in mice bearing PRL-secreting tumors, but not in control mice, with little effect on the tumor size. This effect on TMD was also independent of the ability of ST to reduce cholesterol levels in mice. These findings support a functional interaction between PRL signaling and cholesterol metabolism, leading to heightened sensitivity of PRL-secreting tumors to ST, with potential effects not only on the tumor cells but also in their interaction with the bone microenvironment. This may provide a path for personalized therapy.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Elucidating a novel role for the Bcl-2 family member BAD in breast cancer metastasis - Poster #468

Presenter: Leila Pirayeshfard, University of Alberta

Other Authors: John Maringa Githaka, Shu Luo, Han Huang, Arashdeep Saini, Nicolas Touret, Olivier Julien, Ing Swie Goping

ABSTRACT_ID#2642

ABSTRACT

Breast cancer is the most diagnosed cancer in women and metastasis remains the leading cause of mortality. Understanding the molecular mechanisms underlying metastasis will provide tools for improving clinical outcomes. Our lab identified the BCL2 family protein BAD as a novel regulator of metastasis and a prognostic marker for breast cancer patient survival. BAD is regulated by phosphorylation, and using a genetic mouse model wherein the three serine sites are mutated to alanine (BAD3SA), we demonstrated that BAD3SA inhibits mammary epithelial cell migration in both mammary gland development and breast cancer metastasis. BAD3SA only inhibits cell migration in 3D-culture but not in 2D, suggesting regulation of 3D-specific cell motility processes. To gain molecular insight, we used BioID to identify BAD3SA-binding proteins in 2D and 3D culture by mass spectrometry. Reactome analysis of BAD3SA 3D hits identified significant pathways including Rho GTPase and Integrin cell surface interactions. Importantly, the integrin family protein, ITGB1, was the most enriched BAD3SA interactor. Using proximity ligation assay, we validated BAD3SA:ITGB1 interactions. ITGB1 is a cell surface receptor crucial for cell-cell and cell-extracellular matrix adhesion and motility by modulating adaptor proteins, signaling molecules and cytoskeletal components. Notably, BAD3SA diminished actin and tubulin clustering, consistent with impairment of ITGB1-mediated downstream pathways. For functional validation, we will disrupt BAD3SA:ITGB1 interactions using BAD3SA deletion constructs and assess rescue of the invasion defect. Our data suggest that BAD3SA blocks 3D migration/invasion by interacting with ITGB1. Thus, we have uncovered a new regulatory mechanism of cell invasion in breast cancer metastasis.

LAY SUMMARY

Metastasis is the leading cause of death from breast cancer. Understanding what causes cancer cells to migrate is crucial, as it could lead to prevent metastasis by targeting specific molecules involved in the process. Our lab has identified a protein called BAD as a new regulator of cell migration and metastasis. BAD exists in two forms: phosphorylated and non-phosphorylated. We found that the non-phosphorylated form, called BAD3SA, can inhibit cancer cell migration, though the exact mechanism is still unclear. Our research focuses on understanding how non-phosphorylated BAD achieves this by examining its location within the cell and identifying other proteins it interacts with. We discovered that BAD3SA interacts with Integrin beta-1 (ITGB1), a protein on the cell surface that helps cells attach and move through tissues. This interaction appears to disrupt key structures inside the cell, such as actin and tubulin, which are critical for cell movement. These findings suggest that BAD3SA may block cancer cells spread by interfering with ITGB1 signaling. We aim to further explore how this interaction works to help identify new therapeutic targets for preventing metastasis.

Lightning: B2-Metastasis

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 1/2/3

Tol mice overcome the immunogenicity of reporter proteins and enable improved in-vivo cancer-immunity and metastasis studies - Poster #176

Presenter: Areeba Qureshi, University of Toronto

Other Authors: Ping Xu, Hung-Yang Kuo, Ton Schumacher, Iacovos Michael, Robert Kerbel, Kabir Khan

ABSTRACT_ID#2734

ABSTRACT

Using reporter proteins, such as luciferase and fluorescent proteins, is critical for tracking tumour growth and dissemination in-vivo and distinguishing cancer cells from the tumour microenvironment post-implantation. However, the immunogenicity of reporter proteins in immunocompetent syngeneic mouse models often leads to an exaggerated immune response and subsequent rejection of implanted tumour cells, compromising the physiological relevance of these models for therapeutic studies. To address this, we investigated the utility of a previously described transgenic mouse, known as Tol mice, which exhibit immune tolerance to luciferase and various fluorescent proteins. Our study compared the growth of cancer cells implanted into the mammary fat pad (primary tumour) and intracranially (brain metastasis) in both wild-type (WT) and Tol mice. Bioluminescence imaging revealed complete tumour rejection in WT mice, whereas robust tumour growth was observed in Tol mice in both primary and metastatic settings. Interestingly, in WT mice where tumour growth was observed, administration of immune checkpoint inhibitors reinvigorated underlying immunity. Furthermore, in a subset of Tol mice with brain metastasis, a depletion of bioluminescence signal followed by reappearance after eight weeks, suggesting a state of tumour dormancy. These findings highlight that the Tol mouse model can effectively circumvent the issue of reporter protein-induced immune rejection. Moreover, Tol mice enable the establishment of physiologically relevant orthotopic and metastatic tumour models, which will be crucial for generating robust and reliable data on therapeutic responses. Future studies will further characterize the immune landscape in Tol mice and validate their utility across a spectrum of cancer models.

LAY SUMMARY

1. Foreign proteins used to tag cancer cells (reporter proteins) to facilitate imaging and tracking of tumor progression can trigger an immune response in wild-type (WT) mice, leading to false tumor rejection. Here, we employed a novel mouse model tolerant to reporter proteins (Tol mice) to investigate whether we can enhance the current models and observe tumor growth and therapy responses without introducing bias.
2. The Tol mice were permissive to the growth and imaging of cancer cells tagged with reporter proteins, allowing for improved modelling of primary breast cancer and breast cancer brain metastasis. In contrast, reporter-tagged cells were rejected upon implantation in WT mice.
3. The Tol mice enabled a precise examination of the impact of cancer therapies designed to reactivate the immune system to identify and attack cancer cells. In contrast, similar experiments yielded biased results in wild-type mice due to the foreign nature of the reporter proteins.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Understanding cancer prevention knowledge among adolescents and young adults – a cross-sectional study

- Poster #18

Presenter: Jennifer Anderson, Acute Care Alberta

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#3063

ABSTRACT

Introduction: Risk factor reduction is key to improving cancer outcomes, especially as cancer rates rise among youth and young adults. Thus, we examined cancer knowledge in this group to inform health literacy initiatives. Methods: This cross-sectional study used multiphase probability sampling to recruit Albertans aged 16-39 years. We collected self-reported cancer knowledge and behaviours through an electronic questionnaire. Demographic characteristics and cancer prevention knowledge, including awareness of risk factors and screening programs, were described using means and counts. Predictors of cancer knowledge were identified through backwards stepwise logistic regression using Akaike Information Criterion. Multivariable logistic regression estimated the association between health behaviours (i.e., tobacco use, cannabis use, alcohol use, physical activity level) and poor cancer knowledge, with adjustment for confounders identified in the prediction model. Results: The 1,008 respondents were on average aware of 8/11 cancer risk factors assessed. Cigarettes (91%), secondhand smoke (89%), family history of cancer (87%), HPV infection (81%), alcohol (73%), old age (71%), and obesity (67%) were the most well-known risks. Fewer participants recognized sunburns (59%), physical inactivity (56%), low fruit and vegetable consumption (37%), and red meat consumption (36%) as cancer risks. Younger age, living in central (OR: 1.51, 95%CI: 0.87-2.63) or northern (OR: 2.03, 95%CI: 1.08-3.80) Alberta, trades education (OR: 1.57, 95%CI: 0.82-2.98), lower income, and being an ethnic minority (OR: 0.72, 95%CI: 0.48-1.08) were the most relevant predictors of poor cancer knowledge. After adjustment, we estimated that with each unhealthy behaviour, there is a 1.2-fold increase in the odds of poor cancer knowledge.

LAY SUMMARY

Some modifiable risk factors are not well known among Alberta's youth. Only a third of youth were able to recognize dietary choices, including low fruit/vegetable consumption and red meat consumption, as cancer risk factors. Prevention efforts should focus on increasing education about the importance of diet for health and longevity. No one characteristic can perfectly predict cancer knowledge. Instead, cancer knowledge depends on many important social identities including age, education, location, income, and race. Further, some groups are more educated about some risk factors and less educated about others. For example, people with trades education were significantly less likely to recognize cigarettes and second-hand smoke as risk factors; however, compared to people with university and college degrees, they were more likely to rate physical inactivity as a cancer risk factor. After removing the effect of factors like age, education, and income, there is still a clear relationship between cancer knowledge and behaviours. People living unhealthy lifestyles, by smoking cigarettes, using cannabis, consuming more alcohol, and/or infrequently exercising, are less informed about cancer risk factors. In order to increase knowledge and reduce negative consequences of low knowledge, future cancer prevention efforts will need to take a different approach.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Preventing Cancer in the Gender Minority Population: Preliminary Results from a Knowledge Synthesis of Existing Practice - Poster #650

Presenter: Ace Chan, University of British Columbia

Other Authors: Gabriela Kuczynski, Marina Khonina, Hannah Kia, Travis Salway, Fabio Feldman, Trevor Dummer

ABSTRACT_ID#2988

ABSTRACT

Background: Gender minority people are a part of the 2SLGBTQ+ community and are uniquely affected by stigma and discrimination that targets their gender identity and expression. Cancer prevention strategies must be strengthened in communities that experience systemic discrimination and oppression, as these factors can increase cancer risk by contributing to delayed or foregone healthcare. This scoping review aims to synthesize primary and secondary cancer prevention recommendations found in the literature for GM people. **Methods:** A search protocol was developed and refined in collaboration with a Community Advisory Board (CAB) and a Public Health librarian. Five databases—Medline, Embase, CINAHL, PsycINFO, and LGBTQ+ Source—were searched on August 13, 2023, using terms related to gender identity, cancer, and either cancer screening or cancer-causing substance use. Search results were imported into Covidence, a systematic review software. **Results:** A total of 2,660 articles were retrieved using the search terms. The analysis is currently ongoing, and preliminary findings from the title and abstract and full-text screening will be presented. Emerging themes so far include recommendations on GM-specific cancer screening intervals, GM-specific interventions targeting cancer-causing substance use, and the barriers and facilitators influencing GM cancer screening. Barriers to cancer screening include prior negative experiences with healthcare providers and a lack of provider knowledge about gender minority populations and their specific screening needs. **Conclusion:** GM people need consolidated primary and secondary cancer prevention strategies that are culturally tailored to them, and recommendations across prevention policies, programs, and/or activities need to be consistent.

LAY SUMMARY

We need to make cancer prevention efforts stronger in communities, such as gender minority people, that face discrimination and unfair treatment, because these experiences can lead people to delay or avoid getting the healthcare they need—putting them at higher risk for cancer. This scoping review aims to consolidate information about primary and secondary cancer prevention recommendations for gender minority people as preliminary results demonstrate that prior negative experiences with healthcare providers and a lack of provider knowledge about gender minority populations affect their screening uptake.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Preventing Cancer Through Métis Cultural Revitalization: A Framework for Saskatchewan - Poster #236

Presenter: Maria Diaz, University of Saskatchewan

Other Authors: Gary Groot, Calvin Racette

ABSTRACT_ID#2448

ABSTRACT

The Canadian Métis population experiences disproportionately higher cancer rates, leading to reduced life expectancy. Culture is vital to Indigenous health, offering belonging and improving well-being. This community-based study aimed to create a Métis Cultural Continuity Framework for cancer prevention that can be adapted for use in other Métis communities. Participants were recruited from seven Métis Nation–Saskatchewan (MN-S) cultural events held between June 2022 and March 2023, where researchers were engaged in Métis cultural activities (e.g., beading, hide tanning). Semi-structured telephone interviews with 24 MN-S citizens (12 men, 12 women) were analyzed using the Collective Consensual Data Analytic Procedure (CCDAP), enabling the diverse Research Advisory Committee members to identify key themes. These themes included colonization and its impacts, community, cultural revitalization, strength, and pride. Participants highlighted the profound effects of colonization on their lives and communities while celebrating the resilience and solidarity that define the Métis community. Six key themes were used to develop a Métis Cultural Revitalization Framework for Cancer Prevention, rooted in the Red River Cart's symbolism, a significant cultural emblem for the Métis people. Each theme was conceptualized as a spoke of the cartwheel, playing a vital role in ensuring both stability and progress for the cart and the Métis community. For the Métis community, strong cultural connections are essential for fostering a sense of belonging, reducing risk behaviors, and promoting both mental and physical well-being. These factors are vital for a healthy lifestyle and can help prevent diseases such as cancer

LAY SUMMARY

Métis communities experience higher cancer rates and lower life expectancy compared to the general Canadian population. This highlights the urgent need for culturally relevant cancer prevention strategies. Strong cultural connections may reduce risk behaviors that lead to cancer. Cultural continuity appears to enhance holistic well-being, contributing to a healthy lifestyle, promoting health, and potentially preventing cancer. The Métis Cultural Revitalization Framework for Cancer Prevention, symbolized by the Red River Cart, offers a culturally grounded tool that supports health promotion and can be adapted across other Métis communities in Canada.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Evaluating the Readability, Understandability, Actionability and Health Content of Web-Based Patient Education Materials on Cervical Cancer Screening Through an Equity Lens - Poster #686

Presenter: Petra Famiyeh, Women's College Hospital

Other Authors: Mohaddesa Khawari, Shari Dworkin, Aisha Lofters, Gina Ogilvie, Arlinda Ruco, Laurie Smith, Mandana Vahabi, Kimberly Devotta, Melanie Jeffrey, Nadia Mithani, Tara Jeji, Vijayshree Prakash, Carlee Ranger, Sundas Shamshad, Bee Lee Soh, Nila Devi, Harjeet Kaur, Ambreen Sayani

ABSTRACT_ID#2802

ABSTRACT

Background Cervical cancer is preventable, yet inequities in screening access and uptake persist. Public education materials are central to promoting participation but often overlook the informational and cultural needs of diverse populations. To address this gap, we are co-evaluating web-based patient education materials (PEMs) on cervical cancer screening using a health equity lens in partnership with diverse Women and Individuals with a Cervix (WIC) Methods This patient-partnered, interdisciplinary, content analysis examines English-language PEMs focused on cervical cancer screening, HPV self-sampling, and Pap testing sourced from health authority websites across Canada. A coding framework and data extraction domains were co-developed and refined with patient partners. The framework includes three core components: readability (Flesch-Kincaid), understandability and actionability (Patient Education Materials Assessment Tool – PEMAT), and equity-oriented content aligned with Levesque et al.'s five dimensions of access. The coding schema will be systematically applied across all materials. Thematic analysis will inform recommendations for improving the clarity, inclusivity, and accessibility of education materials. Results A total of 53 materials were identified from 11 provincial and territorial websites; none from Yukon or Nunavut. Most materials had a grade 7 reading level or above with only one meeting the optimal grade 6 level. PEMAT and accessibility scoring are underway. Findings will be analyzed with patient partners to co-develop evidence-informed recommendations for improving public health messaging on cervical cancer screening. Conclusion Outputs of this study include the co-development of a guideline document for health agencies seeking to create public-facing resources that are inclusive and accessible.

LAY SUMMARY

Public outreach and awareness materials play a key role in cervical cancer screening participation Reviewing the materials used to promote cervical cancer screening will help us identify if they are easy to read, understand and act on by the public Our study findings will inform the co-creation of a public outreach messaging guideline to support public health agencies improve accessibility of their screening programs.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Mobilizing Community Health Ambassadors to Support HPV Self-Screening Among Underscreened Populations in Alberta - Poster #282

Presenter: Madison Fullerton, Praxus Health

Other Authors: Gina Vetro, Caitlin Ford, Chelsea D'Silva, Holly Dievert, Bonnie Chiang, Sisi Onobrakpor, Huiming Yang, Jia Hu

ABSTRACT_ID#2346

ABSTRACT

Background: In 2020, the World Health Organization launched a global plan to eliminate cervical cancer by the end of the century. In alignment, Canada aims to achieve this by 2040 through improved access to cervical cancer screening, including HPV self-screening, an approach particularly beneficial for underscreened populations such as immigrants and refugees (i.e., newcomers). This project aimed to explore effective strategies to engage newcomer communities in HPV self-screening across Alberta. **Methods:** Using a community-based health equity approach, we trained 10 Community Health Ambassadors (CHAs) across Alberta in fall 2024. CHAs are trusted community members who support health education and health system navigation in first languages. In January 2025, CHAs were deployed in urban and rural areas to recruit individuals who are: female or a person with a cervix, aged 25–69, unscreened for cervical cancer in the past five years, and are newcomers to Canada within the past 10 years. **Results:** Since deployment, CHAs have engaged newcomer communities through women's health events, public libraries, faith-based organizations, community associations, English language classes, and other resettlement programs. To date, they have reached over 2,000 newcomers, identified approximately 430 eligible participants, and collected around 347 HPV self-screening samples. The CHAs will continue their efforts in the community until August 2025. **Conclusion:** This project demonstrates the importance of culturally relevant, community-led approaches to increase cervical cancer screening among newcomers and potentially other underscreened communities. Findings will inform provincial strategies and contribute to Canada's 2040 goal of eliminating cervical cancer.

LAY SUMMARY

Trusted community members can successfully support culturally safe cervical cancer screening. Through this project, we trained 10 Community Health Ambassadors, newcomers fluent in their community's first languages, to educate peers and distribute HPV self-screening kits. This approach reached over 2,000 individuals and supported nearly 350 newcomers in completing self-collected HPV tests. HPV self-screening is a promising tool to reduce barriers and advance equity in cancer screening. By offering an accessible alternative to clinic-based screening, HPV self-screening helps address cultural, linguistic, and systemic barriers faced by newcomers to Canada. These findings can inform provincial strategies to meet Canada's goal of eliminating cervical cancer by 2040.

Lightning: B3-Prevention and Screening

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 5/7/9

Values shaping risk-stratified breast cancer screening participation among South Asian people in Montreal and Toronto: A constructivist grounded theory study - Poster #680

Presenter: Manisha Pahwa, McGill University

Other Authors: Ananya Chandra, Ipshita Prakash, Simerpreet Sandhanwalia, Tajinder Kaura, Ryoa Chung, Ananya Banerjee, Elizabeth Anne Kinsella, Matthew Hunt

ABSTRACT_ID#2449

ABSTRACT

Objective: Breast cancer screening is evolving from an age- to broader risk-based paradigm (risk stratification). Risk stratification may widen screening-related breast cancer disparities if not designed to be inclusive of diverse values. This research aimed to explore values shaping risk-stratified breast cancer screening participation among people of South Asian ancestry, whose values are unknown and who have fewer screen-detected breast cancers than people of European ancestry in Canada. **Methods:** A constructivist grounded theory study was conducted in Montreal and Toronto among South Asian people aged 30-74 years who could participate in English, French, Punjabi, or Hindi. Maximum variation and theoretical sampling were used for recruitment via hospital and community-based health clinics, places of worship, social media, and researchers' networks. A semi-structured guide was used for one-on-one interviews eliciting values shaping participation in breast cancer risk assessment and screening. Sampling and recruitment were recursive with data collection and constant comparative analysis until theoretical saturation was achieved. **Results:** Among sixteen university-educated participants (mean age 45.5 years), the value of self-determining quality of life guided decisions about whether, when, and how to participate in risk-stratified breast cancer screening. Receiving risk stratification information and psychosocial support from trusted health care providers and peers were values that fostered participation. Accessible, effective, and efficient screening were values believed to enhance quality of life and encouraged participation. Participation was mitigated by distrust of artificial intelligence in screening. **Discussion:** Trustworthy risk-stratified screening that promotes self-determined quality of life may support participation among South Asian populations in Canada.

LAY SUMMARY

Breast cancer screening based on risk rather than age alone may make participation more challenging for groups who already have low participation, in part because of the processes and technologies used to estimate risk. One of these groups is people of South Asian ancestry, who have fewer breast cancers found by screening compared to people of European ancestry in Canada. This research aimed to understand the values of South Asian people that shape their risk-based breast cancer screening participation decisions. This research used a type of qualitative research approach that develops theory about social processes to recruit and interview South Asian people living in Montreal and Toronto. Based on 16 university-educated women with an average age of 45.5 years, this study found that the value of authoring one's own quality of life guided participation decision-making. Specific values believed to enhance quality of life and encourage participation were psychosocial support and trust and accessible, effective, and efficient screening. Participation was deterred by distrust of artificial intelligence in screening. The findings of this research suggested that trustworthy risk-based breast cancer screening that promotes what individuals define as quality of life for themselves may support participation among South Asian populations in Canada.

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

Aberrant Inflammatory Response Associated with Chemotherapy-Resistance in T-Cell Acute Lymphoblastic Leukemia - Poster #566

Presenter: Ovin Amarasinghe, CancerCare Manitoba

Other Authors: Jesslyn Saw, Jacqueline A Boyle, Shokoufeh Abdollahi, David J Curtis, Cedric S Tremblay

ABSTRACT_ID#2326

ABSTRACT

Introduction: T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive subtype of leukemia, which is the most common childhood cancer. Past research comparing bone marrow samples from diagnosis and after treatment identified a population of therapy-resistant Relapse Inducing Cells (RICs) that drive T-ALL relapse. Understanding mechanisms that enable RICs to withstand chemotherapy will inform new and better treatments for T-ALL. **Methods:** More than 40% of T-ALL aberrantly express the oncogenic factor LMO2. The Lmo2Tg transgenic mouse model enables to study RICs, which emerge from the DN3 T-cell progenitor population during T-ALL development. To assess the effect of chemotherapy on RICs, we performed transplantation assays, RNA-seq and ATAC-seq following Chemotherapy (VXL) treatment or Vehicle. **Results:** Our preliminary data show that RICs found in preleukemic Lmo2Tg DN3 cells are enriched following VXL treatment. Transcriptomics data from Lmo2Tg DN3 cells showed that gene expression changes induced by VXL were transient. Pathway analysis revealed an aberrant Interferon (IFN)-gamma signature in Lmo2Tg DN3 cells at 24 hours after VXL treatment. ATAC-seq revealed increased chromatin accessibility to IFN Regulatory Factors (IRF) in therapy-resistant cells. To determine whether aberrant IFN-mediated signaling occurs early after induction chemotherapy, we assessed changes in Lmo2Tg DN3 cells at 24 hours after a single dose of VXL treatment. **Conclusion:** Changes induced by chemotherapy are transient, with VXL treatment selecting for chemoresistant RICs. Transcriptomic analysis of preleukemic Lmo2Tg DN3 cells revealed chemoresistance is associated with aberrant IFN-mediated signaling, suggesting IFN might promote chemotherapy-resistance in T-ALL.

LAY SUMMARY

Acute lymphoblastic leukemia (ALL) is the most common cancer in children, with the T-cell subtype (T-ALL) being particularly aggressive. Patients with T-ALL are treated with chemotherapy, that is quite toxic and induces lifelong side effects. Although most patients respond to treatment, some patients develop resistance to chemotherapy and T-ALL comes back within 2 years after diagnosis. My research project aims to understand why some T-ALL cases resist chemotherapy. Our discoveries will help to develop new ways to treat T-ALL for improving survival and quality of life of patients.

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

Identification of Gemcitabine-Resistant Populations Using scRNAseq in TNBC Patient-Derived Xenografts -

Poster #224

Presenter: Sandrine Busque, McGill University

Other Authors: Hellen Kuasne, Hamed Najafabadi, Constanza Martinez, Paul Savage, Anne-Marie Fortier, Anie Monast, Sandrine Omeroglu, Jamil Asselah, Nathaniel Bouganin, Sarkis Meterissian, Ariel Madrigal, Morag Park

ABSTRACT_ID#2452

ABSTRACT

The standard of care for Triple-negative breast cancer (TNBC) remains neoadjuvant chemotherapy in combination with surgery, radiation, and immunotherapy. While chemotherapy is effective in some patients, up to 50% of patients are intrinsically unresponsive or acquire resistance. These patients exhibit the highest rates of recurrence and distant metastasis. Hence, addressing chemotherapy resistance in metastatic TNBC, is a pressing clinical challenge. We hypothesized that establishing and analyzing models of disease progression of mTNBC would allow us to gain mechanistic insight into secondary resistance and identify potential markers and new therapeutic targets. We generated treatment progression models from a primary TNBC patient-derived xenograft (PDX) and its patient-matched lung metastasis PDX. We treated the metastasis PDX with Gemcitabine, mirroring the patient's treatment, and obtained residual disease that relapsed and, through treatment cycles, developed resistance. We performed single-cell RNA sequencing of these models and demonstrated that resistant cells clustered distinctly from untreated and rebound models, with residual disease split between these populations. We identified markers of a subpopulation in the untreated metastasis, which is maintained at the residual disease stage and is enriched in the resistant tumour, including WFDC2, a small secreted protein used as a biomarker in ovarian cancer. We validated these findings in vivo in tumour lysates and tumour sections at each step of treatment progression. Our current work focuses on characterizing the role of WFDC2 in resistance to gemcitabine in PDX-derived cell lines and in vivo.

LAY SUMMARY

Patient-derived xenograft treatment progression models allows us to model residual disease, relapse and resistance in vivo
Single-cell RNA sequencing of these models identifies markers of gemcitabine resistant populations

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

Improving efficacy of PARP inhibition by targeting ribosome biogenesis in triple-negative breast cancer -

Poster #470

Presenter: Mallory I. Frederick, Centre de recherche du Centre hospitalier de l'Université de Montréal (CRCHUM)

Other Authors: Nicolas Pouderous, Kenan Aloss, Shufeng Zhou, Gerardo Ferbeyre, Saima Hassan

ABSTRACT_ID#2338

ABSTRACT

Breast cancer is the most common cancer in women and a significant contributor to cancer-related mortality. Poly (ADP-Ribose) Polymerase inhibitors (PARPi), such as olaparib, target PARP-1, demonstrating success in patients with DNA repair gene mutations due to synergistically lethal effects. However, PARP-1 activity is not limited to DNA repair. Approximately 40% of PARP-1 molecules reside in the nucleolus, and PARP-1 is involved in regulation of Pol I transcription and shuttling of proteins between the nucleolus and nucleus. Thus, targeting PARP-1 also impacts ribosome biogenesis. Ribosomal proteins are enriched in triple-negative breast cancer (TNBC), an aggressive subtype of breast cancer. We measured sensitivity to a PARPi, olaparib, and two Pol I inhibitors, CX-5461 and BMH-21, in TNBC cell lines. The combination of olaparib with either Pol I inhibitor was synergistic in three PARPi-resistant cell lines, BT549, Hs578T, and MDAMB231. Using immunofluorescence, we showed that in MDAMB231 cells, the combination approach increased induction of DNA damage, indicated by increased percentage of g-H2AX-positive cells, along with nucleolar stress, shown by increased RSL1D1 and translocation of nucleophosmin from the nucleolus to the nucleoplasm. Similarly, the combination increased senescence, demonstrated by increased SA-B-gal staining. Finally, in a recovery assay, we found significant reduction of growth in cells treated with the combination of olaparib and CX-5461, up to 20 days after drug removal. Together, these data show the potential for targeting ribosome biogenesis in combination with PARPi in TNBC.

LAY SUMMARY

Triple-negative breast cancer (TNBC) is an aggressive form of breast cancer. PARP inhibitors are used in TNBC to target the protein PARP-1, which acts in the part of the nucleus where ribosomes are assembled. Combining ribosome biogenesis inhibitors with PARP inhibitors increased the ability of PARP inhibitors to kill TNBC cells by causing DNA damage. The combination was also linked to senescence, a phenotype in which cells stop replicating to avoid specific types of stress. TNBC pre-treated with the combination of PARP inhibitors and ribosome biogenesis inhibitors were unable to recover from the treatment in a 20-day recovery assay.

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

High Resolution Spatial Analyses Identifies Immunotherapy Resistance Mechanisms to CAR T-Cell Therapy in ASPS - Poster #48

Presenter: Gurveer Gill, University of Calgary

Other Authors: Varsha Thoppey Manoharan, Hongrui Lui, Yeon Suh, Bo-Young Ahn, Laura Mah, Michael Monument, Ramy Saleh, Jan-Willem Henning, Mona Shafey, Jennifer Chan, Franz J. Zemp, Douglas J. Mahoney, Sorana Morrissy

ABSTRACT_ID#2765

ABSTRACT

Alveolar Soft Part Sarcoma (ASPS) is an ultra-rare, fusion-driven sarcoma affecting adolescents and young adults. Emerging evidence suggests that ASPS is responsive to immunotherapy, and immune checkpoint inhibitors are now approved in Canada for use in these patients. To advance precision immunotherapy in this disease, our team developed a Chimeric Antigen Receptor (CAR) T cell therapy targeting Glycoprotein-NMB (GCAR1) and tested it in a stage IV ASPS patient. At a GCAR1 dose of 1e6 CAR T-cells/kg, we observed a profound reduction in the size and abundance of metastatic lung lesions in the absence of CRS/ICANs, with the patient achieving RECIST1.1 stable disease for over 6 months. To deeply explore the tumour microenvironment (TME) of ASPS and the spatial variables influencing immunotherapy efficacy in the GCAR1 clinical setting, we used the Visium HD technology to generate transcriptome-wide profiling data at 2um resolution. We profiled three clinical samples from the GCAR1-treated patient, including a primary tumour resection and two biopsy cores taken on-trial at day 35 from a non-responding metastatic lesion. Unsupervised deconvolution (cNMF) identified gene expression programs representing cell types and states of the immune, tumour, and stromal compartments. We found abundant cytotoxic T-cells in the treatment-refractory metastatic lesion, significant co-localization with tumor cells, and strong upregulation of the PD1-PDL1 signalling axis driven by tumor cells. Our results demonstrate that metastatic ASPS tumour cells upregulate PDL1 expression to inhibit T-cell function, suggesting that adjuvant checkpoint inhibitors may be effective in this setting.

LAY SUMMARY

Alveolar Soft Part Sarcoma (ASPS) is a rare and fatal cancer affecting children and young adults. One approach to treating ASPS involves immune checkpoint inhibitors (ICIs), which release natural "brakes" on T-cells – immune cells that can attack cancer – but only partially works in a third of patients. Our team developed a synthetic receptor, GCAR1, that teaches a patient's own T-cells to target and eliminate ASPS cells. We tested our therapy in a patient with metastatic ASPS and observed a reduction in metastases, with no cancer progression for over six months. We studied regions of the patient's tumor that did not respond to our treatment and found that the ASPS cells were engaging natural T-cell brakes. This finding suggests that combining GCAR1 therapy with ICIs could improve outcomes for ASPS patients.

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

mTor Inhibition in the Management of Recurrent Metastatic Solid Pseudopapillary Tumor of the Pancreas -

Poster #16

Presenter: Gabrielle Herzenberg, University of British Columbia

Other Authors: Tariq Bhat, Yaoqing Shen, Gregor Reid, Chris Maxwell, Philipp Lange, Jon Bush, Daniel Renouf, Claire Gowdy, Marco Marra, Janessa Laskin, S. Rod Rassekh, C. James Lim, Rebecca Deyell

ABSTRACT_ID#2317

ABSTRACT

Background: Solid pseudopapillary epithelial neoplasm (SPEN) of the pancreas is rare, has low malignant potential, and predominantly affects young females. While complete surgical resection typically confers an excellent prognosis, systemic therapy for unresectable, recurrent or metastatic disease remains undefined. Case Presentation: A 10y previously-well female presented with a splenic mass and was initially treated with distal pancreatectomy and splenectomy, with histologic confirmation of pancreatic SPEN. She later developed metastatic peritoneal recurrence and underwent cytoreductive surgery, with concurrent whole genome and transcriptome sequencing of tumour and matched germline in a precision medicine study. In addition to a CTNNB1 mutation, her tumour showed outlier high expression of genes of the PI3K/mTOR pathway. In the setting of residual disease, a trial of mTOR inhibitor everolimus was initiated and radiographic improvement was achieved. Concurrently, a personalized chicken chorioallantoic membrane (CAM) xenograft model of the patient's tumour demonstrated response to mTOR inhibitor therapy. The patient discontinued everolimus after 14 months due to fatigue, with disease progression 6 months later. Everolimus was re-initiated, with subsequent radiographic benefit. Conclusion: This case illustrates the potential of precision genomic medicine to identify targetable pathways using transcriptomic data in rare tumors such as pancreatic SPEN. Outlier PI3K/mTOR pathway expression guided the selection of mTOR inhibition as a therapeutic strategy, further supported by a personalized xenograft model. The patient's clinical response to everolimus, aligned with genomic and functional data, underscores the potential value of integrating molecular profiling with patient-derived in vivo models to guide treatment in the absence of established therapies.

LAY SUMMARY

Rare pancreatic tumors like SPEN can come back or spread even after surgery, and there are currently no standard systemic treatments for unresectable SPEN. In this case, we explored a new treatment approach using a medication called everolimus that targets a specific cell growth pathway, the mTOR pathway. Genetic testing of the tumor revealed unusually high activity in the mTOR pathway, a signal that tells cells to grow and divide. Based on this, the patient was treated with everolimus, a drug that blocks this pathway, and her tumor responded well. When the patient went off everolimus due to fatigue, her disease progressed. Then, after going back on everolimus, her disease re-stabilized. We also tested the drug on a personalized tumor model grown in chicken eggs (called a chicken chorioallantoic membrane (CAM) model), and it showed a similar response to mTOR inhibitor therapy. This innovative approach helped confirm that the drug was a good match for the patient's specific tumor biology and is a useful tool to guide treatment decisions.

Lightning: B4-Treatment and Drug Resistance

Monday, November 3, 2025, 4:25:00 PM - Hyatt Imperial 8

Targeting the translational machinery to overcome kinase inhibitor resistance in melanoma - Poster #414

Presenter: Anavasadat Sadr Hashemi Nejad, Université de Montréal

Other Authors: Alejandro Schcolnik-Cabrera, Meriem Takdenti, Zaynab Nouhi, Martin Roffe, Tommy Alain, Laura Hulea

ABSTRACT_ID#2383

ABSTRACT

Cancer cells rewire their mRNA translation programs and their metabolism to support growth and resistance to anti-cancer therapies like oncogenic kinase inhibitors (KIs). Our previous studies delineated translational programs that induce metabolic plasticity, which underpinned adaptation to KIs. Importantly, cancer-specific perturbations in the translational apparatus provide a therapeutic window to selectively target the translational programs of malignant cells while sparing normal tissues. The eukaryotic translation initiator factor 4F (eIF4F) complex is a key mediator facilitating translation, leading to proliferation and survival. Therefore, we hypothesize that targeting the translational machinery via inhibition of eIF4A, a subunit of eIF4F, using an eIF4A inhibitor (eIF4Ai), attenuates resistance to oncogenic KIs by preventing the metabolic reprogramming that supports cancer cell adaptation to therapies. This has been explored in our lab with in vitro and in vivo studies in BRAF inhibitor (BRAFi)-resistant melanoma models. About 50% of malignant melanoma (MM) harbor BRAF mutations, with many developing resistance to BRAFis. We found that eIF4Ais block cell proliferation, induce apoptosis, reduce mRNA translation levels, and strongly affect metabolic processes. This is paralleled by downregulation of target proteins involved in proliferation, survival and metabolic enzymes. To characterize mRNA translational modulation upon eIF4Ai, we performed polysome profiling and subsequent RNA sequencing. Furthermore, to confirm our in vitro findings, the efficacy of eIF4Ai alone and/or plus BRAFi on tumor growth is determined in vivo using cell line-derived xenografts. We anticipate that combined treatment with eIF4Ai and KIs will establish the efficacy of translational inhibitors in overcoming KI resistance in MM cells.

LAY SUMMARY

Cancer cells change how they make proteins and process nutrients to survive treatments like kinase inhibitors; by targeting key mediators of protein-making pathways, we might be able to stop this resistance. Blocking a key subunit of a specific mediator involved in protein synthesis (eIF4A) slows down melanoma cell growth, triggers cell death, and disrupts how these cells manage energy especially in cases resistant to current therapies. Combining eIF4A inhibitors with current targeted therapies (BRAF inhibitors) showed promising results in our laboratory and animal studies, suggesting this approach could help overcome drug resistance in melanoma.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Gut microbiome influences the antitumor response to BCG immunotherapy in bladder cancer - Poster #408

Presenter: Jalal Laaraj, Centre de recherche du Centre hospitalier universitaire de Québec-Université Laval

Other Authors: Gabriel Lachance, Prisca Nadège Koné, Roxane Tourigny, Typhaine Gris, Paul Toren, Alain Bergeron, Sandra Isabel, Yves Fradet, Karine Robitaille, Vincent Fradet

ABSTRACT_ID#2685

ABSTRACT

Introduction: Bacillus Calmette-Guérin (BCG) intravesical immunotherapy is the standard treatment for non-muscle-invasive bladder cancer (NMIBC). Despite its efficacy, still 40% of patients experience recurrence. Recent evidence suggests that the gut microbiome influences clinical efficacy of systemic immunotherapies. We evaluated the impact of gut microbiota on the antitumor activity of BCG in NMIBC patients and mouse models. Methods and Results: We prospectively collected fecal samples from 58 NMIBC patients before initiating BCG therapy and performed both, shotgun metagenomic and 16S rRNA sequencing to profile the gut microbiota. Differential analysis identified three bacterial species enriched in responders (R, n=42) compared to non-responders (NR, n=16). To evaluate the causal relationship between the gut microbiome and response to BCG, we conducted fecal microbiota transplantation (FMT) from R and NR patients into antibiotic pre-treated MBT-2 tumor-bearing mice, followed by weekly intratumoral BCG. FMT from NR significantly impaired BCG efficacy compared to FMT from R. Flow cytometry and bulk RNA sequencing analysis showed that R FMT was associated with potent antitumor immunity and reduced immunosuppressive populations in tumors compared to NR FMT. Oral supplementation of NR avatar mice with the response-associated bacterial species completely restored BCG efficacy, an effect that was mediated by T cell antitumor immunity and reduced myeloid immunosuppressive populations in both tumors and spleens, supporting an immune-potentiating effect. Conclusions: Our findings demonstrate that the gut microbiome impacts BCG immunotherapy response in NMIBC patients. This study paves the way for developing microbiome-targeted strategies to enhance BCG treatment efficacy in bladder cancer.

LAY SUMMARY

The gut microbiome influences bladder cancer treatment: We discovered that the bacteria living in our intestines can influence how well patients respond to BCG immunotherapy, the standard treatment for early-stage bladder cancer. Patients who responded well to BCG had certain beneficial bacteria in their gut. When we transferred fecal samples from these responder patients into mice, the treatment was more effective compared to mice that received samples from non-responders, who showed poorer outcomes. Targeting the gut microbiome can improve BCG treatment efficacy: When we supplemented non-responder avatar mice with the beneficial bacteria found in responders, their response to BCG improved significantly. This suggests that modulating the gut microbiome using live biotherapeutic probiotics could enhance the efficacy of bladder cancer treatment.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Precise tuning of CAR expression mitigates exhaustion and hyperinflammation in CAR T cell therapy - Poster #372

Presenter: Paul Jerard Layug, Paul Albrechtsen Research Institute, CancerCare Manitoba

Other Authors: Andrew Cuddihy, Huda Alfarekh, Katharina Haigh, Jody Haigh, Yale Michaels

ABSTRACT_ID#2400

ABSTRACT

Chimeric Antigen Receptor (CAR) T therapy has revolutionized treatment for B-cell malignancies. CARs are synthetic proteins that redirect T cell specificity to eliminate cancer cells that express a specific antigen, such as CD19. Despite remarkable clinical success, suboptimal durability and lethal side effects, such as cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS), limit their broader use. One major contributor to these limitations is the supraphysiological level of CAR expression upon transduction, leading to T-cell overactivation, driving rapid dysfunction and hyperinflammation. We set out to identify a “Goldilocks” level of CAR expression where T-cell exhaustion, CRS and ICANS are mitigated, while maintaining effective tumour killing. To fine-tune CD19-CAR expression, we utilized our previously established microRNA silencing-mediated fine-tuners (miSFIT) technology. miSFITs are engineered target sites for endogenous microRNAs, enabling precise control over gene expression. We assessed the effect of CAR levels on T-cell cytotoxicity, exhaustion and inflammation in vitro. Our co-culture assays demonstrated that reducing CAR expression resulted in a decrease in co-expression of exhaustion markers (PD-1 and LAG3), and dampened secretion of pro-inflammatory cytokines (IFN- γ , TNF- α , GM-CSF), without compromising tumour clearance across multiple re-challenges. In vivo, T cells engineered with a miSFIT to express intermediate CAR levels exhibited the best tumour control and significantly extended survival in a leukemic xenograft mouse model. Collectively, these findings demonstrate that CAR expression is a critical determinant of T cell function and that precisely tuning CAR levels with miSFITs could enable the production of safer and more durable CAR T therapies.

LAY SUMMARY

Too much is never good: CAR T cell products are made with too much CAR levels, making them hyperactive. This, in turn, leads to harmful side effects such as cytokine storm, and causes them to crash and deteriorate before finishing the job. We developed a solution, called “miSFIT”: This allows us to control the CAR levels, like a dimmer switch, to a “Goldilocks” zone. This way, CAR levels are neither too high to cause harm nor too low to allow cancer cells to escape, but just right to fight cancer effectively. “Goldilocks” CAR T cells are better and safer therapies: Our preclinical testing indicates that these CAR T cell products performed better and persisted longer, supporting their potential as a better option for cancer patients.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Clinical Bacillus Calmette-Guérin Strains Induce Distinct Trained Immunity Profiles - Poster #358

Presenter: Richard Nauman, Queen's University

Other Authors: Peter Greer, Andrew Craig, D. Robert Siemens, Charles Graham

ABSTRACT_ID#3209

ABSTRACT

Bacillus Calmette-Guérin (BCG) immunotherapy remains the standard of care for higher-risk non-muscle invasive bladder cancer (NMIBC), despite an incomplete understanding of its immunotherapeutic mechanism. Trained immunity (TI), a heterologous form of memory acquired by innate immune cells, is thought to be involved. We have previously reported that TI acquisition in peripheral immune compartments is associated with recurrence-free survival post-BCG therapy. Studies on the effects of BCG in the protection against tuberculosis have revealed that different strains of BCG have distinct immunogenic properties; however, a thorough comparison of the TI-inducing properties of different strains of BCG has not been performed. We assessed TI acquisition and secondary responses following administration of BCG-Russia or BCG-TICE. Macrophages from mice treated with BCG-TICE exhibited significantly higher production of several proinflammatory cytokines in response to heterologous stimuli compared to BCG-Russia or vehicle control. BCG treatment led to expansion of hematopoietic stem and progenitor cell (HSPC) populations, however the effect appeared to be independent of strain. Transcriptomic analysis revealed that expression of genes related to several processes differed between the two strains, including antigen presentation, ECM remodeling, and T cell activation. TI is emerging as a crucial component of the mode of action for BCG therapy in NMIBC. Shortages of BCG-TICE have historically posed a clinical challenge, with BCG-Russia often used as a substitute in Canada. Understanding the differential immune responses elicited by these strains informs clinicians about the suitability of BCG-Russia as an alternative to BCG-TICE.

LAY SUMMARY

BCG-TICE elicits stronger innate immune (bone marrow) reprogramming compared to a different strain that is often used as a substitute for TICE. Macrophages, in particular, exhibited distinct cytokine secretion and transcriptomic profiles dependent on BCG strain. This research has direct implications for clinical decision making, suggesting that the immunostimulatory effect of BCG is strain-dependent and certain strains may be inferior to others.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Microbial Metabolite Modulation of CAR-T Therapy - Poster #406

Presenter: Amy Shupe, University of Calgary

Other Authors: Larisa Kovtonyuk, Sacha Benaoudia, Kyle Potts, Doug Mahoney, Kathy D. McCoy

ABSTRACT_ID#2806

ABSTRACT

While chimeric antigen receptor (CAR) T therapy has revolutionized the treatment of hematological malignancies, many patients still do not respond. Recent research has pointed to the microbiome, the collection of microbes that live on and in our bodies, as a key player in the response to immunotherapies. However, the underlying mechanisms by which bacteria influence the responses to cancer and cancer immunotherapies are still largely unknown. Our lab has previously demonstrated that the metabolite inosine, produced by *Bifidobacterium pseudolongum* and other bacteria, improved the efficacy of another form of immunotherapy called immune checkpoint blockade therapy (ICBT), in a context dependent manner. We hypothesized that inosine would also increase the efficacy of CAR-T therapy. Pre-treatment of human CAR-T cells with inosine was found to improve the killing of a Nalm6 human lymphoma cancer cell line in vitro. CAR-T cells treated with inosine showed higher levels of the effector molecules IFN γ , TNF, Granzyme B and Ki67, than untreated CAR-T cells measured via flow cytometry. In addition, CAR-T cells incubated with inosine in vitro also showed superior ability in vivo killing capacity when transferred to germ free mice bearing Nalm6 as compared to untreated CAR-T cells. Overall, these findings could pave the way for the use of microbial products that can increase the efficacy of CAR-T therapy.

LAY SUMMARY

Good gut bacteria may help cancer therapies work better. We studied a molecule, produced by gut bacteria, called inosine, and found it can make a certain form of cancer immunotherapy (chimeric antigen receptor or CAR T cells) more effective. Inosine boosts the ability of CAR-T cells to kill cancer cells. We tested inosine treated CAR-T cells and found that they were better at killing cancer cells and showed more signs of effector function. These findings could pave the way for the use of bacterial products (like inosine) to increase the efficacy of cancer therapy.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Microbiome-derived hydroxyphenyl propanoates enhance antitumour immunity by potentiating gasdermin D activity in tumour-associated myeloid cells - Poster #384

Presenter: Saif Sikdar, University of Calgary

Other Authors: Dae-Sun Kim, Tie Wang, Sacha Benaoudia, Jahanara Rajwani, Kristofor Ellestad, Pauline Douglas, Ruzena Filandrova, Ted Verhey, Katrina Ellestad, Marija Drikic, Darren Derksen, Ian Lewis, Marco Gallo, Sorana Morrissy, David Schriemer, Kathy McCoy, Franz Zem

ABSTRACT_ID#3176

ABSTRACT

The microbiome plays a critical role in immune health and response to immune-stimulating treatments, including cancer immunotherapy. However, the molecular mechanisms by which commensal microbes influence cancer immunology remain poorly understood. Here, we report a class of microbiome-derived metabolites called hydroxyphenyl propanoates (HPP) that enhance tumour immune surveillance and response to immune checkpoint blockade (ICB) therapy in mice. HPPs function as potentiators of innate immune signalling in tumour-associated myeloid cells by promoting cleavage of the pore-forming protein gasdermin D (GSDMD), a critical effector of inflammasome function. Increased secretion of proinflammatory cytokines, such as IL-1 family members, in turn heightens autocrine and paracrine inflammatory signaling in tumour-infiltrating leukocytes. HPP supplementation increases anticancer T cell function, improves disease control and extends overall survival (OS) in tumour-bearing wildtype but not *Gsdmd* knockout mice. In humans, GSDMD activation is associated with a favourable response to PD-1 blockade therapy in patients with advanced stage melanoma. Our study uncovers a molecular mechanism of regulation over host antitumour immunity that is modifiable and can be harnessed for improved cancer immunotherapy.

LAY SUMMARY

Specific gut microbes can help fight cancer: A particular composition of the gut microbiome produces a class of compounds called HPPs, which enhance the immune system's ability to eliminate tumour cells. **Boosting immune activity in tumours:** HPPs uniquely modulate the immune protein GSDMD enabling immune cells to release bioactive cytokines without undergoing cell death. This process activates other cancer-fighting immune cells within the tumour microenvironment. **Potential to improve cancer treatment:** In both mouse models and human studies, increased activity of this immune pathway correlates with improved outcomes during PD-1/PD-L1 immunotherapy. Notably, HPPs remain effective in hosts with diverse microbiomes and can overcome the negative impact of antibiotics on immunotherapy, highlighting their potential for clinical application in cancer treatment.

Lightning: B5-Enhancing Immunotherapy

Monday, November 3, 2025, 4:25:00 PM - Hyatt Doll/Herald

Investigating the Use of PCSK9 Inhibitor Alirocumab for use in Combination Therapy with PD-1 Inhibitors to Treat Melanoma - Poster #396

Presenter: Madison Wickenberg, University of Alberta

Other Authors: Suha Jarad, Dawei Zhang, John Walker, Paul LaPointe

ABSTRACT_ID#2347

ABSTRACT

Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a serine protease responsible for cleavage and endocytosis of the low-density lipoprotein receptor (LDLR). Therefore, PCSK9 inhibitors have been developed for use in patients with high cholesterol. Recent studies have suggested that PCSK9 may also mediate internalization of the major histocompatibility complex 1 (MHC1). Peptides presented in MHC1 provide information to immune cells regarding whether the presenting cell is “foreign” or “self”. MHC1 presentation is highly relevant in cancer cells, as the presentation of novel/mutated peptides allows for T-cell recognition and subsequent destruction of the cancer cells. Inhibiting PCSK9 activity will prevent MHC1 internalization and surface downregulation, leading to increased immune detection of tumours. As a result, we are investigating the use of PCSK9 monoclonal antibody, alirocumab, for use as a cancer immunotherapy. My recent work demonstrates that PCSK9 regulates internalization of MHC1 in melanoma cells, and that this effect is not seen for other immunological receptors. Additionally, I demonstrated that the total protein levels of MHC1 are mediated by PCSK9, corresponding with the concept that PCSK9 causes MHC1 trafficking to lysosomes in a similar manner to its regulation of the LDLR. Preliminary findings also suggest that melanoma cells express an intrinsic level of PCSK9, which may be protective by downregulating MHC1. This emphasizes the need for further research into PCSK9 in cancer, and the potential for alirocumab as an immunotherapy, specifically in combination with existing checkpoint inhibitors.

LAY SUMMARY

Alirocumab, an antibody originally studied for use to treat high cholesterol (hypercholesterimia), has also been shown to decrease tumors in patients. This antibody binds to proprotein convertase subtilisin/kexin type 9 (PCSK9), the protein responsible for downregulating levels of the low-density lipoprotein receptor (LDLR). Recent studies have now shown that PCSK9 also downregulates expression of major histocompatibility complex 1 (MHC1). MHC1 is a receptor important for immune cell recognition of cancer cells. Since alirocumab prevents MHC1 reduction on tumor cells, immune cells can more effectively recognize and target the cancer cells for destruction. The current standard of care treatment for patients with melanoma is immunotherapy, a form of cancer therapy that focuses on boosting immune activation through modulation of various pathways. However, not all patients respond to the most commonly used immunotherapy treatments, so alternatives and combination of multiple treatments are being investigated. Therefore, better understanding the role of PCSK9, and ultimately alirocumab, in melanoma cells is an important area of research as a potential therapeutic option.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

Subsequent primary neoplasm risk among survivors of adolescent and young adult cancer: a population-based study from Alberta, Canada - Poster #8

Presenter: Arafat Ul Alam, Alberta Health Services

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#2553

ABSTRACT

Background: Survivors of adolescent and young adult (AYA) cancers are at an increased risk for subsequent primary neoplasms (SPN), but studies using Canadian data are limited. Here, we quantify the risk of SPNs among AYA cancer survivors in Alberta, Canada. Methods: The Alberta AYA Cancer Survivor Study is a retrospective, population-based cohort of 15-39 year-olds diagnosed with a first neoplasm from 1983–2017. Risk of SPN was assessed overall and among 5-year survivors. Standardized incidence ratios (SIRs), absolute excess risks (AERs; per 10000 person-years), and 30-year cumulative incidences were calculated. Results: Among 24,459 AYA cancer patients, 1,442 SPNs were identified, 1,129 of which occurred after 5-year survival. SPN risk was comparable when assessed overall and among 5-year survivors, with risk being 2-times higher than that expected in the general population, equating to approximately 30 excess SPNs. Among 5-year survivors, the 30-year cumulative incidence of SPNs was 17.7%, with incidence highest among 5-year survivors of Hodgkin lymphoma (HL; 22.7%) and cancers of the colon (23.5%), rectum (26.9%), breast (27.3%), and oral cavity/lip/pharynx (28.9%). Among these survivors, incidence was significantly higher than expected for breast SPNs after HL or breast cancer; digestive SPNs after HL, colon, rectal or breast cancer; respiratory SPNs after HL or breast cancer; hematopoietic SPNs after HL; and head/neck SPNs after oral cavity/lip/pharyngeal cancer. Conclusion: AYA cancer survivors are at an increased risk for all types of SPNs assessed. Earlier, risk-based screening may be warranted, particularly for breast, digestive and respiratory cancers where screening infrastructure already exists.

LAY SUMMARY

People who survived cancer between ages 15 and 39 in Alberta, Canada had twice the risk of developing a new, different cancer compared to the general population. This translates to about 30 more cases of new cancers for every 10,000 survivors each year compared to the general population. Survivors of Hodgkin lymphoma, breast, colon, rectal, and oral cancers had the highest long-term risk, with nearly 1 in 3 to 1 in 4 developing a new cancer within 30 years. Because many of these subsequent cancers are in breast, digestive organs, and lungs—where screening programs already exist—earlier or more frequent screening for survivors may help catch new cancers sooner.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

Colorectal and breast cancer screening adherence among cancer survivors: A systematic review and meta-analysis - Poster #694

Presenter: Sanjeev Bista, University of Calgary

Other Authors: Hannah Harsanyi, Momtafin Khan, Rey Nambayan, Marcus Vaska, Yibing Ruan, Robert J. Hilsden, Darren R. Brenner, Dylan E. O'Sullivan

ABSTRACT_ID#2948

ABSTRACT

Background: Cancer survivors have been shown to be at an elevated risk of developing subsequent colorectal (CRC) and breast cancers (BC) compared to the cancer-free general population. Adherence to CRC and BC screening guidelines among cancer survivors is important to reduce the morbidity and mortality associated with subsequent cancers. Methods: We conducted a systematic review of the prevalence and predictors of adherence to CRC and BC screening among non-CRC and non-BC survivors. Meta-analyses were conducted for predictors that were reported in at least 3 studies. Random effects models were used to pool estimates. Results: A total of 62 studies were included in this review. The pooled prevalences of CRC and BC screening were 0.52 (95% CI: 0.45, 0.58) and 0.66 (95% CI: 0.61, 0.70), and the prevalence varied considerably by the type of first primary cancer. Significant predictors of adherence to CRC screening included having a health care provider, being insured, older age, and higher income and education, while significant predictors of adherence to BC screening included being instructed to participate in screening, a higher number of visits to healthcare facilities, having a cancer specialist as a care provider, having an earlier staged cancer, the presence of no comorbidities, white race, and younger age. Conclusion: Among cancer survivors, the overall rates of CRC and BC screening are lower than the target level for population-based screening, particularly for CRC. Factors associated with non-adherence can be used to develop programs and interventions to increase uptake and adherence to screening among cancer survivors.

LAY SUMMARY

Cancer survivors have been shown to be at an elevated risk of developing subsequent colorectal (CRC) and breast cancers (BC) compared to the cancer-free general population. Despite this elevated risk, rates of adherence to CRC and BC screening among cancer survivors is well below the target for population-based screening. Factors associated with non-adherence identified in this study can be used to develop programs and interventions to increase uptake and adherence to screening among cancer survivors.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

The Cancer Rehabilitation and Exercise – Advanced Cancer (CaRE-AC) Program: A Qualitative Exploration of Intervention Delivery Challenges, Successes, and Sustainability - Poster #746

Presenter: Kelcey Bland, University of British Columbia

Other Authors: Janna Griffioen, Grace Schouten, David Langelier, Helen McTaggart-Cowan, Sarah Neil-Sztramko, Kelly Mackenzie, Amrah Ahamed, Anthony Reiman, Jonathan Greenland, Jennifer Jones, Kristin Campbell

ABSTRACT_ID#3193

ABSTRACT

Introduction: Treatment advances have extended cancer survival, increasing the number of individuals living with advanced cancer. However, treatment can impair physical function and quality of life, making rehabilitation and exercise critical. The Cancer Rehabilitation and Exercise – Advanced Cancer (CaRE-AC) program offers exercise and education sessions tailored for advanced cancer. To foster ongoing success and scalability, barriers and facilitators to CaRE-AC delivery were explored. **Methods:** Semi-structured interviews were conducted with people with advanced cancer who participated in CaRE-AC (n=20) and healthcare providers involved in recruitment and intervention delivery (n=10). Transcripts were inductively coded using conventional content analysis and mapped onto the Consolidated Framework of Implementation Research domains (innovation, inner setting, outer setting, individuals, implementation process) to identify initial themes. **Results:** The innovation domain emphasized person-centered care through adaptability to patients' diverse needs. At the individual level, engagement was influenced by personal barriers (e.g., symptom burden), exercise self-efficacy, and positive provider experiences. The implementation process showed strengths in assessment delivery, structured education, and practitioner enthusiasm, with barriers including unclear recruitment, operational and communication logistics, and a lack of cohesion between education and exercise sessions. A supportive inner setting fostered holistic change, including trust in exercise abilities and validation of shared experiences. Outer setting barriers included limited resource awareness, technology access, and health events. Facilitators included sustained funding, long-term support, and continued connection post-intervention. **Conclusion:** Exploring patient and healthcare provider perspectives revealed factors influencing CaRE-AC implementation, from individual engagement barriers to system-level challenges. Insights generated may help refine and scale the CaRE-AC program.

LAY SUMMARY

1. Rehabilitation and exercise are important for individuals with advanced cancer: While treatment has extended cancer survival, it can also impact physical function and quality of life among people living with advanced cancer. The CaRE-AC program provides tailored exercise and education sessions to support patients in managing these challenges.
2. Understanding barriers and facilitators helps improve program delivery: Interviews with patients and healthcare providers revealed key factors influencing participation, including personal challenges like symptom burden, the importance of provider support, and logistical barriers, such as recruitment and session cohesion.
3. Insights from implementation research can enhance scalability: Identifying strengths and challenges in CaRE-AC delivery, such as sustained funding, trust-building, and continued patient engagement, may help refine the program and expand access to more individuals living with advanced cancer.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

Mixed-Methods Exploration of Stakeholder Perspectives Toward Psychedelic-Assisted Therapies for Cancer-Related Psychosocial Symptoms - Poster #712

Presenter: Haley Schuman, University of Calgary

Other Authors: Linda Carlson

ABSTRACT_ID#3262

ABSTRACT

Background: Psychedelic-assisted therapy (PAT) is a promising intervention for cancer-related psychosocial distress, but its integration into care remains limited by implementation and policy gaps. Methods: A national cross-sectional survey (N = 742) and two qualitative interview studies were conducted to examine stakeholder perspectives on PAT. Survey respondents included PLWC (n = 519), healthcare providers (HCPs; n = 187), and policymakers (PMs; n = 36). The survey assessed attitudes (APQ), perceived knowledge, safety and effectiveness beliefs, and interest in training/access. Between-group comparisons were conducted using non-parametric and regression-based analyses. Semi-structured interviews with PLWC and HCPs further explored implementation barriers, motivations, and ethical considerations, analyzed using reflexive thematic analysis grounded in the Theory of Planned Behaviour. Results: PLWC endorsed the most favorable attitudes toward PAT, significantly higher than HCPs and PMs ($p < .001$), despite reporting the lowest perceived knowledge. PMs expressed the greatest safety concerns and preferred restricting PAT to advanced-stage cases. Psilocybin was perceived as the most effective agent across groups. Key implementation barriers varied: PLWC prioritized financial and provider access, while HCPs and PMs emphasized training, standardization, and evidence. Regression analysis showed that stakeholder group and prior psychedelic experience were positively associated with favorable attitudes, whereas higher self-rated knowledge predicted lower attitude scores ($p < .001$). Interviews echoed these patterns and highlighted ethical tensions around access and regulation. Conclusion: Despite promising receptivity, substantial implementation barriers persist. Addressing safety, training, and policy concerns—while centering stakeholder-informed solutions—is essential for ethical and equitable PAT integration into oncology care.

LAY SUMMARY

1. PLWC are open to psychedelic-assisted therapy (PAT) as a way to address emotional, spiritual, and existential suffering, particularly after treatment or in advanced illness. In our national survey, PLWC showed the most positive attitudes toward PAT, despite reporting the lowest perceived knowledge. Interestingly, those who rated themselves as more knowledgeable were sometimes less supportive of PAT, highlighting the need for accurate, balanced education. Many PLWC (92.8%) supported regional access, viewing PAT as a meaningful tool for coping with fear, loss of meaning, and suffering that conventional care often overlooks.
2. HCPs and policymakers are more cautious, expressing concerns about safety, training, legal uncertainty, and institutional readiness. Providers highlighted the need for clearer guidelines and more robust clinical evidence, especially as patient interest increases. Attitudes toward PAT differed across groups—patients were the most supportive, while providers and decision-makers were more hesitant due to systemic and professional constraints.
3. Interviews revealed complex, substance-specific views. Ketamine was valued for its rapid effects but critiqued for commercialization and dependency risks. Psilocybin sparked polarized views—some saw it as promising for existential distress, while others worried about destabilization. MDMA was viewed as promising for trauma but hindered by concerns about serotonin depletion and regulatory hurdles.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

Living with and beyond advanced cancer: Understanding patient needs and experiences - Poster #768

Presenter: Sarah Scruton, Nova Scotia Health

Other Authors: Stephen Sallows, Julee Pauling, Yelena Aizenberg, Cynthia Kendell, Anthony Reiman, Amy Trottier, Alison Wallace, Robin Urquhart, John Thoms

ABSTRACT_ID#3018

ABSTRACT

Background: Significant developments in the treatment of advanced cancer have led to unprecedented improvements in survival, introducing a new population of Canadians living long term with their disease. However, most existing supports are tailored to those receiving curative intent treatment, and we do not have an understanding of the unique needs people with advanced cancer face or how best to support them. **Methods:** 25 interviews will take place with individuals diagnosed with advanced cancer to qualitatively explore their experiences, unique challenges, and their thoughts on developing supports to meet their needs. Workshops with patients and healthcare providers will be held to co-design a resource or support to be tested in a future study. **Results:** To date, 20 interviews have been completed. Preliminary analyses have identified numerous unmet and unique needs among individuals undergoing long-term treatment. These include being unable to return to work due to lasting treatment effects resulting in financial and emotional strain, limited availability of long-term support, difficulties with meaning-making, barriers to accessing care, and concerns about future treatment options when current therapies fail. Peer support, exercise programs, and family/friend support were mentioned as facilitators to needs being met. Participants emphasized their desire for future interventions focused on navigation, resource libraries, and assistance managing day-to-day activities. **Discussion:** The findings of this study will provide the information needed to co-design interventions to better support individuals living long-term with advanced cancer. This work highlights gaps in existing services, which will be turned into meaningful supports during the upcoming co-design workshops.

Lightning: B6-Survivorship

Monday, November 3, 2025, 4:25:00 PM - Hyatt Stephen AB

Physical Activity Interventions for Pain Management of Aromatase Inhibitor Induced Musculoskeletal Syndrome: A Systematic Review - Poster #744

Presenter: Jenna Sim, University of Calgary

Other Authors: Khloe Bedore, Elaine Gentleman, Imrose Bhullar, Hannah Cripps, Julia Daun, Jonathan Low, Arjun Ghuman, David Langelier, S. Nicole Culos-Reed, Lauren Capozzi

ABSTRACT_ID#3129

ABSTRACT

Introduction: Aromatase inhibitors (AIs) are a highly effective adjuvant therapy for hormone receptor positive breast cancer. A common side effect includes AI-induced musculoskeletal syndrome (AIMSS), which includes primarily myalgias and arthralgias. Many who experience AIMSS stop or pause treatment, increasing risk of recurrence. Physical activity (PA) can help manage many cancer-related side effects, however its effect on AIMSS is less known. Thus, this review aimed to synthesize the evidence on PA interventions for AIMSS, with a primary focus on AIMSS-related pain. Methods: MEDLINE, CHAHL, CENTRAL, and Embase were systematically searched for PA interventions for AIMSS. Titles, abstracts, and full-texts were screened in duplicate. Extraction was completed independently and captured intervention and participant characteristics, AIMSS symptoms, and risk of bias. Results were analyzed via narrative synthesis and harvest plots. Results: The search identified 3148 unique articles, from which 15 reported on PA interventions. Interventions included aerobic exercise (n=4), combined aerobic and resistance exercise (n=5), yoga (n=4), and Tai Chi (n=2). Clinically or statistically significant improvements in AIMSS-related pain were reported in 73% (n=11) interventions. Nine studies measured AIMSS-related pain directly (e.g., Brief Pain Inventory, visual analog scale), while two measured AIMSS-related pain indirectly via quality-of-life measures. Conclusions: Results of this review are promising; PA may reduce AIMSS-related pain. However, the extent of the literature is still relatively limited, thus it is difficult to draw strong conclusions. Future research should consider conducting high-quality intervention studies to determine the ideal components of PA interventions (e.g., frequency, intensity, time, type) to reduce AIMSS-related pain.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Using a Serious Illness Conversation Guide to Improve the Quality of Life of Blood Cancer Patients - Poster #722

Presenter: Olivia Benguigui, Lady Davis Institute, McGill University

Other Authors: Sarit Assouline, Victoria Korsos, Chantal Cassis, Saima Ahmed

ABSTRACT_ID#3026

ABSTRACT

Background: Individuals with hematologic malignancies are more likely to die in acute care settings, including the ICU and under aggressive treatments, compared to those with solid tumours. This underscores the need for improved end-of-life (EOL) care in this population. The Serious Illness Conversation Guide (SICG) is a structured communication tool shown to reduce anxiety and depression in patients with advanced cancer^{1,2}. **Objective:** This ongoing study aims to evaluate whether implementing a serious illness conversation (SIC) with patients likely to die within 12 months from their hematologic malignancy reduces rates of death in acute care settings. Secondary objectives include assessing quality-of-life (QoL) outcomes and patients' perceived value of the SIC. **Methods:** Participants (n=34) are being recruited from the inpatient and outpatient hematology services at the Jewish General Hospital. Eligible patients are receiving active treatment and identified as at risk of dying within one year. Treating clinicians conduct an SIC with each participant. QoL is assessed before and after the SIC using the McGill Quality of Life Questionnaire³⁻⁵. Participants' charts are followed until death, and outcomes are compared to matched historical controls. Planned analyses include logistic regression for EOL outcomes (e.g., ICU death, aggressive care) and paired t-tests for pre-post QoL comparisons. **Preliminary Results:** Among the first 13 deaths, 30.7% occurred in acute care settings. Three of four acute care deaths occurred in the ICU under aggressive treatment; one occurred on a palliative ward. **Conclusion:** Preliminary findings suggest SIC may positively influence EOL care trajectories in hematologic malignancies.

LAY SUMMARY

Preliminary findings suggest that a substantial proportion of participants died in palliative settings and in alignment with their stated preferences after engaging in a timely Serious Illness Conversation (SIC) with their clinician. With full sample data and planned multivariable logistic regression analyses, we aim to demonstrate how SICs improve patient-centred care and end-of-life (EOL) outcomes in hematologic malignancies. Building on evidence from solid tumour populations, early feedback indicates that patients with hematologic malignancies and their families may also find the SIC intervention meaningful and emotionally supportive. As recruitment nears completion, we anticipate gaining further insight into the perceived benefits of SICs in this unique patient group. Beyond direct patient outcomes, this study may yield broader system-level benefits by identifying key facilitators and barriers to delivering high-quality EOL care in hematology. Findings will contribute to defining hematology-specific quality-of-life indicators and guiding improvements in how clinicians approach serious illness conversations, a topic with significant psychological and social implications for patients and their families.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Toward Standardized Clinical Assessment of CIPN in Rehabilitation: Identifying Relevant Tools for Practice - Poster #724

Presenter: Antoine Frasie, Centre de recherche du Centre hospitalier universitaire de Québec-Université Laval

Other Authors: Charles-Antoine Auger, Stephanie Bernard, Kadija Perreault, Maud Bouffard, Sarah Béland, Philippe Bérubé-Mercier, Frédérique Therrien, Philip L. Jackson, Julie Lemieux, Anne Dionne, Jennifer Gewandter, Lynn R. Gauthier

ABSTRACT_ID#3314

ABSTRACT

Background: Rehabilitation shows promise in managing chemotherapy-induced peripheral neuropathy's (CIPN) sensory and motor effects. Assessment tools' relative validity for identifying clinically relevant acute and chronic CIPN remains unclear. Aim: Identify sensory and functional patient-reported (PROs), clinician-reported (ClinROMs), and performance-based (PerfOMs) outcome measures most strongly associated with clinically relevant acute and chronic CIPN. Method: People with stage I-III breast cancer completed the FACT/GOG-NTX-4 at baseline (T0), post-taxane (T1), and three months later (T2). A clinical anchor-based Minimally Important Difference (MID) of ≥ 3.39 from T0 defined MID-CIPN at T1 and T2. PROMs (Short-Form McGill Pain Questionnaire-2 [SF-MPQ-2]; Neuropathic Pain Symptom Inventory; Brief Pain Inventory-Interference), ClinROMs (Total Neuropathy Scale-revised-Short-Form [TNSr-SF]; hand/foot-cold/hot pain threshold; cold/warm, vibration, mechanical detection threshold; Karnofsky Performance Status Scale), and PerfOMs (Short Physical Performance Battery [SPPB], Timed-Up-and-Go) were collected. Stepwise logistic regression identified T1/T2 sensory/functional measures associated with T1/T2-MID-CIPN presence. Results: 114 participants (52.30 ± 11.18 years old; 100% female; 62.3% paclitaxel, 36.8% docetaxel) were included. 64 (51%) and 49 (39%) had MID-CIPN at T1 and T2. T1-MID-CIPN was associated with T1-SF-MPQ2-Neuropathic subscale (OR=4.103; 95%CI: 1.780-9.453), T1-cold pain threshold-hand (CPT-H; OR=1.192; 95%CI: 1.025-1.386), T1-TNSr-SF (OR=1.335; 95%CI: 1.101-1.619). T2-MID-CIPN was associated with T2-TNSr-SF (OR=1.322; 95%CI: 1.084-1.611), T2-SPPB (OR=0.119; 95%CI: 0.015-0.949). Conclusion: Measures associated with acute CIPN were the SF-MPQ-2-Neuropathic PROM, and CPT-H (upper-extremity cold allodynia) and TNSr-SF (upper- and lower-extremity vibration sensation, reflexes, neuropathy/neuropathic pain) ClinROMs. Measures associated with chronic CIPN were the TNSr-SF and the SPPB (lower extremity functioning and balance) PerfOM. Future research should define clinically relevant thresholds and rehabilitation assessment guidelines.

LAY SUMMARY

Rehabilitation is a key component of supportive care to prevent and manage side effects of cancer treatments such as chemotherapy-induced peripheral neuropathy (CIPN). Many assessment tools are used in these care settings, with no consensus about which should be used. To measure clinically relevant acute CIPN, clinicians should consider using a combination of patient reported outcomes and clinician reported outcomes measuring neuropathic pain, cold pain sensitivity, and vibration sensitivity, reflexes, and neuropathy in the upper and lower extremities. To measure clinically relevant chronic CIPN, clinicians should consider using a combination of clinician reported outcomes and performance-based outcomes measuring vibration sensitivity, reflexes, and neuropathy in the upper and lower extremities, and lower-extremity balance and functioning. Future research is needed to understand clinically relevant thresholds for these tools and establish rehabilitation assessment guidelines.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Mixed Methods Evaluation of Implementation Outcomes, Barriers, and Facilitators of an Electronic Patient-Reported Outcome System for Cancer Rehabilitation - Poster #312

Presenter: Christian Lopez, Princess Margaret Cancer Centre, University Health Network

Other Authors: Sarah Neil-Sztramko, Kristin Campbell, David Langelier, Gillian Strudwick, Jackie Bender, Jonathan Greenland, Tony Reiman, Jennifer Jones

ABSTRACT_ID#2681

ABSTRACT

Background: REACH is an electronic patient-reported outcome (ePRO) system that remotely monitors patients' rehabilitation needs during and after treatment, delivering support including links to self-management education and community programs, and recommendations for further evaluation. We aimed to evaluate the implementation of REACH at four Canadian centres for four disease sites (breast, colorectal, head and neck, and lymphoma) and understand barriers and facilitators to implementation. Methods: A 16-month formative mixed methods evaluation was conducted, guided by the Implementation Outcomes Framework and the Consolidated Framework for Implementation Research. Quantitative data included system analytics and patient experience surveys. Qualitative data were collected through focus groups with patients and interviews with clinic leadership and point-of-care staff. Results: Of 722 patients registered, 610 (67% female, 44% breast, median age 57 (21–91), 45% receiving treatment) consented for research. REACH was feasible (median assessment time: 3 minutes; 73% of patients reported ease of use) and showed moderate engagement (99% of patients completed ≥ 1 assessment; 39% of all assessments completed; 68% viewed ≥ 1 recommended resource). Key themes influencing implementation were organizational capacity to prioritize new initiatives, alignment and integration with digital systems, onboarding processes ensuring timely and clear introduction of REACH within clinical workflows, and perceptions of REACH's value and usability. Conclusions: Immediate delivery of tailored resources after ePRO completion was highly valued; however, implementation was hindered by limited integration with organizational decision-making, digital systems, and patient education during clinical encounters. Embedding REACH into a comprehensive digital platform at each centre is needed to support sustained patient engagement.

LAY SUMMARY

REACH is an online tool that helps people living with and beyond cancer report their symptoms and rehabilitation needs from home. After filling out short questionnaires, patients get immediate access to a personalized resource library tailored to their needs. This library provides links to 1) education resources such as videos, websites, and reading materials; 2) programs such as online or in-person classes and workshops led by trained professionals; and 3) suggestions to follow-up with a health care provider for further evaluation or referral. Over 600 patients across four cancer centres used REACH and nearly all completed at least one questionnaire. Most found it easy to use, and many appreciated having a personalized, central place where they could find resources to help with their rehabilitation and symptom management. Successful implementation depended on how well REACH fit into each clinic's existing systems and workflows. Challenges included limited involvement and coordination with clinic leadership, organizational planning, and other patient digital tools, as well as limited time during appointments to introduce REACH to patients.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Developing an AI-Powered Assistant for Personalized Navigation in Cancer Care - Poster #58

Presenter: John-Jose Nunez, BC Cancer

Other Authors: Alan T. Bates

ABSTRACT_ID#2528

ABSTRACT

Patients with cancer often face difficulty navigating a complex system of supportive care services, including counseling, palliative care, and community support. Many are unaware of available options or unsure how to access them—leading to unmet needs, delayed interventions, and greater emotional, social, and physical burden. We are developing an AI-powered cancer care navigation assistant that builds on our previous work creating predictive models to estimate survival and psychosocial needs from oncology consultation notes. This project incorporates generative AI tooling to translate those predictions into clear, patient-friendly explanations and personalized recommendations. These are matched with a curated resource database and delivered through accessible channels such as SMS, email, web chat, or voice—helping patients identify and connect with appropriate resources. We will present an initial proof-of-concept version of the assistant, demonstrating how our predictive and generative AI models support real-time, personalized recommendation delivery. We will also share results from early testing and highlight technical design choices, such as explainability features to build patient trust and a modular system that supports future expansion. This work has been shaped by consultations with potential research users—including patients, clinicians, and system leaders—to identify where AI can most meaningfully improve the cancer care experience. The session will use interactive discussion to gather feedback on desired features, interface design, and integration into clinical workflows. By enabling timely access to supportive care, this AI navigation tool aims to improve outcomes, reduce healthcare system strain, and serve as a scalable model for AI-driven care navigation across oncology and beyond.

LAY SUMMARY

AI can help identify patient needs earlier. We've developed predictive AI models that analyze oncologists' notes to identify patients who may need extra support—including psychiatry and counselling services—early in their cancer journey. Combining Generative and Predictive AI can empower patients to navigate cancer resources. Using generative AI, we translate those predictions into easy-to-understand messages that help patients explore relevant resources—such as support groups, transportation help, or financial aid—at the moments they may find them most useful. We're building a navigation assistant to make this support more accessible. Our proof-of-concept AI assistant will deliver these personalized recommendations through familiar channels like text message, email, or chat, so patients can access support without needing to navigate a complex system on their own.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Enhancing Pediatric Radiation Therapy Through Video-Based Distraction and Empowerment - Poster #546

Presenter: Heather Roy, RadFlix

Other Authors: Natalie Logie, Tatiana Ritchie

ABSTRACT_ID#3086

ABSTRACT

Radiation therapy can be a distressing experience for pediatric patients, often leading to high levels of anxiety and, in many cases, the need for sedation or general anesthesia to complete treatment. Video distraction therapy is an innovative, patient-centered tool that can be used to transform the radiation experience by allowing children to watch their favorite movies during treatment. Video-based distraction therapy addresses the psychosocial needs of pediatric patients by reducing fear, anxiety, and the emotional burden associated with cancer care. Preliminary implementation of RadFlix as a video distraction tool has demonstrated multiple psychosocial benefits. Children report feeling more in control during treatment sessions, with many stating that time passes more quickly and the overall experience is less frightening. Importantly, some younger patients who previously required anesthesia are now able to tolerate therapy while awake, reducing both clinical risks and healthcare costs. By incorporating a familiar and enjoyable activity into the clinical environment, RadFlix supports emotional well-being and helps restore a sense of normalcy and joy during a challenging time. This approach underscores the critical role of psychosocial care in pediatric oncology, where emotional support and engagement are essential components of effective treatment. RadFlix represents a scalable, non-invasive solution that aligns with holistic care models, emphasizing the importance of treating not just the disease, but the whole child.

LAY SUMMARY

Psychosocial interventions can reduce the need for sedation: Integrating positive distraction techniques into radiation treatment can enable younger patients to complete therapy awake, decreasing reliance on anesthesia and its associated risks and costs. Emotional support improves treatment adherence and experience: By addressing fear and anxiety through engaging, child-friendly interventions, patients are more likely to tolerate and complete treatment, enhancing both clinical outcomes and emotional well-being. Holistic care enhances quality of life during cancer treatment: Research in this area highlights the importance of treating the psychological and emotional aspects of illness not just the physical, especially in vulnerable populations like children, who benefit greatly from normalization and a sense of control.

Lightning: B7-Improving Cancer Care

Monday, November 3, 2025, 4:25:00 PM - Hyatt Walker/Bannerman

Beyond the Binary: Engaging Trans & Gender Diverse people to improve cancer health services research - Poster #560

Presenter: Morgan Stirling, University of Manitoba

Other Authors: Cath Poitras, Kael McKenzie, Kieren de la Rue, Jay Gallant, Julia Peterson, Benjamin Carroll, Tristan Bilash, Natalie Mulaire, Jenny Duong, Annette Schultz, Janice Ristock, Nathan Nickel, Alyson Mahar

ABSTRACT_ID#2791

ABSTRACT

Background Community-engaged research is critical for addressing the inequities Trans and Gender Diverse (TGD) people experience across the cancer continuum. Despite the positive impact of engaged research, TGD people are rarely engaged in cancer research. This oversight increases TGD people's risk of harm in research (e.g. reinforcing damaging narratives about who TGD people are) and limits efforts to establish inclusive evidence-based care approaches. Engaging TGD people in cancer research supports developing equity-oriented models of care by privileging TGD people's voices and experiences. Methods Beyond the Binary is a community-based, mixed-methods study investigating approaches for improving the inclusion and representation of TGD people in cancer health services research. A community advisory board comprised of 9 TGD individuals across Canada with various types of cancer experiences are active research partners guiding this study. Advisors receive an honorarium. Engagement is grounded in principles of partnership, co-learning, mutual benefit, and commitment. Results Advisors actively participate in decision-making, priority-setting and developing study materials. We collaboratively created a terms of reference to guide our work together, which has included publishing a scoping review on TGD people's representation in cancer research and co-developing the study's data collection tools. Key to our success is establishing a supportive and transparent research environment, including employing flexible engagement methods to ensure all perspectives are heard. Conclusion Engaging TGD people in cancer research is imperative for improving the evidence base. Lessons learned to foster engagement will be discussed. We will share recommendations that support researchers engaging TGD people in cancer research.

LAY SUMMARY

- Engaging Trans and Gender Diverse people in cancer research requires establishing relationships based on trust, respect, and reciprocity.
- Engaging Trans and Gender Diverse people significantly improves our ability to ensure this research matters to the community.
- We think sharing our experiences will help other researchers with engaging Trans and Gender Diverse people in their own work.

POSTER PRESENTATIONS

Includes Lightning Presentations

Organized by Category and Poster Number

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Pulse of my Heart: Using Mixed-Methods to Explore an App Aimed at Reducing Death-related Distress in Mothers with Early-onset Cancer - Poster #2

Presenter: Muskan Manhas, Acadia University

Other Authors: Lianne Tomfohr-Madsen, Fiona Schulte, Stephanie Gauvin, Cara MacInnis

ABSTRACT_ID#2337

ABSTRACT

Psychological distress is high among those diagnosed with cancer who are parents of young children. These individuals must navigate heightened death anxiety related to fears about their children (e.g., fears of not being there for their children or not seeing them grow up). Considering growing rates of early-onset cancer diagnoses, providing tools to manage this distress is key. In light of this, we examined the feasibility/proof-of-concept of an intervention designed to reduce psychological distress faced by parents with early-onset cancer diagnoses. The mobile application-based writing exercise guided participants through prompts drawn from well-established social psychology literature on generating continuing bonds and interpersonal closeness, ultimately culminating in a legacy document for one's children. The work was directly informed by the lived experience of members of the research team and therefore developed through both patient and scientist perspectives.

Participants (N = 27, M Age = 40.5 years) completed the exercise. All individuals had children at or under 12 years of age. Quantitative feasibility metrics suggested the feasibility of the intervention. Preliminary quantitative analyses revealed participants had increased sense of meaning in life and decreased death anxiety from pre-to-post intervention. Qualitative interview findings further solidified that the exercise was deemed feasible by participants. Participants responded positively, describing the process as manageable and the experience as meaningful. This work highlights an innovative intervention with the potential to reduce distress and promote meaning-making in broader segments of this vulnerable population. Next, a RCT is planned to evaluate efficacy and broaden understanding of the intervention's psychological impact.

LAY SUMMARY

- Mothers with early-onset cancer are known to experience uniquely intensified psychological distress (e.g., due to fear of not seeing children grow up and/or worries about not forming lasting connections with them). This study examined the feasibility/proof-of-concept of a mobile application-based writing exercise that focused on allowing participants to create a legacy document for their children.
- Through the analysis of mixed-methods data, proof-of-concept and feasibility were established. Participants considered the exercise psychologically beneficial and meaningful. Preliminary analyses revealed that the exercise led to greater sense of meaning and reduced death anxiety.
- Additional work is planned to better understand and further assess the exercise's psychological impacts on a broader scale.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Empowering Adolescents Affected by Pediatric Cancer Through Exercise - Poster #3

Presenter: Amanda Wurz, Kids Cancer Care Foundation of Alberta

Other Authors: Carolina Chamorro-Vina, Chantelle Ball, S. Nicole Culos-Reed

ABSTRACT_ID#2883

ABSTRACT

Background: The Pediatric cancer patients and survivors Engaging in Exercise for Recovery (PEER) program by Kids Cancer Care (KCC) offers structured physical activity (PA) for children and adolescents affected by cancer since 2013. However, adolescents participate less than younger children, missing out on physical and psychosocial benefits. This reduced participation could hinder their chances of adopting an active lifestyle and impact their long-term health. **Aim:** Explore factors influencing adolescents' PA and inform the development of an adolescent-specific PEER program **Methods:** With informed consent, a survey was distributed to adolescents (12–17 years) diagnosed with cancer and adolescent-aged siblings at KCC summer camps (n=90) and through the KCC membership (n=160). The survey included demographic, medical, and PA questions (both closed and open-ended), including PA levels and perceived barriers and motivators. Closed-ended responses were analyzed descriptively; open-ended responses were analyzed using content analysis. **Results:** Sixty-two adolescents (Mage=13±1.89 years; 28 diagnosed with cancer, 34 siblings) completed the survey (n=57 camp, n=14 email). Most (76%) were not currently participating in PEER. Most (89%) reported being active by the completion of The Godin-Shephard leisure-time physical activity questionnaire. Top barriers to PA included lack of time, energy, and motivation. Top motivators included health benefits, mood improvement, and enjoyment. Four main themes emerged: physical, psychological, social, and opportunity-related factors. Top PA interests included badminton, volleyball, swimming, soccer, and climbing. **Conclusions:** Results directly informed the development of an adolescent-specific PEER program with five modules that is successfully running. Findings underscore the value of centering end-users' perspectives in program development.

LAY SUMMARY

Adolescents with cancer face unique challenges that affect their physical activity. While most teens in our study said they are active, many aren't joining structured programs like PEER. They told us that time, low energy, and lack of motivation often get in the way. Teens are motivated by how being active makes them feel. The top reasons they want to be active include staying healthy, improving their mood, and simply having fun. Activities they're most interested in include badminton, volleyball, swimming, soccer, and climbing. Listening to teens helps us build better programs. Based on what we learned, we redesigned the PEER program specifically for adolescents. The new version focuses on fun, skill-building sports and offers support to help teens feel more confident, socially connected, and ready to participate in other activities offer in the community setting.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Reducing Elevated Cardiometabolic Risk in Adolescents with Cancer through an Integrated Multidisciplinary Intervention (Protocol) - Poster #4

Presenter: Andra Dima, Université de Montréal

Other Authors: Véronique Bélanger, Johanne Kerba, Mélanie Napartuk, Isabelle Bouchard, Caroline Meloche, Nicolas Prud'homme, Serge Sultan, Caroline Laverdière, Léandra Desjardins, Daniel Sinnett, Valérie Marcil

ABSTRACT_ID#2425

ABSTRACT

INTRODUCTION Le cancer demeure la principale cause de décès liés aux maladies chez les enfants au Canada. Malgré les avancées en chimiothérapie et en radiothérapie, ces traitements agressifs entraînent des effets secondaires sévères qui compromettent l'apport alimentaire et l'état nutritionnel. Ils augmentent également le risque de complications cardiométaboliques pendant et après les traitements, les adolescents étant particulièrement vulnérables comparés aux jeunes enfants. **PROBLÉMATIQUE** L'adoption d'un mode de vie sain a le potentiel de réduire ce risque. Pour répondre à ce besoin, nous avons développé le programme multidisciplinaire VIE (Valorisation, Implication, Éducation), qui propose des interventions personnalisées en nutrition, activité physique et soutien psychosocial pour les jeunes patients atteints de cancer et leurs parents. Mis en œuvre au CHU Sainte-Justine entre 2017 et 2022, l'intervention nutritionnelle du programme a démontré son efficacité auprès des enfants. Cependant, l'approche familiale traditionnelle a obtenu des résultats mitigés auprès des adolescents, limitant ainsi son efficacité globale pour ce groupe d'âge. **HYPOTHÈSE** Nous émettons l'hypothèse qu'en adaptant l'intervention nutritionnelle dans le cadre du nouveau programme "VIE-Ado", spécifiquement conçu pour répondre aux besoins des adolescents, nous pourrions améliorer l'adhésion et ainsi maximiser son impact dans la prévention des complications cardiométaboliques pendant et après les traitements. **OBJECTIFS** 1. Co-développer l'intervention nutritionnelle en collaboration avec des anciens participants adolescents du programme VIE original. 2. Mettre en œuvre l'intervention VIE-Ado auprès d'adolescents nouvellement diagnostiqués et évaluer sa faisabilité, son acceptabilité et son adhésion, ainsi que son impact sur les comportements alimentaires et la santé cardiométabolique pendant et après les traitements.

LAY SUMMARY

Les adolescents demeurent une population largement sous-étudiée, malgré des données soulignant l'importance cruciale de répondre à leurs besoins spécifiques, notamment en contexte oncologique. Bien que les interventions sur les habitudes de vie aient démontré leur efficacité dans la prévention des complications cardiométaboliques dans d'autres populations, leur impact en oncologie pédiatrique reste peu documenté. Cette étude se distingue en étant la première à implanter une intervention personnalisée dès les premières semaines suivant le diagnostic, entièrement intégrée au parcours de soins en oncologie et conçue spécifiquement pour les adolescents. La méthodologie mixte employée constitue en elle-même une contribution majeure. Reconnue comme l'approche recommandée pour le développement d'interventions personnalisées en santé, la triangulation des données quantitatives et qualitatives assure la validité des résultats, favorisant ainsi une adaptation progressive et fondée empiriquement de l'intervention. Ce projet comble un manque important dans la littérature, en proposant un modèle novateur, ciblé et transférable à plus grande échelle, ouvrant la voie à des retombées à la fois cliniques et scientifiques majeures.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Understanding Medical Assistance in Dying Utilization in the Adolescent and Young Adult Cancer Population

- Poster #5

Presenter: Emilie Muth, University of Calgary

Other Authors: Miranda Fidler-Benaoudia, Andrew Harper, Nicole Maseja, Siwei Qi, James Silvius, Barry Bultz, Linda Watson, Claire Link, Janet Vandale

ABSTRACT_ID#2814

ABSTRACT

Background: Medical Assistance in Dying (MAID) use is increasing in Canada, but little is known about its use among adolescents and young adults (AYAs). We investigated MAID utilization, and reasons for choosing MAID, in the AYA cancer population. Methods: This mixed-methods retrospective study includes all individuals diagnosed with cancer at 15-39 years in Alberta who accessed MAID between 2017-2022. Descriptive statistics and mixed effects modeling was used to describe the individuals and symptom burdens in the last year of life, respectively. Chart review was undertaken to understand the context surrounding choosing MAID where qualitative thematic analysis was used to identify salient themes. Results: 34 AYA cancer patients were included, of whom 53% were female, 91% resided in Calgary or Edmonton, and 30% had children. Most common cancer diagnoses were gastrointestinal carcinomas (62%), breast carcinomas (24%), and central nervous system tumors (21%). 18 patients (53%) had a palliative care referral less than 3 months before death, only 2 of whom were diagnosed during this period. Symptom complexity rose in the last 6 months of life, with a clinically significant rise noted at 5-6 months before death for all 9 symptoms. Notably, depression and anxiety severity decreased in the last three months of life. Salient qualitative themes for choosing MAID include wanting control, avoiding a traumatic death for their children, and acceptance of their situation. Interpretation: More responsive palliative care referrals for AYA cancer patients could avoid symptom burden crisis in this population and potentially lead to better end-of-life care and quality-of-life.

LAY SUMMARY

AYAs are an overlooked population that faces distinct challenges during cancer care due to their unique life and developmental stage, requiring specialized and tailored health system responses. However, they are underrepresented in oncology, palliative care, and MAiD research. The young cancer patients in this study who accessed MAID experienced a unique rise in symptom burden 5-6 months before death, but half did not receive a palliative care referral until 3 months prior to death. The aim of palliative care is the prevention and relief of suffering. Palliative care referrals for AYAs should be more reactive to symptom burden changes because once symptoms start increasing, they increase rapidly. Ultimately, referrals 3-months prior to death is insufficient for AYAs because their symptom burden is already climaxing. This information should encourage providers to engage palliative care services earlier to prevent and reduce the suffering of young cancer patients at end-of-life.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Predictors of alcohol use among adolescent and young adult cancer survivors in Alberta: an exploratory mixed methods study - Poster #7

Presenter: Hannah Sell, Alberta Health Services

Other Authors: Ann Toohey, Tyler Cavanaugh, Matt Crist, Dylan O'Sullivan, Miranda Fidler-Benaoudia, Andria Morielli

ABSTRACT_ID#2423

ABSTRACT

Background: Adolescent and young adult (AYA) cancer survivors are at increased risk of late effects of cancer, including the development of a subsequent primary cancer. Alcohol has been linked to several types of cancer and high rates of heavy and binge drinking have been reported in this population. Little is known about key predictors of alcohol consumption in this population. The aim of this study is to explore factors influencing alcohol consumption among AYA cancer survivors in Alberta. **Methods:** We employed a participatory, mixed-methods approach that combined inclusion of (a) AYA cancer survivors and subject matter experts in research activities via advisory, consultative roles; and (b) multiple data sources including published literature, surveys, and a focus group. **Results:** Focus group and survey data collection and analysis are ongoing. A literature review found few studies focused on AYA cancer survivors; still, factors associated with increased alcohol consumption in survivors included emotional discomfort (depression, anxiety), influence of others (family/friends, healthcare providers), lower resiliency, positive attitudes towards substance use, male sex, lower education, lower age at drinking initiation, younger current age, greater time since completing treatment, greater fear of cancer recurrence or another health condition, current smoking, and lack of knowledge about negative effects of alcohol on health. **Conclusion:** Findings will guide the adaptation of an existing paper-based workbook into a web-based alcohol reduction tool tailored to support AYA cancer survivors. Additional research, age-specific information, and practical supports are needed to reduce the health, social, and economic burdens of cancer in this population.

LAY SUMMARY

There is a growing population of adolescent and young adult (AYA; aged 15-39 years) cancer survivors who are expected to live for many decades following cancer treatment. Engaging in healthy behaviours may reduce the risk of cancer recurrence, second cancers, chronic disease, and improve survival in this population. Alcohol has recently been highlighted as an important modifiable risk factor for cancer and has been linked to several types of cancer, including breast, colorectal, esophageal, laryngeal, liver, mouth, pharyngeal, stomach, and pancreatic. Unfortunately, AYA cancer survivors may consume alcohol at higher rates than their peers and engage in risky alcohol behaviours such as heavy and binge drinking. Little is known about why AYA cancer survivors consume alcohol or how to influence change in alcohol behaviours in this population. Moreover, there is limited age-specific information, cancer research, resources, and practical supports to address AYA cancer survivor's experiences with cancer at a young age. To address this gap in knowledge and resources, we aim to learn more about alcohol consumption in AYA cancer survivors to create an online alcohol reduction tool for this population.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Subsequent primary neoplasm risk among survivors of adolescent and young adult cancer: a population-based study from Alberta, Canada - Poster #8

Presenter: Arafat Ul Alam, Alberta Health Services

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#2553

ABSTRACT

Background: Survivors of adolescent and young adult (AYA) cancers are at an increased risk for subsequent primary neoplasms (SPN), but studies using Canadian data are limited. Here, we quantify the risk of SPNs among AYA cancer survivors in Alberta, Canada. Methods: The Alberta AYA Cancer Survivor Study is a retrospective, population-based cohort of 15-39 year-olds diagnosed with a first neoplasm from 1983–2017. Risk of SPN was assessed overall and among 5-year survivors. Standardized incidence ratios (SIRs), absolute excess risks (AERs; per 10000 person-years), and 30-year cumulative incidences were calculated. Results: Among 24,459 AYA cancer patients, 1,442 SPNs were identified, 1,129 of which occurred after 5-year survival. SPN risk was comparable when assessed overall and among 5-year survivors, with risk being 2-times higher than that expected in the general population, equating to approximately 30 excess SPNs. Among 5-year survivors, the 30-year cumulative incidence of SPNs was 17.7%, with incidence highest among 5-year survivors of Hodgkin lymphoma (HL; 22.7%) and cancers of the colon (23.5%), rectum (26.9%), breast (27.3%), and oral cavity/lip/pharynx (28.9%). Among these survivors, incidence was significantly higher than expected for breast SPNs after HL or breast cancer; digestive SPNs after HL, colon, rectal or breast cancer; respiratory SPNs after HL or breast cancer; hematopoietic SPNs after HL; and head/neck SPNs after oral cavity/lip/pharyngeal cancer. Conclusion: AYA cancer survivors are at an increased risk for all types of SPNs assessed. Earlier, risk-based screening may be warranted, particularly for breast, digestive and respiratory cancers where screening infrastructure already exists.

LAY SUMMARY

People who survived cancer between ages 15 and 39 in Alberta, Canada had twice the risk of developing a new, different cancer compared to the general population. This translates to about 30 more cases of new cancers for every 10,000 survivors each year compared to the general population. Survivors of Hodgkin lymphoma, breast, colon, rectal, and oral cancers had the highest long-term risk, with nearly 1 in 3 to 1 in 4 developing a new cancer within 30 years. Because many of these subsequent cancers are in breast, digestive organs, and lungs—where screening programs already exist—earlier or more frequent screening for survivors may help catch new cancers sooner.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Fertility Preservation Consultation among Adolescent and Young Adult Cancer Patients in Alberta: A Population-based Retrospective Cohort Study - Poster #9

Presenter: Henry Halder, Cumming School of Medicine, University of Calgary

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#2761

ABSTRACT

Fertility is a major concern for adolescent and young adult (AYA; 15-39 years) cancer patients, due to the long-term infertility risks associated with cancer treatments. This retrospective cohort study examined factors associated with fertility preservation consultation (FPC) among AYA cancer patients in Alberta diagnosed between 2003 and 2022, identified through the Alberta Cancer Registry. Additional clinical and sociodemographic information was collected from linked administrative health data. The main outcome was FPC within 1-year of diagnosis, defined using the Practitioner Claims billing codes (606 and 628). Logistic regression was used to identify associated factors with FPC. Among 19,207 cases, only 3.1% attended FPC within 1-year of diagnosis. Younger age groups (odds ratio [OR]: 15-19 = 2.07, 20-24 = 2.67, 25-29 = 3.80, 30-34 = 2.75), more recent cancer diagnoses (OR: 2008-2012 = 2.08, 2013-2017 = 3.25, 2018-2022 = 1.99), residents of Calgary (OR: 3.22), Central (OR: 1.17), or Edmonton (OR: 1.20) zones, and patients with moderate (OR: 4.92) to high (OR: 4.04) fertility risk cancers were associated with higher odds of attending a FPC within 1-year of diagnosis. Whereas males (OR: 0.32), residents of the North zone (OR: 0.84), patients with non-carcinoma cancers (OR: 0.49), and those who died within 1-year of diagnosis (OR: 0.46) had lower odds. Our findings highlight disparities in FPC among AYA cancer patients in Alberta, underscoring the need for targeted interventions to improve awareness and accessibility to fertility-related services. Addressing these gaps can also help AYA cancer patients to make informed reproductive health decisions.

LAY SUMMARY

Fertility preservation consultation (FPC) within 1-year of diagnosis remains low among adolescent and young adult (AYA) cancer patients in Alberta, despite the long-term infertility risks associated with cancer treatments. FPC depends on who and where you are living in Alberta. AYAs who are younger, reside in Calgary, Central, or Edmonton zones, and are diagnosed with cancers associated with moderate to high infertility risk are more likely to attend a FPC. Males, residents of the North zone, patients diagnosed with non-carcinoma cancers, and those with poor prognoses (i.e., death within 1-year of diagnosis) are less likely to attend FPC, highlighting disparities in cancer care within the AYA population. Addressing these gaps is essential to help all AYA cancer patients make informed decisions about their reproductive health.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Differential DNA Damage and Repair burden in proliferating neural progenitor cells - Poster #10

Presenter: Askar Yimit, University of Calgary

Other Authors: Peter Brownlee, Sean Nesdoly, Sorana Morrissy, Jennifer Chan, Aaron Goodarzi

ABSTRACT_ID#2844

ABSTRACT

While radiation therapy offers strong anti-cancer benefits, it can also have serious side effects for children undergoing brain tumor treatment. These include disruptions in white matter development, delays in functional and cognitive growth, and an increased risk of secondary tumors within the area that was treated. Age-dependent susceptibility to these adverse effects is tightly linked to critical windows of neurodevelopment, particularly during the periods of active neural cell differentiation, maturation, and the rapid onset of myelination in infancy and early childhood. We previously demonstrated that oligodendrocyte precursor cells (OPCs) exhibit heightened sensitivity to IR-induced DNA damage compared to other neural stem/progenitor cells (NSPC) populations – a difference that is associated with their decreased ability to form RAD51 filaments (a critical step in RAD51-mediated homologous recombination repair of DNA double-strand breaks). Recently, we performed genome-wide chromatin immunoprecipitation followed by sequencing (ChIP-seq) to map endogenous γ H2AX binding sites—a sensitive marker of DNA damage response—in both S-phase OPCs and NSPCs. Our data identified 71,794 γ H2AX-enriched regions in OPCs versus 29,154 in NSPCs, highlighting the elevated genotoxic stress burden in OPCs. Interestingly, we observed γ H2AX binding is depleted at transcription start sites and transcription termination sites, however OPCs show more γ H2AX peaks compare to NSPCs across all protein-coding genes. Furthermore, our data show cell type specific γ H2AX binding sites. Together, these findings establish a comprehensive and unbiased genomic framework for elucidating the mechanisms underlying OPC vulnerability to DNA damage. Future studies will examine different DNA damage pattern in response to radiation.

LAY SUMMARY

Children's brains are especially vulnerable to radiation: Radiation therapy for brain tumors can interfere with brain development in young children, particularly during early stages of brain growth such as cell differentiation and myelination, which can lead to delayed learning and higher cancer risk later. Oligodendrocyte precursor cells (OPCs) are more sensitive to radiation damage: Compared to other brain stem cells, OPCs show more DNA damage after radiation because they are less efficient at repairing it through a key pathway (RAD51-mediated repair). OPCs experience higher stress at the genetic level: Genome-wide analysis revealed that OPCs had over twice as many DNA damage markers (γ H2AX sites) as other neural stem/progenitor cells, indicating they suffer more genetic stress from radiation. These patterns were different depending on the cell type and gene location. These findings provide a clearer genetic map of how radiation affects brain cells differently and may help guide future strategies to protect sensitive brain cells during cancer treatment.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

The biomolecular origin of childhood brain radiation sensitivity during anti-cancer radiotherapy - Poster #12

Presenter: Askar Yimit, University of Calgary

Other Authors: Peter Brownlee, Sean Nesdoly, Sorana Morrissy, Jennifer Chan, Aaron Goodarzi

ABSTRACT_ID#3283

ABSTRACT

While radiation therapy offers strong anti-cancer benefits, it can also have serious side effects for children undergoing brain tumor treatment. These health consequences include disruptions in white matter development, delays in functional and cognitive growth, and an increased risk of secondary tumors. Age-dependent susceptibility to these adverse effects is tightly linked to critical windows of neurodevelopment, particularly during the periods of active neural cell differentiation, maturation, and the rapid onset of myelination in infancy and early childhood. We have demonstrated that oligodendrocyte precursor cells (OPCs) exhibit heightened sensitivity to radiation-induced DNA damage compared to other neural stem/progenitor cells (NSPC) populations – a difference that is associated with their decreased ability to form RAD51 repair factor filaments during homologous recombination-mediated repair of DNA double-strand breaks. To gain greater insight into the molecular origins of childhood brain radiation sensitivity, we have performed genome-wide chromatin immunoprecipitation followed by sequencing (ChIP-seq) to map endogenous γ H2AX binding sites (a sensitive marker of DNA double strand break formation) in OPCs and NSPCs. Our data identifies 71,794 γ H2AX-enriched regions in OPCs versus just 29,154 in NSPCs, highlighting elevated genotoxic stress in OPCs. Interestingly, we observed γ H2AX in OPC is enriched within gene bodies of transcribed genes. I will present this and supporting data using direct sequencing of DNA double strand breaks in cells using the novel Induce-seq platform. My findings establish a comprehensive and unbiased genomic framework for elucidating the mechanisms underlying OPC vulnerability to radiation.

LAY SUMMARY

Radiation harms developing brain cells: While radiation therapy helps treat brain tumors, it can severely affect brain development in children—especially during early stages of growth when the brain is rapidly forming white matter and myelin. OPCs are especially vulnerable: Among brain cells, oligodendrocyte precursor cells (OPCs) are much more sensitive to radiation-induced DNA damage than neural stem/progenitor cells (NSPCs). This is mainly because OPCs are less efficient at repairing DNA double-strand breaks, particularly due to impaired RAD51-mediated repair. OPCs show greater DNA damage at active genes: Using genome-wide mapping (ChIP-seq), researchers found that OPCs have over twice as many DNA damage sites (marked by γ H2AX) compared to NSPCs. These damage hotspots are mostly located within actively transcribed genes, revealing a strong link between gene activity and DNA breakage in OPCs. This was further validated using a new technique called Induce-seq.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

“Caught in Between”: Youth Cancer Care Experiences in a System Not Built for Them - Poster #13

Presenter: Joanne Tay, University of Windsor

Other Authors: Mohammad Jarrar, Jessica Kichler

ABSTRACT_ID#2387

ABSTRACT

Background: Adolescence is a formative period marked by identity development, growing independence, and evolving relationships. A cancer diagnosis during this stage can significantly disrupt these processes. Youth often face barriers to developmentally appropriate cancer care, particularly in regions with limited specialized services. They are also frequently excluded from care decisions, leading to disempowerment. While caregivers and healthcare providers play central roles in supporting youth, their perspectives remain underexplored, especially in non-urban settings. Addressing these gaps is essential to improving youth cancer care. **Objective:** This study explored the lived experiences of youths with cancer and their caregivers in Southwestern Ontario, alongside the perspectives of healthcare providers, to better understand strengths and gaps in local cancer care delivery. **Method:** Semi-structured interviews were conducted with youths (n=2), caregivers (n=6), and healthcare providers (n=6). Transcripts were analyzed using reflexive thematic analysis to generate key themes. **Results:** The six interrelated themes were: (1) Physical toll of treatment on youths; (2) Psychosocial effects on youths and caregivers; (3) Quality of interrelationships across the care triad; (4) Daily caregiving challenges and responsibilities; (5) Caregivers' critiques of cancer care systems; and (6) Need for youth-centered care that goes beyond medical treatment. **Conclusion:** Findings underscore the need to integrate youth perspectives, better support caregivers, and equip providers to deliver developmentally responsive, youth-centred cancer care that reflects the complexities of adolescence and family life. Future research should prioritize co-developing and evaluating regional, youth-centric cancer care models that reflect the complexity of adolescent experiences and family dynamics.

LAY SUMMARY

Adolescents with cancer face significant life disruptions: Cancer treatment upends key aspects of adolescence, identity, independence, and relationships, while youths are often excluded from care decisions, leaving them feeling powerless. Caregivers are overwhelmed: Parents report intense emotional and practical stress, struggling with fragmented care and feeling unsupported by the healthcare system. Youth-centered care is crucial to better care that young people deserve: To improve outcomes, cancer care must address the unique needs of adolescents, involving both youths and caregivers in decisions and ensuring care reflects the complexity of their experiences.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Engaging People with Lived Experience in Cancer Research Priority Setting: An Evaluation of the Adolescent and Young Adult Cancer Priority Setting Partnership Final Workshop - Poster #14

Presenter: Chantale Thurston, AYA CAN - Canadian Cancer Advocacy

Other Authors: Fiona Schulte, Tristyn Ranger, Tamara Rader, Brianna Henry, Mohamed Abdelaal, Michelle Blue, Timothy Buckland, Stefanie Del Gobbo, Lexy Dobson, Emily Gallant, Cheryl Heykoop, Mackenzie Jansen, Lorna Larsen, Nicole Maseja, Sapna Oberoi, Vinesha Ramasamy, Marlie Smith, Evan Taylor, Nadia Wendowsky, Sara Beattie, Jacqueline Bender, Kathryn Birnie, Sheila Garland, Lindsay Jibb, Melanie Noel, Perri Tutelman

ABSTRACT_ID#3031

ABSTRACT

Background: There is increasing interest in co-designing cancer research agendas together with people with lived experience (PWLE). However, there is limited empirical research to guide this practice. The aim of this project was to characterize the experiences of the PWLE who participated in the Canadian adolescent and young adult (AYA) cancer research priority setting partnership (PSP) final workshop. **Methods:** The AYA Cancer PSP project followed the James Lind Alliance methodology to establish the top 10 research priorities for AYA cancer research in Canada. The final step in the process is a final priority setting workshop attended by patients, caregivers, and clinicians where consensus is reached on the top 10 priorities using an adapted nominal group technique. Following the workshop, participants completed a questionnaire with closed- and open-ended questions about their experience. **Results:** Twenty-three individuals attended the workshop (15 patients/caregivers, 8 clinicians), of which, 19 (83%) completed the post-workshop questionnaire (12 patients/caregivers, 7 clinicians). All agreed (31%) or strongly agreed (69%) that they were able to share their thoughts and opinions in the workshop, and all but one participant agreed (25%) or strongly agreed (69%) that the process of determining the top 10 was fair and robust. Qualitatively, participants shared aspects of the workshop that were beneficial (e.g., group discussion) and areas for improvement (e.g., balance of patient/caregiver vs clinician perspectives). **Conclusions:** This investigation offers novel data on the experiences of PWLE engaged in cancer research priority setting and points to areas for future research to optimize engagement.

LAY SUMMARY

- Adolescent and young adult (AYA) patients, caregivers, and clinicians successfully participated in a virtual workshop to reach consensus on the top 10 research priorities for AYA cancer in Canada.
- The patients, caregivers, and clinicians who participated in the workshop generally rated their experiences favourably. They also identified aspects of the experience that could be improved.
- These findings help to advance to our understanding of how to meaningfully engage people with lived experience in cancer research priority setting activities.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Bridging the Divide: Strengthening Youth Oncology Support in Regional Communities - Poster #15

Presenter: Joanne Tay, University of Windsor

Other Authors: Mohammad Jarrar, Jessica Kichler

ABSTRACT_ID#2389

ABSTRACT

Background: Despite advancements in oncology care, systemic barriers continue to shape the experiences and outcomes of youths with cancer, their caregivers, and healthcare providers. Youth and families often report feeling underserved, particularly in psychosocial support, continuity of care, and transition services. Providers also face structural limitations, including resource constraints and fragmented systems, that hinder the delivery of comprehensive, high-quality care. These challenges are especially pronounced in regional communities, where youth cancer services remain underexamined. **Objective:** This study examined how oncology care systems function in practice, identifying structural strengths and gaps in service delivery to inform the development of equitable, integrated models of youth-centred care through stakeholder perspectives. **Method:** Semi-structured interviews were conducted with youth with cancer (n=2), caregivers (n=6), and healthcare providers (n=6). Transcripts were analyzed using reflexive thematic analysis to generate key themes. **Results:** Three themes highlighted structural enablers and constraints within youth oncology care systems: (1) The value and limitations of holistic support services, (2) The accessibility and availability of local resources, and (3) The quality and continuity of youth-centred oncology care. **Interest holders recommended:** (1) Enhancing equitable access to supportive services, and (2) strengthening system capacity, collaboration, and integration to better support youth and families. **Implications:** Findings underscore the need to invest in sustainable, youth-centred oncology care models that integrate medical, psychosocial, and developmental supports. Future research should examine how service coordination, provider training, and peer support mechanisms can be adapted and scaled to meet the unique needs of youth and families in regional communities.

LAY SUMMARY

Youth cancer care is fragmented: Many youths and families report feeling underserved, especially in rural areas. Key challenges include limited psychosocial support, poor continuity of care, and difficulties during care transitions. Systemic barriers hinder care delivery: Healthcare providers face resource constraints and fragmented systems that prevent them from delivering high-quality, comprehensive care to youth with cancer. Equitable, youth-centred care is essential: Interest holders recommend improving access to support services, enhancing system collaboration, and integrating medical and psychosocial care. This is crucial to ensuring sustainable, coordinated care that meets the distinct needs of youth and families, particularly in smaller communities.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

mTor Inhibition in the Management of Recurrent Metastatic Solid Pseudopapillary Tumor of the Pancreas - Poster #16

Presenter: Gabrielle Herzenberg, University Of British Columbia

Other Authors: Tariq Bhat, Yaoqing Shen, Gregor Reid, Chris Maxwell, Philipp Lange, Jon Bush, Daniel Renouf, Claire Gowdy, Marco Marra, Janessa Laskin, S. Rod Rassekh, C. James Lim, Rebecca Deyell

ABSTRACT_ID#2317

ABSTRACT

Background: Solid pseudopapillary epithelial neoplasm (SPEN) of the pancreas is rare, has low malignant potential, and predominantly affects young females. While complete surgical resection typically confers an excellent prognosis, systemic therapy for unresectable, recurrent or metastatic disease remains undefined. Case Presentation: A 10y previously-well female presented with a splenic mass and was initially treated with distal pancreatectomy and splenectomy, with histologic confirmation of pancreatic SPEN. She later developed metastatic peritoneal recurrence and underwent cytoreductive surgery, with concurrent whole genome and transcriptome sequencing of tumour and matched germline in a precision medicine study. In addition to a CTNNB1 mutation, her tumour showed outlier high expression of genes of the PI3K/mTOR pathway. In the setting of residual disease, a trial of mTOR inhibitor everolimus was initiated and radiographic improvement was achieved. Concurrently, a personalized chicken chorioallantoic membrane (CAM) xenograft model of the patient's tumour demonstrated response to mTOR inhibitor therapy. The patient discontinued everolimus after 14 months due to fatigue, with disease progression 6 months later. Everolimus was re-initiated, with subsequent radiographic benefit. Conclusion: This case illustrates the potential of precision genomic medicine to identify targetable pathways using transcriptomic data in rare tumors such as pancreatic SPEN. Outlier PI3K/mTOR pathway expression guided the selection of mTOR inhibition as a therapeutic strategy, further supported by a personalized xenograft model. The patient's clinical response to everolimus, aligned with genomic and functional data, underscores the potential value of integrating molecular profiling with patient-derived in vivo models to guide treatment in the absence of established therapies.

LAY SUMMARY

Rare pancreatic tumors like SPEN can come back or spread even after surgery, and there are currently no standard systemic treatments for unresectable SPEN. In this case, we explored a new treatment approach using a medication called everolimus that targets a specific cell growth pathway, the mTOR pathway. Genetic testing of the tumor revealed unusually high activity in the mTOR pathway, a signal that tells cells to grow and divide. Based on this, the patient was treated with everolimus, a drug that blocks this pathway, and her tumor responded well. When the patient went off everolimus due to fatigue, her disease progressed. Then, after going back on everolimus, her disease re-stabilized. We also tested the drug on a personalized tumor model grown in chicken eggs (called a chicken chorioallantoic membrane (CAM) model), and it showed a similar response to mTOR inhibitor therapy. This innovative approach helped confirm that the drug was a good match for the patient's specific tumor biology and is a useful tool to guide treatment decisions.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Understanding cancer prevention knowledge among adolescents and young adults – a cross-sectional study

- Poster #18

Presenter: Jennifer Anderson, Acute Care Alberta

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#3063

ABSTRACT

Introduction: Risk factor reduction is key to improving cancer outcomes, especially as cancer rates rise among youth and young adults. Thus, we examined cancer knowledge in this group to inform health literacy initiatives. Methods: This cross-sectional study used multiphase probability sampling to recruit Albertans aged 16-39 years. We collected self-reported cancer knowledge and behaviours through an electronic questionnaire. Demographic characteristics and cancer prevention knowledge, including awareness of risk factors and screening programs, were described using means and counts. Predictors of cancer knowledge were identified through backwards stepwise logistic regression using Akaike Information Criterion. Multivariable logistic regression estimated the association between health behaviours (i.e., tobacco use, cannabis use, alcohol use, physical activity level) and poor cancer knowledge, with adjustment for confounders identified in the prediction model. Results: The 1,008 respondents were on average aware of 8/11 cancer risk factors assessed. Cigarettes (91%), secondhand smoke (89%), family history of cancer (87%), HPV infection (81%), alcohol (73%), old age (71%), and obesity (67%) were the most well-known risks. Fewer participants recognized sunburns (59%), physical inactivity (56%), low fruit and vegetable consumption (37%), and red meat consumption (36%) as cancer risks. Younger age, living in central (OR: 1.51, 95%CI: 0.87-2.63) or northern (OR: 2.03, 95%CI: 1.08-3.80) Alberta, trades education (OR: 1.57, 95%CI: 0.82-2.98), lower income, and being an ethnic minority (OR: 0.72, 95%CI: 0.48-1.08) were the most relevant predictors of poor cancer knowledge. After adjustment, we estimated that with each unhealthy behaviour, there is a 1.2-fold increase in the odds of poor cancer knowledge.

LAY SUMMARY

Some modifiable risk factors are not well known among Alberta's youth. Only a third of youth were able to recognize dietary choices, including low fruit/vegetable consumption and red meat consumption, as cancer risk factors. Prevention efforts should focus on increasing education about the importance of diet for health and longevity. No one characteristic can perfectly predict cancer knowledge. Instead, cancer knowledge depends on many important social identities including age, education, location, income, and race. Further, some groups are more educated about some risk factors and less educated about others. For example, people with trades education were significantly less likely to recognize cigarettes and second-hand smoke as risk factors; however, compared to people with university and college degrees, they were more likely to rate physical inactivity as a cancer risk factor. After removing the effect of factors like age, education, and income, there is still a clear relationship between cancer knowledge and behaviours. People living unhealthy lifestyles, by smoking cigarettes, using cannabis, consuming more alcohol, and/or infrequently exercising, are less informed about cancer risk factors. In order to increase knowledge and reduce negative consequences of low knowledge, future cancer prevention efforts will need to take a different approach.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Adolescents and Young Adults Becoming Physically Active After Cancer Trial (AYA PACT): Study overview and baseline characteristics - Poster #19

Presenter: Leanne Dickau, Alberta Health Services

Other Authors: Kerry S. Courneya, Christine M. Friedenreich, Miranda Fidler-Benaoudia

ABSTRACT_ID#2902

ABSTRACT

Introduction: Survivors of adolescent and young adult (AYA) cancer experience late-effects due to their cancer and its treatment. Interventions exploring whether exercise can improve physical fitness and quality of life after cancer in this population are needed. Methods: The Adolescents and Young Adults Becoming Active After Cancer Trial (AYA-PACT) is a randomized controlled trial examining if a home-based, mobile health physical activity intervention can increase physical activity levels by at least 90 minutes/week in AYA cancer survivors compared to a control group of AYA cancer survivors. The primary outcome is change in minutes of moderate-vigorous intensity physical activity (MVPA) time per week between the two groups from baseline to 12 months, as measured by an ActiGraph® device. Changes in the following secondary outcomes will also be assessed: cardiorespiratory and muscular fitness, sedentary and sleep behaviours, frailty, psychosocial health, and theory-based mediators of MVPA. Participants: Albertans diagnosed between the ages of 15-39 years with a first primary cancer, and within 12 months of active treatment completion, were eligible for enrollment. Results: 287 AYA cancer survivors were randomized to the intervention (n=145) or control (n=142) group. Most participants were female (78%) and living in Calgary (37%) or Edmonton (25%). Breast (27%), thyroid (18%) and cervical (7%) were the most common cancer sites. The median age at diagnosis was 35.0 years, while the median age at participation was 36.6 years. As of May 2025, 213 (85%) and 176 (84%) of participants completed measurements at the 6- and 12-month timepoint, respectively.

LAY SUMMARY

Cancer diagnoses are increasing in the adolescent and young adult population (AYA). Treatment from cancer can cause long-lasting side effects. Exercise has been shown to positively affect both symptom control and quality of life. The goal of the study is to see if a home-based physical activity intervention can increase physical activity in AYA cancer survivors. The study has enrolled 287 participants and has good retention rates at the six-month (85%) and twelve-month (84%) time points.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Rates of Non-Fatal Self-Injury Among Adolescent and Young Adult Survivors of Cancer: A Population-Based Matched Cohort Study - Poster #20

Presenter: Jingxuan He, University of Toronto

Other Authors: Sumit Gupta, Paul Nathan, Rinku Sutradhar

ABSTRACT_ID#2732

ABSTRACT

Background: A cancer diagnosis in adolescent and young adults (AYA) can result in prolonged psychological distress. While risk of non-fatal self-injury (NFSI) shortly after diagnosis has been studied, little is known about the risk in long-term survivors. This study aims to examine NFSI risk among AYA survivors of cancer and compare them with matched cancer-free controls in Ontario, Canada. **Methods:** This was a retrospective population-based matched cohort study. All 5+ years survivors of AYA cancer, primarily diagnosed at ages 15 to 39 between 1987 and 2016, were 1:5 matched to cancer-free controls based on birth year, sex, and forward sortation area. To examine the association between survivorship and the risk of NFSI, adjusted Cox proportional hazards regression models were implemented, accounting for the matched design. Analyses were further stratified by median age at diagnosis. **Results:** The cohort included 48,374 survivors and 241,870 matched controls. In the first five years after reaching the five-year survival mark, survivors had a significantly higher risk of NFSI compared to controls (adjusted hazard ratio [HR], 1.15; 95% CI, 1.01-1.31; $P=0.029$); no significant risk difference was found beyond five years. Among individuals diagnosed at age 33 years or younger, survivors experienced an elevated risk within the first five years (HR, 1.27; 95% CI, 1.08-1.49; $P=0.003$), while no significant difference was observed in those diagnosed at older ages. **Conclusion:** AYA cancer survivors diagnosed at younger ages faced significantly increased risks of NFSI. These findings highlight the importance of long-term psychosocial surveillance and mental health support into survivorship.

LAY SUMMARY

1. Five-year AYA survivors of cancer had a 15% higher risk of non-fatal self-injury (NFSI) compared to matched cancer-free controls within the initial five years following survivorship.
2. This elevated risk of NFSI was found among survivors diagnosed at 33 years or younger.
3. Long-term surveillance for mental health is needed for younger survivors.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Oncofertility knowledge and use among survivors of adolescent and young adult cancer - Poster #21

Presenter: Malak Ibrahim, University of Calgary

Other Authors: Miranda Fidler-Benaoudia

ABSTRACT_ID#2941

ABSTRACT

Introduction: Each year, 9000 adolescents and young adults (AYAs) are diagnosed with cancer in Canada. AYAs have unique and often unmet needs due to experiencing cancer during critical developmental, social, and reproductive years. Although fertility is one of the greatest concerns among AYAs, the extent to which oncofertility consultations or related services are used remains uninvestigated in an Albertan population. More research is needed to understand factors influencing family building in this population, including their knowledge, needs, and potential barriers and facilitators to fertility care. **Objective:** This study aims to understand the impact of cancer during adolescence and young adulthood on fertility, and explore oncofertility knowledge, fertility experiences, and sexual health in AYA cancer survivors in Alberta using an electronic REDCap questionnaire. **Methods:** To inform questionnaire development, PubMed and CINAHL databases were systematically searched with terms including oncofertility, fertility preservation, and adolescent and young adult cancer. A combination of pre-validated tools, such as the PROMIS-Sexual Function and Satisfaction scale, and original questions derived from other studies were included. After questionnaire generation is complete, we will conduct a population-based cross-sectional study using a population-based mail-out via the Alberta Cancer Registry. All living Albertan AYA cancer survivors diagnosed from 2015-2024 and aged at least 18 years at mail-out will be contacted. **Results:** 127 questions are included in the female iteration of the questionnaire, and 120 questions in the male iteration. Piloting and review of questionnaire is ongoing with patient advisors and co-investigators, with the expectation of survey mail-out occurring in Fall 2025.

LAY SUMMARY

Fertility is important to AYA cancer survivors, but uptake of fertility services is low in Alberta. AYAs have unique and often unmet needs due to experiencing cancer during critical developmental, social, and reproductive years. Questionnaires can help investigate AYAs knowledge, experiences, and information needs to improve fertility care in Alberta

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Reducing Inequities in Community Services for Young Adults Affected by Cancer: Co-Development of an Innovative Guide - Poster #22

Presenter: Karine Bilodeau, University of Montreal

Other Authors: Charlotte Gélinas-Gagné, Katia Dumont, Ludovic Tamaro, Nicolas Fernandez, Leandra Desjardins, Billy Vinette, Dominique Tremblay, Denise Page, Marco Décelles, Deborah Pascale, Pascale Rousseau, Serge Sultan

ABSTRACT_ID#2932

ABSTRACT

CONTEXT: Young Adults (YA) living with and beyond cancer are among the most underserved population, both within the healthcare system and in community-based support. Despite significant psychosocial needs, they remain under-researched and inadequately supported. This project aims to bring together YA living with cancer and key stakeholders to strengthen the community support available to this population. **OBJECTIVE:** Through a co-development approach, the project seeks to create a shared framework that will serve as a practical guide for community-based cancer organizations. This guide will support the adaptation of existing services to better meet the needs of YA, a first step toward a more inclusive, relevant, and accessible community services offering. **METHODS:** The nominal group technique was used to foster collaboration between community cancer organizations and YA during three virtual meetings. This structured method included four key steps: 1) silent idea generation, 2) idea sharing, 3) group discussion, and 4) prioritization through voting. **RESULTS:** Eighteen individuals participated in the project, including nine young adults (mean age = 33.3 years, range 28–39) diagnosed with cancer (33% breast, 67% hematological), and nine professionals working in community-based oncology settings, representing seven Canadian organizations (four provincial [Quebec], three national). Six virtual small-group meetings held via Teams enabled the progressive co-construction of the guide. **CONCLUSION:** Ultimately, the developed guide will serve as a concrete tool to support over 60 community oncology organizations in adapting their services and resources to better reflect the realities of YA, contributing to greater equity in care and support for this underserved population.

LAY SUMMARY

Young adults with cancer are underserved by healthcare and support services in Canada and face unique psychosocial challenges. To better leverage community-based cancer organizations, a guide was co-developed to support the adaptation of community services to the needs of young adults. Through this project, community oncology services can be tailored to better meet the needs of young adults, helping to reduce disparities in care and support for this population.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Overall and late mortality among 24 459 survivors of adolescent and young adult cancer in Alberta - Poster #23

Presenter: Miranda Fidler-Benaoudia, Alberta Health Services

Other Authors: Taylor Hughes, Ruth Diaz, Sarah McKillop, Paul Nathan

ABSTRACT_ID#2388

ABSTRACT

Background Adolescent and young adult (AYA) cancer survivors are at an increased risk of premature mortality due to their cancer and its treatment. **Methods** The Alberta AYA Cancer Survivor Study is a retrospective, population-based cohort of individuals diagnosed with a first primary neoplasm at age 15–39 years in Alberta, Canada, between 1983 and 2017. Standardised mortality ratios, absolute excess risks (AERs; per 10,000 person-years), and cumulative mortality probability were calculated. Causes of death were categorised as due to (i) recurrence/progression of the AYA cancer, d(ii) subsequent primary neoplasm (SPN), and (iii) non-neoplastic causes. **Findings** Among the 24,459 survivors, 5916 deaths were observed, which was 11.4 times (95% CI 11.1–11.7) that expected, equating to 191.6 (186.2–196.9) excess deaths; correspondingly, 5-year survivors had 4.2 times (4.0–4.4) more deaths than expected, equating to 74.3 (69.8–78.8) excess deaths. Increased age at diagnosis, poorer neighbourhood income quintile, AYA cancer type, and initial treatment plan were identified as important risk factors for mortality. While recurrence/progression was the main cause of excess mortality, the majority of deaths beyond 10 years from diagnosis were due to SPNs and non-neoplastic causes among survivors of endometrial cancer, testicular cancer, and Hodgkin lymphoma. Cumulative mortality significantly decreased among more recently diagnosed survivors for all-cause, recurrence/progression, and SPN deaths, suggesting that long-term survival is improving. **Interpretation** AYA cancer survivors experience substantial excess mortality. Given the high burden of late SPN and non-neoplastic deaths, survivors of endometrial cancer, testicular cancer, and Hodgkin lymphoma are notable populations that might benefit from prevention strategies.

LAY SUMMARY

Our comprehensive investigation of mortality among AYA cancer survivors identified significant increased risks of mortality. With most of the excess mortality due to recurrence or progression deaths, particularly before 5-year survival, considerable efforts must remain focused on maintaining long-term disease-free survival. However, our study identifies three AYA survivor populations for whom opportunities for prevention exist and interventions could lead to a meaningful decrease in morbidity and late mortality. These findings have clear public health implications: to serve as a catalyst for developing strategies seeking to reduce or mitigate late effects in these populations; and to support the need for a health system assessment to understand how transitions to healthy survivorship for AYA cancer survivors can be improved.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

A Qualitative Exploration of the Psychosocial Burdens Faced by Adolescents and Young Adults with Hematologic Malignancies - Poster #26

Presenter: Mashiad Mostafa, BC Cancer Research Institute | PHSA

Other Authors: Jonathan Avery, Karen J. Goddard, A. Fuchsia Howard, Andrea Lo, Stuart Peacock, Meera Rayar, Clara Westwell-Roper, Helen McTaggart-Cowan

ABSTRACT_ID#2321

ABSTRACT

Background: Adolescents and young adults (AYAs) diagnosed with hematologic malignancies (HMs) between the ages of 15-39 years face long-term physical and psychological challenges persisting beyond cancer. In this study, we qualitatively described the mental health challenges, personal coping strategies, and interactions with psychosocial support services. Methods: We conducted one-on-one semi-structured virtual interviews with AYAs with an HM diagnosis in the last 10 years. A semi-structured topic guide, co-created with patient partners, explored the psychosocial burdens faced by AYAs with HMs in British Columbia, Canada. Interviews were transcribed and thematically analyzed. Results: Nineteen AYAs (n = 11 women, 7 men, and 1 agender) participated; three were in active treatment and 16 in the after-treatment phase. Participants discussed how systemic barriers, physical challenges, and uncertainty surrounding cancer diagnosis and treatment deteriorated their functional ability to perform daily tasks, contributing to their psychological distress. Participants utilized a range of coping tools, relying on their family members, friends, care providers and psychosocial support. Many adopted mechanisms to repress their mental health awareness (i.e., self-preservation and forced normalcy) during the diagnosis and treatment phases. As treatment progressed, individuals sought emotional resonance (peer support) beyond their primary anchors (i.e., friends and family) and took control of their psychosocial well-being through knowledge and building resilience. A few opted for over-the-counter drugs and substances to aid in managing distress. Conclusion: AYAs seek long-term, age-specific psychosocial care starting at diagnosis, with follow-ups for early recognition and management of cancer trauma.

LAY SUMMARY

Mental health challenges go beyond cancer: Many adolescents and young adults (AYAs) with blood cancer experience long-term psychological impacts on their future life choices and desired social roles. Coping is as unique as individual needs: AYAs acknowledge that coping is not a one-size-fits-all approach, utilizing a wide range of strategies for their mental health challenges. While some rely on friends and family, others seek emotional resonance from peers who have had similar experiences. Some individuals turn to self-preservation, seeking a sense of normalcy to help them get through treatment. A few have found solace in over-the-counter drugs and cannabis. AYAs need early, ongoing, and age-appropriate support: Participants desired tailored psychosocial support that starts at the time of diagnosis, with periodic follow-ups to cope with the ongoing cancer-induced trauma.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Engineered T-Cells Targeting Minor Histocompatibility Antigens Exhibit Potent Anti-Leukemia Activity - Poster #27

Presenter: Nasser Masroori, CR-HMR

Other Authors: Annabelle Minguy, Christina Sawchyn, Vibhuti Dave, Jessica Trottier, Jean-Philippe Bastien, Jaime Lionel Sanchez-Dardon, Nafiseh Keshavarzian, Frédérick Antoine Mallette, Denis-Claude Roy

ABSTRACT_ID#3088

ABSTRACT

Although significant progress has been made in cancer treatment, it remains a leading cause of death in children and adults. Gene and cell engineering technologies—particularly T-cells engineered with CARs—have demonstrated remarkable success in treating B-cell malignancies. However, extending CAR-T cell therapy to non-B cell cancers has been hindered by the lack of suitable target antigens. In earlier work, we identified novel MiHAs preferentially expressed on hematopoietic cells. In a Phase-I clinical trial, leukemia patients who relapsed after an allogeneic stem cell transplantation were treated with ex-vivo expanded anti-MiHA T-cells. These cells exhibited encouraging activity and an excellent safety profile. Building on these findings, we are developing a next-generation T-cell therapy using genetic engineering. We isolated and characterized MiHA-specific TCRs from ex-vivo expanded T-cells derived from healthy donor PBMCs. To validate functionality, we used CRISPR/Cas9 to knock out endogenous TCRs in Jurkat T-cell lines and transduced them with the MiHA-specific TCR constructs. These modified Jurkat cells showed robust expression of the CD69 activation marker upon antigen recognition. We further engineered primary human T-cells by knocking out their endogenous TCRs and introducing MiHA-specific TCRs. Following expansion, these cells underwent thorough phenotypic and functional assessments. When co-cultured with MiHA-pulsed or MiHA-expressing leukemia cells, the engineered T-cells demonstrated potent cytokine secretion and strong cytotoxic activity, specifically targeting MiHA-positive cells. In vivo, these cells significantly extended survival in mouse models of ALL and AML. Together, these results provide strong preclinical evidence supporting the therapeutic potential of primary T-cells engineered to target MiHAs in hematologic cancers.

LAY SUMMARY

Targeting Minor Histocompatibility Antigens (MiHAs) for Leukemia This work advances T-cell therapy by focusing on MiHAs—antigens selectively expressed on hematopoietic cells—as promising targets for treating leukemia, especially in high-risk patients with hematologic malignancies. **Precise T-Cell Engineering Using CRISPR/Cas9 and Lentivirus** By knocking out native TCRs and introducing MiHA-specific TCRs into primary human T-cells, the study achieved robust, antigen-specific activation and function. These engineered T-cells demonstrated strong cytokine secretion and cytotoxicity against MiHA-positive leukemia cells. **Demonstrated Preclinical Efficacy and Translational Potential** In vivo experiments in mouse models of AML and ALL showed that MiHA-specific T-cells significantly prolonged survival. These findings, along with earlier clinical safety data, provide strong preclinical support for the development of MiHA-targeted T-cell therapies in hematologic malignancies.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

An environmental scan of services for adolescents and young adults diagnosed with cancer across Canadian pediatric and adult tertiary care centres - Poster #28

Presenter: Nicole Rutkowski, Arthur J.E. Child Comprehensive Cancer Center

Other Authors: Sara Beattie, Fiona Schulte, Chantale Thurston, April Boychuk, Marie de Guzman Wilding, Jan-Willem Henning, Perri Tutelman

ABSTRACT_ID#3043

ABSTRACT

Background: Adolescents and Young Adults (AYA: 15-39 years) diagnosed with cancer face unique medical and psychosocial challenges requiring specialized care. The purpose of this study was to conduct an environmental scan of AYA-specific programming and services currently offered across Canadian tertiary care centres. **Methods:** Key informants from pediatric and adult cancer centers in Canada were asked to complete a survey regarding AYA-specific programming and services at their site. Informants reported on program logistics, AYA specialized staff and training opportunities, collaboration between centers, funding, and specific areas of interest for AYA care such as palliative care, fertility, fatigue, and sexual health. Survey responses were collected between June 2024 and April 2025. **Results:** Surveys were completed from 14/17 (82%) pediatric sites and 19/23 (83%) adult sites. About half of pediatric sites (n=8/17) and adult sites (n=9/23) reported offering some degree of AYA-specific cancer services and programming. Among centres that did not report offering any services or programming, approximately a third reported to be working on developing AYA programming. Age range for AYA services was 15-40 years. Only 6 sites reported to offer specialized AYA training. Several barriers were reported such as the need for collaboration among institutions and improvement of oncofertility services. **Conclusion:** AYA-specific programs and services are available at about half of the pediatric and adult tertiary care centres surveyed from across Canada. Disparities exist in regards to geographic availability of services, the range of services available, and the populations served. Findings will inform the development of AYA services in Canada.

LAY SUMMARY

Only about half of Canada's major pediatric and adult cancer hospitals have special programs for young people with cancer. Available programs were offered to people aged 15-40 and included services such as support for family and caregivers, information and support around return to work or school, fertility, sexual health, and palliative care. A few hospitals reported working on programming, including making websites with information for young people, creating education for doctors and nurses providing care to teens and young adults, programs that provide information on sexual health, and hiring more people trained to care for teens and young adults, but reported support and training were limited. Some hospitals for children and adults reported working together or sharing staff, but costs, training opportunities, difficulties working across two sites, and lack of support from management were obstacles. The study found big differences in where and how care is provided across Canada, which means that teens and young adults with cancer may have access to many, a few, or no services depending on where they live in Canada. Programs were paid for mostly by private donations, which may play a role in why some areas in Canada may not offer services.

Adolescent and Young Adult Cancer

Sunday, November 2, 2025

Pregnancy outcomes in adolescent and young adult (AYA) cancer survivors in Alberta, Canada - Poster #29

Presenter: Taylor Hughes, Alberta Health Services

Other Authors: Miranda Fidler-Benauodia

ABSTRACT_ID#2664

ABSTRACT

Background: Fertility is a primary concern identified by adolescent and young adult (AYA) cancer patients. As literature on pregnancy rates and outcomes among AYA cancer survivors is limited, we aimed to understand the impact of cancer on subsequent chance of pregnancy and perinatal health. Methods: We examined pregnancy outcomes and complications after cancer diagnosis in a subpopulation of the Alberta AYA Cancer Survivor study. This is a retrospective, population-based cohort including 10833 females diagnosed with a first primary neoplasm between 15-39 years of age in Alberta during 2005-2022. Preliminary descriptive results are provided here. Linkage to matched cancer-free controls to compare outcomes to the general population is ongoing. Results: 5262 AYAs had at least one birth between 2005-2024, equating to 9463 total births, of which 2984 (31.2%) occurred after diagnosis. Nearly all were live births (99.5%) regardless of timing since diagnosis; however, there was a greater proportion of preterm births after diagnosis compared to before (14.6% versus 8.4%). The most common cancer diagnosis sites preceding births were thyroid (n=798, 27.1%), central nervous system (n=500, 17.0%), and breast (n=301, 10.2%). The median time from cancer diagnosis to first birth was 3.29 years (IQR=1.57, 5.68). There were greater proportions of risky pregnancies requiring supervision and hypertension complicated pregnancies after diagnosis compared to before (risky pregnancy: 10.4% versus 5.2%, hypertension: 10.1% versus 7.4%). Comparisons to matched controls will be discussed. Conclusions: We provide a preliminary description of pregnancy outcomes in AYA cancer survivors in Alberta, Canada and will discuss outcome rates in comparison to the general population.

LAY SUMMARY

The Alberta AYA Cancer Survivor study is the largest AYA-specific cancer cohort in Canada and is one of the few AYA cohorts in the world that includes the entire AYA age range (15-39) and all AYA cancer sites. This study provides a rare opportunity to compare pregnancy outcomes between AYA cancer survivors and the general population, and to see how different factors, like cancer type, age at diagnosis and treatment might affect those outcomes. After survival, fertility is the second biggest concern for AYA cancer patients; yet, there is not much information available on this topic. The limited research that does exist suggests that pregnancy complications, infertility and poor pregnancy outcomes are more frequent in AYA cancer survivors, but most of this research is potentially outdated and not based in Canada. The findings of this study will fill that gap by providing modern, Canadian-based evidence to inform future research, guideline development, and clinicians and AYA cancer survivors about pregnancy after cancer.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Exploring the needs of adolescents and young adults with cancer at diagnosis to inform the co-design of an intervention - Poster #30

Presenter: Perri Tutelman, University of Calgary

Other Authors: Emily Memedovich, Sara Beattie, Se'era Anstruther, Megan Hessel, April Boychuk, Jan-Willem Henning, Dana Male, Sarah McKillop, Bobbi Michaud, Fiona Schulte

ABSTRACT_ID#2455

ABSTRACT

Background: Adolescents and young adults (AYAs) diagnosed with cancer have unique needs at the time of diagnosis which are often left unmet. The objectives of this study were to: (1) explore the experiences and unmet needs of AYAs with cancer around the time of diagnosis; and (2) determine the key components of an intervention to support AYAs at the time of diagnosis. **Methods:** A qualitative descriptive design using semi-structured focus groups was conducted. AYAs (N=14, 19-39 years old, 64% women) in Alberta, Canada who were within 2 years of diagnosis participated in one of two focus groups (n=6-8 participants per group). Focus groups were audio-recorded, transcribed, and analyzed using conventional content analysis. **Results:** Participants described various unmet needs around the time of diagnosis related to information (e.g., lack of awareness of supports available, whom to contact), supportive care (e.g., financial, mental health, rehab-related needs), and difficulty with navigating and advocating within the healthcare system. Participants uniformly endorsed the need for an intervention offered around the time of diagnosis to support AYAs with these unmet needs. Key components for the intervention focused on: desired knowledge about available supports and services available, mental health psychoeducation and self-management strategies, and self-advocacy skills. Participants also highlighted their desire to meet other young patients. **Conclusions:** AYAs newly diagnosed with cancer face many unmet needs. Participants perceived a benefit to addressing these unmet needs through a universally-offered intervention. Findings will be used to inform the co-design of an intervention for AYAs newly diagnosed with cancer.

LAY SUMMARY

·Adolescents and young adults (AYAs) diagnosed with cancer have many unmet needs around the time of diagnosis. These unmet needs are often related to needing more information about the programs and services available, needing support with the psychological and social impacts of cancer, and needing to learn how to navigate and advocate for themselves within the healthcare system. ·AYAs reported that having access to a program around the time of diagnosis that addresses these unmet needs would be beneficial. Specifically, they expressed that a program should provide information about the services available within the system to support their needs, strategies to manage the mental health impacts of cancer, and learning about how to navigate and advocate within the healthcare system. ·The learnings from this study will guide the development of a new, innovative program that will help support AYAs around the time of diagnosis.

Adolescent and Young Adult Cancer

Monday, November 3, 2025

Understanding the fertility, sexual health and mental health needs of Adolescents and Young Adults (AYA) with cancer in Eastern Ontario - Poster #32

Presenter: Veronica Reaume, Division of Medical Oncology, Department of Medicine, The Ottawa Hospital Cancer Centre, Ottawa, ON

Other Authors: Sarah Cleyn, Haydn Bechthold, Alicia Hilderley, Andrea Librado, Kylie Mcneil, Tinghua Zhang, Amirtha Srikanthan

ABSTRACT_ID#2353

ABSTRACT

Adolescents and young adults (AYA; 15-39 years) with cancer face unique challenges due to their life stage. This study aimed to evaluate factors impacting fertility, sexual health, and mental health within the Eastern Ontario AYA population. An anonymous electronic survey, co-designed by AYA patients, was administered to AYA patients diagnosed between 2015-2023 at the Ottawa Hospital. Questions focused on fertility, sexual health, mental health, cancer diagnosis and treatment, barriers in supportive care, overall quality and challenges of cancer care. Descriptive statistics were reported. Covariates between groups were compared using Chi-square/Fisher's Exact tests and independent t-tests. Two-tailed test were used for statistical tests at 5% level significance. Three hundred thirteen AYA responded (response rate 30.2%), being predominantly female (71.8%), in a relationship at diagnosis (60.4%), and employed fulltime (59.5%). 37.4% were current patients. Median age at diagnosis was 33 years (range 16-39 years). Most respondents discussed fertility before therapy (77.8%); however, more males were offered fertility preservation (65.9% vs 52%, $p < 0.05$), more females reported negative sexual health impacts (54.6% vs 36.6%, $p < 0.05$). Most respondents identified negative mental health impacts (86.3%), without gender difference. Negative sexual health impact was associated with: age > 30 years at diagnosis, being unemployed/on disability, and a parent at diagnosis ($p < 0.05$); cancer status (current patient vs survivor) did not have association. No factors impacted mental health. AYA program development in Eastern Ontario should focus on mental health, fertility preservation discrepancies, and sexual health, particularly in older AYA patients, those unemployed/on disability, and those who are parents at diagnosis.

LAY SUMMARY

Adolescents and young adults (AYA, ages 15-39 years) diagnosed with cancer face unique challenges due to their life stage that are not well addressed by regional cancer programs, particularly fertility, mental health, and sexual health care. Within our study, sexual health is more likely to be negatively impacted in older AYA patients (over 30 years old), those unemployed or on disability, and those who are parents at diagnosis. There are gender differences in who is offered fertility preservation and who reports negative sexual health impacts in AYA patients. Women are more likely to have negative sexual health impacts than men during their cancer care. Men are more likely to be offered fertility preservation before cancer treatment than women, despite both men and women having a desire for fertility preservation. A negative impact on mental health is common in AYA cancer patients. No factors investigated impacted the likelihood of negatively impacted mental health for AYA patients.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Opportunities created by Connect care for cancer prevention-oriented patient care and research - Poster #35

Presenter: Adetola Aboyeji, Primary care Network

Other Authors: Gary Teare, Kamala Adhikari

ABSTRACT_ID#2611

ABSTRACT

Background Connect Care (CC) is Alberta's province-wide Epic-based electronic health record (EHR) system. Integrating Prevention into Connect Care for Health (IPiC-Health) aims to embed Screening, Brief Intervention, and Referral (SBIR) for tobacco use, alcohol use, and physical inactivity into CC-facilitated clinical workflows across Alberta Health Services (AHS). This intervention supports behavior change to improve cancer-related outcomes. IPiC-Health uses a quasi-experimental, mixed-methods design to assess implementation and effectiveness. Objective To describe how CC is leveraged through IPiC-Health to enable cancer prevention-oriented patient care and research. Methods SBIR was integrated into CC workflows using embedded tools and resources. CC-based standardized workbench reports and chart reviews were used to extract patient-level SBIR data into a centralized AHS SQL database. Patients identified at medium or high risk were flagged, and SQL views were created to monitor SBIR delivery in real time. Monthly feedback reports are shared with clinical sites. Eligible participants were recruited using CC data for follow-up at 1, 6, and 12 months to assess behavior change and health service use. Results SBIR has been implemented in four inpatient clinics and five ambulatory units. Over 7,000 patients have been screened, with 45% (n=3,601) categorized as medium or high risk. Of these, 14% (n=531) received brief intervention. Up to 11 feedback reports have been shared per site. So far, 53 participants completed 1-month follow-up, 66 at 6 months, and 22 participated in qualitative interviews. Conclusion CC enables real-time, prevention-oriented care and research, supporting a learning health system to advance cancer prevention in Alberta.

LAY SUMMARY

Integration of SBIR into Connect Care workflows enables real-time, prevention-focused care: Embedding Screening, Brief Intervention, and Referral within Alberta's EHR system allows proactive identification and management of cancer-related behavioral risk factors at the point of care. Connect Care supports both clinical care and research through structured data and feedback loops: Standardized data extraction and monthly reporting enable continuous quality improvement and foster a learning health system. IPiC-Health demonstrates the feasibility and early impact of EHR-enabled cancer prevention strategies: With thousands of patients screened and hundreds receiving intervention, the project highlights how EHR integration can scale cancer prevention efforts across health systems.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Mapping the Immunoepitome at Population Scale - Poster #36

Presenter: Aleksander Palkowski, Arnie Charbonneau Cancer Institute, University of Calgary

Other Authors: Michal Waleron, Javier Alfaro

ABSTRACT_ID#3131

ABSTRACT

The efficacy of cancer immunotherapies and vaccines across diverse human populations hinges on understanding population-specific variations in major histocompatibility complex (MHC)-driven antigen presentation. As MHC class I allele frequencies differ significantly between population groups, so too may their collective immunoepitomes. This study investigates the extent of these differences by comparing antigen presentation profiles across 21 global populations sourced from the National Marrow Donor Program (NMDP) haplotype frequency database. We employed a dual strategy, combining predictive modelling of 3 million randomly sampled peptides with NetMHCpan 4.1 against population-specific haplotypes, and an empirical analysis of physically-detected peptides included in 1,467 samples. Our predictive models suggest a substantial shared immunoepitome, with an average of 35% of peptides being presented between any two individuals, irrespective of their population of origin. Analysis of experimentally-verified peptides corroborates this broad similarity, revealing that most populations occupy overlapping territories on a shared MHC motif landscape. However, we identified two significantly distinct groups in terms of haplotype frequencies, with East Asian populations forming a distinct cluster in their antigen presentation patterns. Critically, our analysis uncovered a profound sampling bias within current public immunoepitomic datasets, which are overwhelmingly representative of European Caucasian haplotypes. These findings suggest that while a core set of promiscuous antigens may be viable for broad-coverage vaccines, population-specific differences must be considered for global efficacy. We conclude there is an urgent need to diversify immunoepitomics research to ensure the development of equitable and effective cancer therapies for all populations.

LAY SUMMARY

People's immune systems are more similar than expected: By analyzing how different populations present cancer "fingerprints" to the immune system, we found a surprising degree of similarity. Despite global diversity, any two individuals share about one-third of these on average, which is promising for the development of widely effective cancer vaccines and other immunotherapies. Some key differences exist: Despite the similarities, the analysis found that people of East Asian descent have a different pattern of immune presentation. This suggests that a "one-size-fits-all" approach to vaccines may not be equally effective for everyone, and population-specific differences need to be considered. Scientific data is heavily biased: The public data used for this type of research is overwhelmingly based on people of European Caucasian descent. This is a major blind spot that could lead to less effective treatments for other populations. To create truly global cancer therapies, research must urgently become more diverse.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Optimizing Experimental Protocols for High-Fidelity Cancer Cell Classification via Single-Cell Vibrational Profiling - Poster #38

Presenter: Ali Al-Khaz'Aly, Cumming school of Medicine/University Of Calgary

Other Authors: Ali Al-Khaz'Aly, Matthias Amrein, Jared Topham

ABSTRACT_ID#3205

ABSTRACT

Cellular vibrations are nanoscale mechanical oscillations generated by metabolic activity and cytoskeletal dynamics that serve as promising biophysical markers for cellular and cancer diagnostics. This study establishes a standardized framework for Cell Vibrational Profiling (CVP) using optical tweezers and a novel statistical pipeline, the Vibration Scanner Analysis Suite (VSAS), to systematically evaluate how experimental conditions influence vibrational signatures and classification accuracy. The VSAS framework employs Partial Least Squares Discriminant Analysis (PLS-DA) to process high-dimensional vibrational data (100 Hz–50 kHz), with permutation testing ($p < 0.001$) and 10-fold cross-validation confirming model robustness. Three critical parameters were investigated using U251 glioblastoma and A549 lung adenocarcinoma cells: culture environment, cell cycle synchronization, and media viscosity. Results demonstrated that petri-dish cultures minimized hydrodynamic noise, achieving superior differentiation ($F1 = 0.89$) compared to microfluidic chambers ($F1 = 0.79$). Cell cycle synchronization via serum deprivation reduced intra-class variance by standardizing metabolic activity, enhancing PLS-DA classification ($F1 = 0.82$). Media viscosity modifications using agarose showed only minor classification improvements ($F1 = 0.80$). This work highlights the critical role of experimental standardization in CVP by linking culture conditions and synchronization protocols to classification performance. The VSAS achieved 92% recall in distinguishing U251 from A549 cells under optimized conditions, demonstrating viability for real-time intraoperative diagnostics. These advancements address key barriers to clinical adoption, positioning CVP as a transformative tool for cancer detection and surgical applications while reducing reliance on invasive histopathological methods.

LAY SUMMARY

The study introduces a standardized Cell Vibrational Profiling (CVP) framework using optical tweezers and the Vibration Scanner Analysis Suite which through advanced statistical analysis reliably distinguishes between cancer cell types by optimizing experimental conditions such as culture environment and cell cycle synchronization. Results show that petri-dish cultures and synchronized cell cycles significantly improve classification up to $F1$ of 0.89 and 92% recall while changes in media viscosity offer only minor benefits highlighting the importance of experimental standardization for reproducible vibrational profiling. This work not only advances laboratory techniques, but demonstrates how a data driven statistical analysis can reliably be used to improve experimental protocols. These advancements position CVP as a promising less invasive alternative to traditional histopathology for real-time cancer diagnostics and surgical applications.

Immunosenescence Characterises Failure of Bacillus Calmette Guérin Therapy in Patients with Bladder Cancer - Poster #39

Presenter: Alexandra Robins, Queen's University

Other Authors: Andrew Garven, Jean-François Paré, Ana Vera-Rodríguez, Rohan Sampy, Richard Nauman, Madhuri Koti, Lynne-Marie Postovit, Andrew Craig, Peter Greer, Robert Siemens, Tiziana Cotechini, David Berman, Amber Simpson, Charles Graham

ABSTRACT_ID#3207

ABSTRACT

Introduction: Following tumour resection, the standard-of-care treatment for high-risk non-muscle invasive bladder cancer (NMIBC) for over three decades has been Bacillus Calmette-Guérin (BCG) immunotherapy. Despite the well-established clinical benefit of BCG, up to 50% of patients suffer disease recurrence or progression to muscle-invasive cancer. Yet, the immunologic mechanisms underlying BCG responsiveness remain poorly understood, highlighting the need to identify pre-existing immune states that predispose patients to treatment failure. **Methods:** Peripheral blood mononuclear cells (PBMCs) were collected from 16 patients with intermediate- or high-risk NMIBC prior to BCG immunotherapy and after five weekly instillations of BCG during induction immunotherapy (n = 32 samples). Single-cell RNA and assay for transposase accessible chromatin (ATAC) sequencing were performed to assess transcriptomic and chromatin accessibility changes across immune cell populations. Pathway enrichment and differential expression analyses were used to identify immune signatures associated with BCG response. **Results:** Single-cell analysis revealed that circulating monocytes and NK cells from BCG non-responders exhibited pre-existing activation of inflammatory and metabolic pathways consistent with immunosenescence. In contrast, BCG responders showed immune quiescence prior to treatment and robust activation of immune and metabolic pathways following BCG therapy. These transcriptional differences between BCG responders and non-responders were accompanied by chromatin accessibility changes and validated in an independent bulk RNA-seq cohort. **Conclusion:** Pre-existing immune dysfunction, marked by inflammatory and metabolic dysregulation, impairs the response to BCG immunotherapy in NMIBC. Peripheral immune profiling prior to therapy may serve as a non-invasive strategy to predict treatment outcomes and guide personalized therapeutic approaches.

LAY SUMMARY

1. **Baseline Inflammation Predicts Treatment Success** Before receiving BCG therapy, some patients already show signs of an overworked immune system, marked by chronic inflammation and metabolic stress. This state, known as immunosenescence, may prevent their immune cells from mounting an effective response to BCG, contributing to treatment failure.
2. **Successful BCG Response Requires Activation of Immune and Metabolic Pathways** Patients who benefit from BCG therapy tend to have an immune system in a quiescent state before treatment. Once BCG is administered, their immune cells respond robustly, activating genes and pathways linked to inflammation and metabolism. This suggests that timing and immune readiness are critical for BCG to work effectively.
3. **Blood-Based Immune Profiling Could Lead to Personalized Bladder Cancer Treatment** This study shows that analyzing immune cells in the blood, before and after BCG therapy, could help identify who is most likely to respond. In the future, this type of testing may allow clinicians to tailor treatment plans, avoiding delays in care and sparing patients from ineffective therapy.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Machine Learning Approach to Extracting Symptoms Information from Oncology Clinical Narratives - Poster #40

Presenter: Cheligeer Cheligeer, Alberta Health Services

Other Authors: Grace Heemeryck, Maeve Wickham, Kenneth Lai, Ali Manouchehrinia, Linda Watson, Emily Walker

ABSTRACT_ID#2433

ABSTRACT

Background: Anxiety and other patient-reported outcomes are key indicators of patient complexity. Although EMR reporting has improved, historical structured data remains incomplete, with symptoms often recorded only in unstructured notes. This limits predictive analytics for complexity and resource planning, which rely on complete longitudinal data. We developed a machine learning (ML) system to extract symptoms from clinical notes. Methods: We extracted EMRs data from 36,370 incident breast cancer patients treated in Alberta (2010-2022). Existing structured anxiety scores were first retrieved. To identify additional unstructured mentions, we used pattern matching to flag notes potentially referencing anxiety. A large language model (LLM) then labeled these as anxiety present, absent, or not discussed. These weakly labeled notes were used to train a neural network, which was evaluated against a manually reviewed validation set. Results: Of 36,370 patients, 20,765 (57.1%) had structured anxiety data, with 15,197 (73.2%) reporting non-zero anxiety scores. For the rest, the LLM (Phi4:14b) reviewed 40,100 flagged notes, labeling 28,971 (72.2%) as present, 1,213 (3.0%) as absent, and 9,916 (24.7%) as not discussed. The trained neural network achieved 83.0% sensitivity (95% CI: 77.3–87.9), 83.4% PPV (95% CI: 78.0–88.7), and an AUC-ROC of 93.5 (95% CI: 91.1–95.6). The framework identified 15,605 additional patients (42.9%) with unstructured anxiety mentions absent from structured data. Conclusions: This ML approach successfully identifies anxiety symptoms reported in clinical notes, generating robust structured data for complexity measurement. The method is generalizable to other extraction tasks, supporting scalable transformation of clinical narratives into structured data.

LAY SUMMARY

In Alberta, many breast cancer patients experience anxiety, but nearly half have no formal record of it in structured medical data. We built a system that reads doctors' notes to uncover signs of anxiety that might otherwise go unnoticed. The system uses a large language model (similar to tools like ChatGPT, Claude, and Grok) to review clinical notes and determine whether anxiety is present, absent, or not mentioned. The system then learns (artificial neural networks) from these findings to detect signs of anxiety directly from the notes. The system finally identified over 15,000 additional patients showing signs of anxiety that were not captured in structured records. This approach helps make the invisible visible, giving care teams a more complete picture of patients' emotional well-being. The same system can be adapted to find other symptoms or concerns—like pain or depression—supporting more patient-centered cancer care.

Machine Learning-Based Radiomics for Predicting Tumor Progression in Metastatic NSCLC Under Immune Checkpoint Inhibitor Therapy - Poster #41

Presenter: Alexander Iwasyk, University of Calgary

Other Authors: Reza Kakavand, Joe Kendal, Annalise Abbott, Hila Yashar, Peter Connell, Michael Monument, Brent Edwards

ABSTRACT_ID#3025

ABSTRACT

Background: Non-small cell lung cancer (NSCLC) is a leading cause of cancer-related mortality, with metastatic cases presenting treatment challenges due to disease heterogeneity. Immune checkpoint inhibitors (ICIs) have improved patient survival; however, assessing bone lesions is particularly difficult due to poor sensitivity of RECIST1.1, the standard for measuring tumor response. Machine learning may offer a standardized and quantitative approach to assessing treatment response using medical imaging. Methods: In this retrospective study, bone metastases from 104 NSCLC patients were 3D modeled, and radiomic features such as tumor size, shape, and texture were extracted from pre- and post-treatment imaging using PyRadiomics. Features were grouped into five texture-based categories and combined with clinical variables including sex, age, BMI, and radiotherapy status to train a Random Forest model to predict treatment response. Model performance was assessed using five-fold cross-validation and compared to radiologist assessments using MD Anderson criteria. Results: Post-treatment radiomics outperformed pre-treatment radiomics for predicting tumor response. Among clinical variables, sex and BMI improved model accuracy. The sex-augmented model achieved the highest performance: F1-score: 0.772 ± 0.098 , AUC: 0.923 ± 0.059 , sensitivity: 0.781 ± 0.098 , and specificity: 0.807 ± 0.067 . Radiologist agreement showed strong concordance (kappa statistic > 0.9). Conclusion: A limitation is the absence of a comparative control group not receiving ICIs. Post-treatment radiomics combined with clinical variables such as sex, enhance tumor response prediction, though radiologist interpretations remained superior, underscoring the need for larger training datasets.

LAY SUMMARY

1. RECIST1.1 is the current standard for assessing tumor response, but it performs poorly for bone metastases due to its reliance on simple linear measurements. This approach often misses the complex structural changes seen in bone lesions. In contrast, radiomics may provide a comprehensive evaluation of the tumor architecture by measuring shape- and texture-based features, enabling a more detailed and objective assessment of tumor response over time, especially in hard-to-measure metastatic sites.
2. A random forest is a type of machine learning model that combines many decision trees to make accurate predictions. This study utilized this model to predict tumor response using radiomics features. The model performed best when using post-treatment images and patient characteristics like sex and BMI. However, radiologist assessment remained superior, highlighting limitations of the model, such as a small training dataset or potentially the heterogeneity of metastatic disease.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Multi-Cohort Ensemble Learning Framework for Vaginal Microbiome-Based Endometrial Cancer Detection - Poster #42

Presenter: Dollina Dodani, University of British Columbia

Other Authors: Aline Talhouk

ABSTRACT_ID#3095

ABSTRACT

The vaginal microbiome has emerged as a promising source of non-invasive biomarkers for gynecologic cancers, but inconsistent bioinformatics and limited reproducibility have impeded clinical translation. Endometrial cancer is the second most common gynecological cancer worldwide, yet there are no established screening programs. A vaginal microbiome signature derived from minimally invasive testing could assist in early detection. We conducted a multicohort analysis using publicly available 16S rRNA gene sequencing data from five studies (N=265) to assess the predictive value of vaginal microbiome profiles for endometrial cancer. After benchmarking bioinformatics pipelines to ensure the reproducibility of commonly reported metrics, such as alpha/beta diversity and differentially abundant taxonomic features, we selected a robust preprocessing approach. We then developed an ensemble classifier that integrates microbial features with individual characteristics, including age, body mass index (BMI), and ethnicity. The combined model accurately identified all individuals with endometrial cancer in an independent validation cohort, achieving an AUROC of 0.93 (95% CI: 0.71–0.93), negative predictive value of 1.0 (95% CI: 0.59–1.0), and sensitivity of 1.0 (95% CI: 0.74–1.0). Alpha diversity was sensitive to processing pipelines, while beta diversity patterns were driven by patient factors than disease status. Certain taxa, including *Peptoniphilus*, showed consistent association with endometrial cancer cases across all five studies. This work demonstrates the feasibility of using vaginal microbiome data, coupled with individual characteristics, for EC screening and risk stratification. Our open-source pipeline and rigorous evaluation support its utility as a reproducible, non-invasive tool in precision oncology.

LAY SUMMARY

In this study, we present the development of an ensemble AI classifier for detection of endometrial cancer by utilizing vaginal microbiome profiles from five independent cohorts. Specifically, We replicated previously reported microbial associations with endometrial cancer, such as an increase in microbial diversity among malignant individuals compared to those with benign conditions. Using a harmonized bioinformatics pipeline, we identified various species of *Peptoniphilus* associated with health conditions across all five datasets. Our resulting ensemble classifier, trained on minimally invasive vaginal samples, integrates microbiome features with host characteristics, including age, BMI, and ethnicity. This model demonstrated high predictive performance and generalizability in a held-out test set with an AUROC of 0.93 (95% CI: 0.71-0.93), sensitivity of 1.0 (95% CI: 0.74-1.0), and a negative predictive value of 1.0 (95% CI: 0.59-1.0). These findings support the potential use of vaginal microbiome profiling as a minimally invasive approach for early detection of endometrial cancer.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

LEI: Large Language Model-based Extraction of Information for the Ontario Hereditary Cancer Research Network - Poster #43

Presenter: Jochen Weile, Ontario Institute for Cancer Research

Other Authors: Andres Melani, Pratham Hemlani, Elif Tuzlali, Brandon Chan, Harriet Feilotter, Lauren Hughes, Raymond Kim, Sarah Ridd, Lincoln Stein, Kathy Chun, Benjamin Haibe-Kains, Melanie Courtot

ABSTRACT_ID#3345

ABSTRACT

Large-scale clinical databases are essential for healthcare operations, research and decision-making. However, most clinical information is found in unstructured sources from which relevant data must be extracted. This extraction is done manually, which is time-consuming, expensive, and prone to errors. Such is the case of the Ontario Hereditary Cancer Research Network (OHCRN), which manually processes molecular test reports from multiple laboratories to establish a unified registry for individuals with hereditary cancer syndromes in Ontario. By centralizing germline genomic data and making them accessible to researchers, OHCRN would facilitate cancer research and help connect individuals with clinical trials and support groups. Yet, extracting data from reports is challenging since each laboratory handles its own format and shares reports as scanned documents. Automated tools, especially Large Language Models (LLMs), can address these issues. We propose LLM-based Extraction of Information (LEI), an automated data extraction pipeline that extracts relevant data from molecular lab reports and structures it for loading into the OHCRN database. LEI was evaluated by measuring the F1-score on the extraction of eighteen entity types, with the results validated by expert clinical data curators, and comparing this measure against fifteen extraction tools in the biomedical domain. Extraction times were also measured and compared against manual extraction times. LEI demonstrated quality on par with and surpassing other existing LLM-based extraction methods, while reducing extraction time by approximately 20-fold. LEI's accurate and fast clinical data extraction from unstructured texts will support OHCRN's mission of unifying and making data accessible for hereditary cancer research.

LAY SUMMARY

LEI demonstrated an overall accuracy exceeding 80% and achieved over 20-fold time savings in healthcare data extraction from unstructured texts. Results indicate that LEI facilitates efficient and accurate data extraction, aiding OHCRN experts in improving lab report processing. LEI has the potential to be adapted for use in other biomedical contexts that require data extraction from unstructured sources.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Identifying Novel Cancer Genes Through Noncoding Mutation Burden in Regulatory Elements - Poster #44

Presenter: Eftyhios Kirbizakis, McGill University

Other Authors: Swneke Bailey

ABSTRACT_ID#3187

ABSTRACT

Somatic mutations in the noncoding genome are abundant in cancer but remain poorly understood due to the limited interpretability of regulatory DNA. While recurrence has been a useful signal in identifying protein-coding drivers, this strategy often fails in noncoding regions, where functional annotations and mutation mechanisms are less well defined. We present an alternative framework to identify putative noncoding drivers by assessing aggregated mutation burden across cis-regulatory elements, specifically, enhancers and promoters involved in chromatin loops. Using in-house whole-genome sequencing data from esophageal adenocarcinoma cancer patients and H3K27ac HiChIP to define active enhancer–promoter interactions, we analyze the somatic mutational landscape within structurally linked regulatory elements. Rather than relying on single-site recurrence, we test whether functionally grouped regions interacting with a given gene show an enrichment of mutations across patients. This approach reframes recurrence in the noncoding genome, enabling the identification of novel candidate cancer genes affected through regulatory disruption and offers a strategy for mapping noncoding mutation burden within the functional 3D genome and has the potential to uncover overlooked regulatory drivers of oncogenesis.

LAY SUMMARY

- Most cancer research focuses on mutations in genes, but we're studying mutations in the noncoding genome, the parts that don't make proteins but influence how genes are expressed.
- Traditional methods look for the exact same mutation appearing in many patients. Instead, we look for whether the regulatory regions linked to a gene—like enhancers and promoters—are repeatedly mutated across patients, even if the exact mutation varies.
- By focusing on regions that physically interact in 3D, we aim to find new cancer-related genes that may be overlooked by traditional methods.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Enhancing a Breast Cancer (BC) Learning Health System (LHS) Through Data Integration - Poster #46

Presenter: Andrea Eisen, McMaster University

Other Authors: Jonathan Ranisau, Ashirbani Saha, Mark Levine, Angelo Carreon, Joel Kemppainen, Chaojun Chen, Francesca D'Angelo, Daria Grafodatskaya, Jeremy Petch

ABSTRACT_ID#3172

ABSTRACT

Background: We developed a prospective, patient-level data and analytics LHS platform that captures comprehensive clinical data (141 elements) from the electronic medical record (EMR) of over 7000 BC patients seen at a regional cancer centre (RCC) between 2014 and 2022. Data warehousing and natural language processing (NLP) are combined to support quality improvement and research. The RCC adopted a new EMR, Epic, in 2022. We now report on methodological enhancements and challenges encountered in adapting the LHS to Epic, including the integration of genetic testing (GT) and analysis of neoadjuvant chemotherapy (NAC). **Methods:** Data scientists and clinicians collaborated using BC treatment pathways as a framework. Structured data elements were extracted from Epic; when these were unavailable, NLP techniques supplemented data extraction from clinical records. Both were linked at the individual patient level and analyzed by descriptive statistics for face-validity (for NAC) and feasibility (for GT). **Results:** The updated LHS cohort includes 10,000 BC patients. Extracting individual GT test results using NLP techniques is feasible. Relying on clinicians to enter stage and treatment plan data in EPIC leads to missing information: we have found alternative linkage methods to address this. We are validating our preliminary findings regarding the use of NAC. **Significance:** This proposal demonstrates a scalable approach for updating an LHS thereby enhancing the clinical utility of routinely collected cancer data through NLP-driven feature extraction. Future work will include breast reconstruction data, additional institutions and linkage to provincial administrative databases to broaden the LHS's research scope and utility.

LAY SUMMARY

Our team built a Breast Cancer Learning Health system, which is a platform that collects detailed medical information on breast cancer patients from hospital records so we can assess the quality of care they receive. It has now expanded to include 10,000 patients and works with both older and newer electronic systems (like Epic), and includes information about genetic testing and chemotherapy given before surgery. When certain details are missing, the team uses natural language processing (a type of AI that reads written reports) to find and extract important facts. The system is designed to be expanded to other hospitals and linked with larger health databases. This makes it a powerful tool for improving care, and supporting research across the healthcare system.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Towards a Quantitative Phenotypic Progression Model of Prostate Cancer - Poster #47

Presenter: Fumiya Inaba, BC Cancer Research Institute

Other Authors: Willie Wu, Mira Keyes, Calum MacAulay, Martial Guillaud

ABSTRACT_ID#3227

ABSTRACT

Robust prognostic systems are a crucial element of prostate cancer (PCa) management. Despite recent clinical advancements, precise prognostic systems with widespread availability and -omics level granularity remain a challenge. We propose a quantitative phenotypic progression model of prostate cancer analyzing both tissue architecture and cell morphometry, which may be used to complement the current PCa grading system. Our system analyzes large-scale DNA organization (LDO) of cell nuclei on biopsies stained with Feulgen-thionin, a DNA-stoichiometric dye which is scanned and analyzed by our in-house pipeline. Architecture analysis computes 50 features per biopsy, and LDO analysis leverages a multiple instance learning paradigm to generate a score for each patient from 140 features. A cohort of PCa patients (n=335) from the Prostate Cancer Centre are studied. There are loosely three groups of patients relevant to our study: patients with Gleason score (GS) 6 with no signs of progression (n=34), patients with GS9 deceased within 2 years of consultation (n=28) and patients who have received radical therapy (n=273). For training purposes, 75% of GS6 and GS9 patients are assigned to the training group (n=48) and the remaining 25% to the test group. Patients who received radical therapy are treated as an independent validation group. On the test set, architectural and LDO analysis achieves 90% and 86% accuracy respectively. LDO and architectural scores are calculated for remaining patients. We will further discuss the potential of this quantitative phenotype space, potentially characterizing a quantitative signature for progressive PCa.

LAY SUMMARY

Quantitative analysis of tissue architecture is prognostic of aggressive prostate cancer Quantitative analysis of DNA organization within cell nuclei is prognostic of aggressive prostate cancer Analysis of tissue architecture and large-scale DNA organization can create a quantitative phenotype which may be used as "signatures" to identify patients at higher risk for aggressive prostate cancer.

Monday, November 3, 2025

High Resolution Spatial Analyses Identifies Immunotherapy Resistance Mechanisms to CAR T-Cell Therapy in ASPS - Poster #48

Presenter: Gurveer Gill, University of Calgary

Other Authors: Varsha Thoppey Manoharan, Hongrui Lui, Yeon Suh, Bo-Young Ahn, Laura Mah, Michael Monument, Ramy Saleh, Jan-Willem Henning, Mona Shafey, Jennifer Chan, Franz J. Zemp, Douglas J. Mahoney, Sorana Morrissy

ABSTRACT_ID#2765

ABSTRACT

Alveolar Soft Part Sarcoma (ASPS) is an ultra-rare, fusion-driven sarcoma affecting adolescents and young adults. Emerging evidence suggests that ASPS is responsive to immunotherapy, and immune checkpoint inhibitors are now approved in Canada for use in these patients. To advance precision immunotherapy in this disease, our team developed a Chimeric Antigen Receptor (CAR) T cell therapy targeting Glycoprotein-NMB (GCAR1) and tested it in a stage IV ASPS patient. At a GCAR1 dose of 1e6 CAR T-cells/kg, we observed a profound reduction in the size and abundance of metastatic lung lesions in the absence of CRS/ICANs, with the patient achieving RECIST1.1 stable disease for over 6 months. To deeply explore the tumour microenvironment (TME) of ASPS and the spatial variables influencing immunotherapy efficacy in the GCAR1 clinical setting, we used the Visium HD technology to generate transcriptome-wide profiling data at 2um resolution. We profiled three clinical samples from the GCAR1-treated patient, including a primary tumour resection and two biopsy cores taken on-trial at day 35 from a non-responding metastatic lesion. Unsupervised deconvolution (cNMF) identified gene expression programs representing cell types and states of the immune, tumour, and stromal compartments. We found abundant cytotoxic T-cells in the treatment-refractory metastatic lesion, significant co-localization with tumor cells, and strong upregulation of the PD1-PDL1 signalling axis driven by tumor cells. Our results demonstrate that metastatic ASPS tumour cells upregulate PDL1 expression to inhibit T-cell function, suggesting that adjuvant checkpoint inhibitors may be effective in this setting.

LAY SUMMARY

Alveolar Soft Part Sarcoma (ASPS) is a rare and fatal cancer affecting children and young adults. One approach to treating ASPS involves immune checkpoint inhibitors (ICIs), which release natural "brakes" on T-cells – immune cells that can attack cancer – but only partially works in a third of patients. Our team developed a synthetic receptor, GCAR1, that teaches a patient's own T-cells to target and eliminate ASPS cells. We tested our therapy in a patient with metastatic ASPS and observed a reduction in metastases, with no cancer progression for over six months. We studied regions of the patient's tumor that did not respond to our treatment and found that the ASPS cells were engaging natural T-cell brakes. This finding suggests that combining GCAR1 therapy with ICIs could improve outcomes for ASPS patients.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Using Natural Language Processing to Create a Virtual Tumor Repository for Cancer Research - Poster #49

Presenter: Jean Claude Bigenimana, Provincial Health Services Authority

Other Authors: Umaimah Zanif, Parveen Bhatti, Jonathan Simkin, Lovedeep Gondara

ABSTRACT_ID#2974

ABSTRACT

Background The British Columbia Generations Project (BCGP), a cohort of ~30,000 people recruited from BC to support research into cancer prevention and early detection, established a virtual tumor tissue repository (VTR). The repository consisted of data but with new cancers being diagnosed among BCGP participants each year, manual abstraction of data from pathology reports is not efficient for updating the VTR. **Purpose** The BC Cancer Registry (BCCR) receives all cancer pathology reports in electronic format. We developed an automated pipeline, using NLP, to extract the key data elements from the pathology reports needed by BCGP to support the VTR. **Methods** We applied a three-tiered automated approach combining regular expressions, large language models (LLM) and fine-tuned transformer-based language models to extract patient identifiers, specimen case/accession number, pathology laboratory, hospital name, specimen type, specimen site, agent type (e.g., formalin), tumor grade, Gleason score, and formalin-fixed paraffin-embedded block from pathology reports. A set of 864 pathology reports (12% of 6,728) were manually annotated to create training data for the models. **Results** The proposed automated process achieved accuracies ranging from 92%-99% on all extracted data elements. The residual errors are attributed to the nature of the raw text where a given data field can be described in multiple different ways. The extracted data is stored in a structured tabular format for easier analysis. **Conclusion** Automated extraction approach reduces time, errors, human effort, and handles large volume of data. Additionally, it will enable researchers to easily find, and access tumor specimens needed for various studies.

LAY SUMMARY

We built an automated process using NLP with Advanced AI models (Large Language Models-LLM) that extract targeted data elements from electronic pathology reports, saving lots of time and effort compared to do it manually. Our system used mixed method including regular expressions for patterns matching, large language models (LLMs) for contextual understanding, and fine-tuned transformer-based machine learning models for domain-specific task to accurately extract clinical data such as patient identifiers, specimen case/accession number, pathology laboratory, hospital name, specimen type, specimen site, agent type, tumor grade, Gleason score, and formalin-fixed paraffin-embedded block from pathology reports with about 92–99% accuracy. This approach significantly reduces time, manual workload, and risk of error in clinical data collection, enabling scalable processing of thousands of pathology reports, supporting researchers in accessing needed data more easily and facilitating data management efforts.

Enhancing Early Lung Cancer Diagnosis via Deep Learning and Radiomics for Sub-Centimeter Nodule Classification in LDCT Screening Programs - Poster #52

Presenter: Ian Janzen, BC Cancer Research Institute

Other Authors: Rohan Abraham, Sukhinder Khattri, Renelle Myers, Ren Yuan, Stephen Lam, Calum MacAulay

ABSTRACT_ID#2827

ABSTRACT

Lung cancer remains the leading cause of cancer-related mortality in Canada, with early detection via Low-Dose Computed Tomography (LDCT) screening programs shown to significantly reduce mortality. However, the identification and classification of small lung nodules (diameter ≤ 11 mm) presents challenges due to their limited pixel-based information and the high volume detected during screenings. To address these issues, we developed a computer-aided diagnosis (CADx) pipeline integrating deep learning segmentation with radiomics-based machine learning for identified nodules. Using data from the Pan Canadian Screening Study (PanCan, N=6,944 nodules, 136 cancers), a self-attention UNet was employed for 2D axial segmentation of each nodule, generating three distinct masks: nodule core, core plus edge parenchymal tissues, and peritumoral region. Radiomic features were extracted from these masks and refined through feature selection algorithms to create a highly discriminating signature. This radiomic signature was used to train a logistic regression (LR) model for binary classification of cancerous vs benign nodules. Cross-validation yielded an AUC of 0.970 (sensitivity: 0.911, specificity: 0.951). The model identified 12 cancerous nodules overlooked by the Brock model score ($\leq 5\%$) and predicted malignancy up-to 24 months pre-clinical confirmation. A validation cohort from the International Lung Screening Trial (ILST, N=2,943 nodules, 36 cancers), is currently under analysis to assess generalizability and medical utility. This approach demonstrates that integrating baseline LDCT radiomics with deep learning segmentation can enhance sub-centimeter lung cancer identification, improving decision-making in screening programs. Pending validation on the ILST dataset, this method shows promise for optimizing treatment protocols in lung screening programs.

LAY SUMMARY

This study shows the effectiveness of integrating deep learning segmentation with radiomic features for identifying small, difficult-to-classify lung nodules (≤ 11 mm) detected in LDCT lung screening programs. The developed methods achieved excellent performance metrics (AUC=0.970, sensitivity=0.911, specificity=0.951) and successfully identified cancerous nodules up to 24 months before clinical confirmation, potentially enabling earlier interventions and better patient outcomes. The prediction model identified 12 cancerous sub-centimeter nodules that conventional screening methods (e.g., the Brock model) would have misclassified as benign during the baseline screening scan, while still maintaining low false positive rates.

Multi-Transcriptomic Analysis of Double-Negative T Cells Unveils Prognostic Subsets in Head and Neck Squamous Cell Carcinoma - Poster #53

Presenter: Jinsu An, University of Calgary | Arnie Charbonneau Cancer Institute

Other Authors: Ilana Lebon, Beum-Keun Cho, Chung-Gyu Park, Minsu Kim, Jongbok Lee, Pinaki Bose

ABSTRACT_ID#2417

ABSTRACT

Introduction: HPV-Negative head and neck squamous cell carcinoma (HNSCC) is associated with high morbidity and mortality, with nearly half of patients presenting with nodal metastasis at diagnosis. An immunosuppressive tumor microenvironment, driven by aberrant cytokine signaling and impaired immune effector function underscores the importance of the tumor-immune microenvironment (TIME) in disease progression. Here, we report the first identification and functional characterization of distinct double-negative T cell (DNT; CD3⁺CD4⁺CD8⁺) subsets within HNSCC TIME. Methods & Results: We integrated single-cell RNA-sequencing (scRNAseq) data comprising 135,319 cells from 47 patients across five publicly available datasets of primary, treatment-naïve HPV-negative HNSCC. Our analysis revealed five unique DNT clusters, including two functionally distinct subsets: FOXP3⁺ regulatory DNTs (DNTregs) and GZMB⁺ cytotoxic DNTs. Functional assays confirmed these findings: CD25⁺FOXP3⁺ DNTs suppressed CD8⁺ T cell proliferation, while ex vivo-expanded DNTs exhibited robust cytotoxicity against HNSCC cell lines. Analysis of bulk RNA-seq data from TCGA (n=235) revealed that patients with high expression of the regulatory DNT signature were associated with shorter progression-free interval (p=0.041) and worse disease-specific survival (p=0.035). In contrast, high expression of the cytotoxic DNT signature correlated with improved outcomes (p=0.001). Spatial transcriptomic analysis of 12 in-house samples revealed distinct localization of DNT subsets: GZMB⁺ cytotoxic DNTs were enriched in the tumor leading-edge alongside cytotoxic CD8⁺ T cells, whereas FOXP3⁺ DNTregs were concentrated within the tumor core, often near myeloid cells and CD4⁺ Tregs. Conclusion: Our study reveals the immune-regulatory functions of distinct DNT subsets, uncovering their previously unrecognized roles in HNSCC pathogenesis and progression

LAY SUMMARY

- **Rare, Understudied Immune Cells Matter:** We focused on a rare and understudied type of immune cell called “double-negative T cells” (DNTs), which play important but previously unclear roles in head and neck cancers.
- **Two Types, Two Outcomes:** We found that some DNTs can suppress the immune system and are linked to worse patient outcomes, while others are more effective at killing cancer cells and are linked to better outcomes.
- **Location in the Tumor Is Important:** The cancer-fighting DNTs tend to be at the edge of tumors, while the suppressive DNTs are found deeper inside, possibly helping tumors evade the immune system.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

openmpp: An R package for dynamic microsimulation modelling using OpenM++ - Poster #54

Presenter: John M. Hutchinson, University of Calgary

Other Authors: Matthew T Warkentin, Yibing Ruan, Darren R Brenner

ABSTRACT_ID#2513

ABSTRACT

Background: Microsimulation is a computational technique used extensively in public health, epidemiology, and oncology to simulate life histories based on individual-level characteristics to estimate population-level outcomes and inform evidence-based decision-making. OpenM++ is an open-source platform and domain-specific language to build and analyze complex microsimulation models. OpenM++ is widely used in academia and government agencies, including within the OncoSim family of cancer models. OpenM++ has an easy-to-use graphical interface but programmatic approaches offer greater transparency, reproducibility, and efficiency. **Objectives:** To address this challenge, we developed the openmpp R package, which provides a programmatic interface to the OpenM++ microsimulation platform. **Implementation:** openmpp wraps the OpenM++ Web Service API to provide OpenM++ users a programmatic interface for the R language. The package provides a suite of functions for easy communication with the OpenM++ API, including a set of R6 classes that enable real-time communication to ensure that changes made to microsimulation objects in the R session are synchronized with OpenM++. **Results:** The openmpp R package simplifies the process of creating and managing microsimulations, performing scalable and reproducible computations, and gathering results for further processing and reporting. Users interact with R objects in a familiar way while achieving real-time synchronization with external resources. Several studies on cancer have performed microsimulations using openmpp (PMID: 39943836, 39106352, 40178943). **Conclusion:** openmpp provides a simplified interface for the R language to perform microsimulations using OpenM++. openmpp has been published in the Journal of Open Source Software (JOSS) and can be installed from the Comprehensive R Archive Network (CRAN).

LAY SUMMARY

OpenM++ is a powerful open-source and cross-platform software and domain-specific language to develop, use, and deploy dynamic microsimulation or agent-based models. The openmpp R package provides a programmatic interface to the OpenM++ microsimulation platform to simplify the process of creating and managing microsimulations, performing scalable and reproducible computations, and gathering results for further processing and reporting. openmpp has been used for several cancer studies. It is published in the Journal of Open Source Software and available for researchers to install and use for their microsimulation studies.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

The Canadian Cancer Statistics Dashboard (CCSD) - Poster #56

Presenter: John M. Hutchinson, University of Calgary

Other Authors: Tamer N Jarada, Eliya Farah, Yanbo Liu, Matthew T Warkentin, Larry F Ellison, Elizabeth Holmes, Ryan R Woods, Ioana Nicolau, Donna Turner, Lorraine Shack, Natalie Fitzgerald, Monika Dixon, Jennifer Gillis, Alain A Demers, Darren R Brenner

ABSTRACT_ID#3009

ABSTRACT

Introduction: Cancer statistics play a critical role in epidemiological surveillance and healthcare resource planning. Historically, the Canadian Cancer Statistics (CCS) annual reports have served as the primary resource for disseminating cancer statistics in Canada. However, interactive dashboards can provide a complementary approach for exploring cancer data. Objectives: To develop the Canadian Cancer Statistics Dashboard (CCSD) and provide a user-friendly, publicly available, and up-to-date interactive data platform to increase the awareness and impact of cancer statistics in Canada. Methods: The CCSD is based on the Canadian Cancer Statistics report developed through a collaboration between the Canadian Cancer Society, Statistics Canada, and the Public Health Agency of Canada. We developed the CCSD using a custom code base, which allowed for customization without any changes to the source code. We populated the CCSD using the most recent data provided to the Canadian Cancer Registry by the provincial and territorial cancer registries. Results: The CCSD is available at cancerstats.ca in both English and French. The dashboard has been updated to include Economic Impact statistics to align with the 2024 Special Economic Report published by the CCS. The dashboard contains figures for trends in incidence, mortality, survival, impact, cost, and prevalence. Conclusion: The CCSD supports a shift toward interactive, customizable, and timely reporting of cancer data. This approach to data reporting has the potential to make cancer statistics more accessible and increase engagement. The CCSD will receive ongoing maintenance and updates as new data become available.

LAY SUMMARY

Up-to-date and easily accessible statistics are essential for cancer control in Canada. We have developed the Canadian Cancer Statistics Dashboard (CCSD) to disseminate the findings of the Canadian Cancer Statistics report using web-based, customizable charts and spreadsheets. By making these data available through a user-friendly website, members of the public can easily access and interpret cancer statistics in Canada. The CCSD is publicly available at cancerstats.ca in both English and French and includes data for incidence, survival, deaths and the overall economic cost of cancer.

Comparison of suggested first-line metastatic melanoma systemic therapy from publicly available artificial intelligence models versus medical oncologists - Poster #57

Presenter: Joanna Mycek, Arthur J.E. Child Comprehensive Cancer Centre

Other Authors: Jose Monzon, Omar Khan, Igor Stukalin, Meghan Mahoney, Tina Cheng

ABSTRACT_ID#3098

ABSTRACT

Artificial Intelligence (AI) tools, including large-language models (LLMs), have become ubiquitous, and are used by patients and providers in oncology. Three publicly-accessible LLMs (ChatGPT, DeepSeek and OpenEvidence) were asked to recommend the best first-line treatment for metastatic melanoma, using 10 hypothetical clinical cases. We compared these results with those of three medical oncologists specializing in cutaneous malignancies, blinded to each other's answers. Four prompt styles were used, containing multiple variables including AI role (patient vs oncologist), patient age, gender, autoimmune disease history, ECOG, BRAF and c-kit mutation status. Our primary outcome was concordance between the three LLMs; our secondary outcome was concordance of LLMs with the oncology majority opinion. All three LLMs agreed on a treatment recommendation 70% of the time, while all medical oncologists were in agreement 50% of the time. In all remaining cases, two of three oncologists agreed on a recommendation. Concordance with majority oncology opinion was 80% (ChatGPT), 70% (DeepSeek), and 80% (OpenEvidence). In 1/10 cases, LLMs suggested a treatment option not recommended by any oncologists. In cases with oncology consensus, LLMs provided the same recommendation 80% of the time. Altering AI role (patient vs oncologist) did not change proposed treatment. This pilot demonstrated LLMs provide treatment recommendations for metastatic melanoma, with reasonable consistency and accuracy. Given their public accessibility, providers should be prepared for the possibility of patients consulting these tools. We plan to use a real-world retrospective cohort to further test the performance metrics of publicly-available LLMs in the future.

LAY SUMMARY

- Large-language models provided treatment recommendations for metastatic melanoma with reasonable consistency and accuracy
- Concordance with majority oncology opinion was between 70-80%.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Developing an AI-Powered Assistant for Personalized Navigation in Cancer Care - Poster #58

Presenter: John-Jose Nunez, BC Cancer

Other Authors: Alan T. Bates

ABSTRACT_ID#2528

ABSTRACT

Patients with cancer often face difficulty navigating a complex system of supportive care services, including counseling, palliative care, and community support. Many are unaware of available options or unsure how to access them—leading to unmet needs, delayed interventions, and greater emotional, social, and physical burden. We are developing an AI-powered cancer care navigation assistant that builds on our previous work creating predictive models to estimate survival and psychosocial needs from oncology consultation notes. This project incorporates generative AI tooling to translate those predictions into clear, patient-friendly explanations and personalized recommendations. These are matched with a curated resource database and delivered through accessible channels such as SMS, email, web chat, or voice—helping patients identify and connect with appropriate resources. We will present an initial proof-of-concept version of the assistant, demonstrating how our predictive and generative AI models support real-time, personalized recommendation delivery. We will also share results from early testing and highlight technical design choices, such as explainability features to build patient trust and a modular system that supports future expansion. This work has been shaped by consultations with potential research users—including patients, clinicians, and system leaders—to identify where AI can most meaningfully improve the cancer care experience. The session will use interactive discussion to gather feedback on desired features, interface design, and integration into clinical workflows. By enabling timely access to supportive care, this AI navigation tool aims to improve outcomes, reduce healthcare system strain, and serve as a scalable model for AI-driven care navigation across oncology and beyond.

LAY SUMMARY

AI can help identify patient needs earlier. We've developed predictive AI models that analyze oncologists' notes to identify patients who may need extra support—including psychiatry and counselling services—early in their cancer journey. Combining Generative and Predictive AI can empower patients to navigate cancer resources. Using generative AI, we translate those predictions into easy-to-understand messages that help patients explore relevant resources—such as support groups, transportation help, or financial aid—at the moments they may find them most useful. We're building a navigation assistant to make this support more accessible. Our proof-of-concept AI assistant will deliver these personalized recommendations through familiar channels like text message, email, or chat, so patients can access support without needing to navigate a complex system on their own.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Uncovering patterns in splicing across B cell malignancies - Poster #59

Presenter: Krysta Coyle, Simon Fraser University

Other Authors: Laura Hilton, Kostiantyn Dreval, Callum Brown, David Scott, Ryan Morin

ABSTRACT_ID#3186

ABSTRACT

Cancers originating from mature B cells at various stages of differentiation represent a diverse set of cancers. There are numerous pathological subtypes, each comprising malignancies with distinct biology and clinical outcomes. Based on growing evidence for the role of splicing factors in mature B cell neoplasms, we hypothesize that regulation of splicing is essential for B cell differentiation and maturation. We expect that splicing aberrations in B cell malignancies are related to normal patterns of alternative splicing. We leveraged RNA-seq data from over 3100 tumours as part of our Genomic Analysis of Mature B-cell Lymphomas (GAMBL) project. We used additional RNA-seq data from normal tissues with distinct B-cell maturation states to identify splicing events which are differentially regulated throughout normal B cell maturation. Annotated and novel events were then used to query alternative splicing in a subset of tumor samples using a combination of Illumina-based and Oxford Nanopore sequencing. Our initial analysis identified a need for stringent quality control (QC). We established library size, 3' bias, and exon coverage cut-offs as QC metrics to determine sample inclusion in our study. Library preparation methods (polyA-selection versus ribosomal RNA depletion) contributed to significant batch effects. We are continuing to investigate correction methods to integrate additional data. In non-malignant samples, we identified 129 known and 267 novel splicing events which display differential regulation during B cell maturation and warrant further investigation within subtypes of B cell malignancies. Ultimately, we are pursuing alternative splicing as a therapeutic vulnerability common among mature B cell neoplasms.

LAY SUMMARY

Cancers of mature B cells can be very different, despite beginning in the same cell type. Patients with these cancers face many different clinical outcomes. RNA regulation is one way these cancers differ from each other. Alternative splicing is a method of RNA regulation that is important in how cells develop. Understanding how alternative splicing changes as B cells develop will help us find vulnerabilities in B cell cancers that we can exploit for new treatments.

Mitochondrial Dynamics and Pathway Activation Differ Between FL Subtypes and Predict Transformation to DLBCL - Poster #60

Presenter: Chun Hei Yiu, BC Cancer Research Centre

Other Authors: Jacky Chun Hei Yiu, Glenn Chang, Andrew Roth, Andrew Weng, Adi Steif

ABSTRACT_ID#2505

ABSTRACT

Follicular lymphoma (FL) is a slow-growing non-Hodgkin lymphoma, with a 2-3% annual risk of transformation into aggressive disease, predominantly diffuse large B-cell lymphoma (DLBCL). Recent studies identified two molecular subtypes of FL—germinal center-like (GC-like FL) and memory-like (Mem-like FL), with the latter associated with a higher risk of transformation. Prior studies also showed substantial increases in oxidative phosphorylation (OXPHOS) pathway expression upon FL transformation, but the role of mitochondrial remodelling in FL progression has not been comprehensively characterized. We analyzed a transformation cohort (N = 11) with paired FL and DLBCL biopsies profiled using single-cell RNA and DNA sequencing. FL subtypes were assigned using a gene classifier, and subtype-specific transformation features were assessed via gene expression analysis. Our results show that OXPHOS pathway upregulation is a consistent feature of transformation in both FL subtypes, though driven by distinct mechanisms. Mem-like pairs exhibited significant increases in mitochondrial DNA copy number (mtDNA-CN) not observed in GC-like pairs, and showed increased expression of mitochondrial biogenesis genes. Notably, higher expression of OXPHOS-related genes and the mitochondrial transcription factor TFAM was associated with shorter time to transformation. In Mem-like FL, mtDNA-CN also showed a strong phylogenetic signal, suggesting a link between clonal evolution and disease progression. These findings reveal distinct mitochondrial dynamics between FL subtypes and highlight mitochondrial regulation as a predictor of transformation risk. This work enhances our understanding of FL biology and supports the development of improved risk stratification and predictive models for transformation to DLBCL.

LAY SUMMARY

Using single-cell RNA and DNA sequencing on 11 patients whose follicular lymphoma (FL) transformed into aggressive lymphoma (DLBCL), we found that both major subtypes of FL—germinal center-like (GC-like) and Memory-like (Mem-like)—show increased activity in a key energy-producing process called oxidative phosphorylation (OXPHOS) during transformation, but through different biological mechanisms. Notably, only Mem-like FL showed significantly higher levels of mitochondrial DNA and elevated expression of genes involved in mitochondrial production, including key regulators like TFAM and TFB2M. These changes were absent in the GC-like subtype. Higher mitochondrial activity in the Mem-like subtype was strongly linked to faster transformation and clonal evolution. This suggests that mitochondrial features, such as mitochondrial DNA copy number and OXPHOS pathway expression, could serve as predictive markers. These findings highlight the importance of mitochondrial regulation in FL transformation and may help identify high-risk patients earlier, enabling more personalized treatment strategies.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

A workflow for in-silico discovery of binders for cancer associated genes - Poster #61

Presenter: Kiran Narta, University of Calgary

Other Authors: Sorana Morrissy

ABSTRACT_ID#3342

ABSTRACT

The rapid advancements in cancer genomics in recent years have uncovered numerous cancer-associated genes and patient-specific antigens and mutations, paving the way for personalized therapeutic strategies including tailored binders.

Traditionally, binders for cancer-associated genes were derived from antibodies naturally occurring in animals, like camelids. However, these methods are often costly and time-consuming. Therefore, there is a need for fast and efficient methods to develop high-affinity binders for cancer-associated targets. The advancement in computational structural biology offers a potential to design accurate in-silico binders. In this study, we established a computational workflow to identify binders for proteins of interest. As a test case, we used GPNMB as a target. GPNMB is a transmembrane glycoprotein frequently overexpressed in cancers and has emerged as a promising target for immunotherapeutic approaches. We modeled GPNMB using AlphaFold3 and used extracellular domains to predict binders. The binder sequences were generated using BindCraft, yielding 100 acceptable candidates. To ensure specificity and minimize off-target effects, these binders were analyzed against proteins with GPNMB homologous structures (PMEL and TMEM130). Additionally, cross-species orthology of the binders was also evaluated. The binding metrics from the computationally predicted complexes were compared against ground truth complexes, consisting of experimentally validated anti-GPNMB antibodies with varying binding efficiencies, and also including species-specific antibodies. This analysis enabled the selection of top candidates of binders for experimental validation. This work establishes a robust pipeline for the discovery of binders targeting proteins or peptides of interest, with applications ranging from laboratory research to personalized clinical therapies.

LAY SUMMARY

We developed a computational workflow to discover binders for cancer-associated genes.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Contrasting Survival Models for Dynamic Prediction of Active Treatment Duration among Cancer Patients in Alberta, Canada - Poster #62

Presenter: Kenneth Lai, Alberta Health Services

Other Authors: Ali Manouchehrinia, Maeve Wickham, Cheligeer Cheligeer, Grace Heemeryck, Emily Walker

ABSTRACT_ID#2937

ABSTRACT

Background: Accurate prediction of active treatment duration (ATD) is essential for health system planning in cancer care. Selecting an appropriate survival model is critical when analyzing electronic medical records (EMR) data with censoring and time-varying features. We used EMR data collected in Alberta, Canada to assess the viability of deep learning, tree-based, and proportional hazard models for time-to-event analysis. Method: We applied a landmarking approach to test gradient-boost, Cox, Deephit and Dynamic DeepHit predictions of ATD for cohorts of breast (n=29,465) and hematological (n=15,725) cancer patients treated in Alberta from 2010-2023. ATD is defined as the interval from the first cancer-related consult/treatment visit to the last treatment-related visit. Key landmark times were 1/3/6/12 months. Static features remained constant, while dynamic features either accumulated or were kept as monthly time-varying inputs, depending on the model. Model performance was evaluated with concordance index (CI) and feature importance on a hold-out test set. Results: Traditional models such as Cox regression showed limited function with high-dimensional features. For breast cancer at the 1-month landmark, the CIs were 0.7532 (gradient-boosted), 0.7357 (Cox), 0.6957 (DeepHit), and 0.7552 (Dynamic DeepHit). At 6-months, the CI increased by 0.0121, 0.0248, 0.0142, and 0.0453 for each model, respectively, indicating the value of dynamic models in supporting time-varying features. Feature importance rankings were consistent across models, with patient age and cancer characteristics (grade, stage) being particularly predictive. Conclusions: Deep learning-based survival models provide a data-driven alternative to traditional models, that can better leverage time-varying features and yield higher predictive performance.

LAY SUMMARY

Deep learning-based survival models are more effective than traditional methods like Cox regression at handling large volumes of features, especially in high-dimensional healthcare data. Dynamic models can capture how patient data evolve over time, differing from traditional models that rely on summarized values. This is particularly important when using historical electronic medical records as time-varying data may reflect disease progression and treatment response. Deep learning-based dynamic models like Dynamic DeepHit combine the strengths of deep learning with time-varying modeling, improving performance over time as more longitudinal patient data is accumulated.

Non-Invasive, Label-free Imaging of 3D Cell Culture with Dynamic Optical Coherence Microscopy - Poster #63

Presenter: Kimiya Mousavi, Bc Cancer Research Institute

Other Authors: Yujie Hu, Adrian Tanskanen, Gina McNeill, Alexander Beristain, Myeong Jin Ju, Pierre Lane

ABSTRACT_ID#2841

ABSTRACT

MOTIVATION: Organoids are complex, three-dimensional (3D) cell culture models that emulate in vivo organs (cancerous and non-cancerous). Conventional imaging methods rely on destructive sectioning or incorporating fluorescent agents which may impact behaviour and limit longitudinal studies. This work seeks to develop and validate a non-invasive, label-free 3D microscopy platform using intracellular dynamics as a contrast mechanism. **BACKGROUND:** Trophoblast organoids (TB-Orgs) are a model system for the early human placenta consisting of an outer layer of cytotrophoblasts (CTBs) which differentiates an inner core of syncytiotrophoblasts (SCTs). Hypoxic TB-Orgs have reduced SCT differentiation; however, quantifying this using 2D sectioning is unreliable. Optical coherence microscopy (OCM) provides high resolution ($< 1 \mu\text{m}$) 3D imaging. By collecting repeated OCM cross-sections, subtle motions (intracellular metabolism) can be captured and interpreted as a colourmap (dynamic OCM). We hypothesize that dynamic OCM can noninvasively differentiate CTBs from SCTs in TB-Orgs without sectioning or staining. **METHODS:** Hypoxic (3% oxygen) and normal (20% oxygen) TB-Orgs embedded in Matrigel were imaged with the OCM system (3 batches, $n=6$ per batch). Intracellular dynamics were mapped to HSV color space, encoding mean frequency (hue), frequency variability (saturation), and intensity (value). Segmentation thresholds were applied to quantify CTB ring and SCT core. **RESULTS:** Dynamic OCM provided contrast and detail comparable to immunohistochemistry, identifying cavities and distinct layers; CTBs have a higher frequency response than SCTs. **IMPACT:** This technique shows potential as a non-invasive tool for 3D analysis of organoid differentiation, with broader applications in cancerous models to longitudinally examine the impact of hypoxia.

LAY SUMMARY

Current methods of imaging for 3D cell culture models requires slicing and staining the sample through histology or incorporating fluorescent markers that may alter cell function and limit application to endpoint analysis. Optical Coherence Microscopy (OCM) is an imaging method that can differentiate micron-scale structures within cells. As parts of cells vibrate, they metabolize energy causing subtle changes in how they interact with light. Dynamic OCM provides vibrant 3D maps of structures inside cells without harming the sample. This technique distinguishes between cytotrophoblasts (the outer layer of cells) and syncytiotrophoblasts (inner organoid core) by interpreting dynamic activity patterns, enabling cross sectional images without the need for staining or sectioning. Samples grown in low oxygen and normal conditions will be imaged to quantify the differences between them.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

ELM for Predicting Tumor Group from Pathology Reports - Poster #64

Presenter: Jean-Claude Bigenimana, Provincial Health Services Authority

Other Authors: Lovedeep Gondara, Shebnum Devji, Raymond Ng, Gregory Arbour

ABSTRACT_ID#2442

ABSTRACT

Background: Globally, the cancer burden is projected to reach 18.5 million deaths and 35.3 million new cases by 2050. Population Based Cancer Registries (PBCRs) collect standardized data from pathology reports which are unstructured text containing vital diagnostic information. Manual data extraction for cancer registries, especially tumor group assessment (900 person-hours/100,000 reports), is a significant bottleneck. Purpose: The British Columbia Cancer Registry (BCCR) uses eMaRC, a rule-based engine, for automated tumor grouping. However, eMaRC struggles with complex language, leading to inaccuracies. To improve tumor group classification, we developed ELM (Ensemble of Language Models). ELM combines smaller discriminative language models (SLMs) with a generative large language model (LLM). Methods: ELM employs an ensemble of six finetuned SLMs, with three analyzing the top and three the bottom of pathology reports to maximize coverage despite SLM input token limits. Agreement from five SLMs determines the tumor group. If consensus isn't met, the report and a curated prompt are sent to the LLM for arbitration. Results: On test data, ELM achieved average precision and recall of 0.94 and 0.88, respectively, for classifying reports into nineteen tumor groups. Comparative analysis against single models, partial ensembles, and an SLM-only ensemble confirmed ELM's superior performance. Conclusions: ELM effectively combines an SLM ensemble with an LLM for robust tumor group classification, ensuring high performance while optimizing LLM use.

LAY SUMMARY

A novel hybrid AI model (ELM), combining an ensemble of smaller language models (SLMs) with a large language model (LLM) for arbitration, significantly improves the accuracy and efficiency of classifying tumor groups from complex pathology reports, addressing the limitations of existing rule-based systems and simpler AI approaches. The ELM architecture is designed to optimize the use of resource-intensive LLMs by primarily relying on an SLM ensemble for classification and only escalating ambiguous cases to the LLM, thereby achieving high performance (0.94 precision, 0.88 recall) cost-effectively. This automated approach offers a viable solution to reduce the substantial manual workload (e.g., 900 person-hours per 100,000 reports for tumor grouping) in cancer registries, leading to faster and more reliable data collection crucial for cancer surveillance.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

AI for Predicting Pembrolizumab Monotherapy Response in High PDL1 Advanced NSCLC - Poster #65

Presenter: Liton Devnath, BCCRC

Other Authors: Ian Janzen, Cheryl Ho, Barbara Melosky, Stephen Lam, Ren Yuan, Calum MacAulay

ABSTRACT_ID#2591

ABSTRACT

Pembrolizumab monotherapy is an anti-PD-L1 immunotherapy that is approved as a first-line treatment for non-small-cell lung cancer (NSCLC) patients with high PD-L1 expression ($\geq 50\%$). However, approximately 55% of these patients do not respond. Early identification of likely non-responders is critical to enable timely transition to alternative treatments. This pilot study analyzed a retrospective cohort of NSCLC patients treated with first-line PD-L1 monotherapy, divided into a discovery set (n: 97; 27 non-responders) and an external test set (n: 17; 9 non-responders). Treatment response was assessed using baseline and follow-up CT scans in accordance with RECIST v1.1 criteria. A transfer learning approach was applied to extract deep learning (DL) features from CT images to differentiate responders from progressive disease patients. A log-rank test ranked the discriminative power of the 128 DL features found for the discovery set. Two types of support vector machine classifiers, an SVM with radial basis function (SVM-RBF) and polynomial kernel (SVM-Poly), were evaluated using the top-ranked features. Classifier performance was assessed at three scales: slice, lesion, and patient, and two approaches: with and without clinical descriptors. The SVM-RBF model performed best when trained on the top 10 features combined with clinical data, achieving an AUC of 0.75 (sensitivity: 81.5%, specificity: 81.4%) and an external test set F1 score of 72.9%. Integrating CT-based DL features with clinical descriptors demonstrated more balanced and robust performance, offering a promising tool to identify patients at risk of progression on pembrolizumab monotherapy to support first-line treatment decisions in high PD-L1 NSCLC.

LAY SUMMARY

A pilot study analyzed a retrospective cohort of NSCLC patients treated with first-line PD-L1 monotherapy. Transfer learning approach used to extract deep learning features from CT images. Integrating CT-based DL features with clinical descriptors demonstrated a balanced and robust performance.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Perception of Exercise and Receptiveness to Mobile Technology in Cancer Survivors Living in Rural and Remote Areas - Poster #66

Presenter: Myriam Filion, University of Alberta

Other Authors: Becky Franks, Dea'vion Godfrey, Carina McClean, Anna L. Schwartz, Diana J. Wilkie, Erin Maslowski, Saunjo L. Yoon

ABSTRACT_ID#2714

ABSTRACT

Background: Cancer survivors in underserved areas face additional barriers, such as limited access to oncology exercise programs, lack of experienced trainers, and limited facilities, contributing to health inequities in cancer incidence and outcomes. **Purpose:** To explore cancer survivors' thoughts on exercise and mobile technology to understand their preferences for exercising with a mobile application (app) during and after treatment in remote areas. **Methods:** Three focus-group sessions were conducted using semi-structured interviews regarding two themes: 1) exercise experience during and after treatments and 2) receptiveness to using an exercise app. Eligible participants were adult cancer survivors or caregivers living in Medically Underserved Areas, who consented to being audiotaped. The discussions were transcribed verbatim and analyzed via a content analysis approach with consensus. **Results:** Fifteen participants attended the focus groups. Survivor's cancer types included breast cancer, neuroendocrine carcinoma, melanoma, leukemia, papillary carcinoma, and multiple myeloma. The participants understood the benefits of exercise after diagnosis but expressed a need for more guidance during treatment. Geographic location shaped their activities, with most engaging in daily physical tasks such as activities related to farming and daily living rather than structured exercise. Most participants were receptive to using an exercise app, especially if it could help manage fatigue. They suggested key features such as videos, live trainers, health-tracking metrics, exercise checklists, visual benchmarks, and programs tailored to different fitness levels to enhance motivation to exercise with an app. **Conclusions:** These results emphasize the need for personalized resources, guidance, and on-demand accessibility to an exercise oncology app.

LAY SUMMARY

Rural cancer survivors face extra challenges staying active. Many don't have easy access to exercise programs or guidance during or after treatment, which can affect their recovery and long-term health. Cancer survivors want more support and tools to stay active. Survivors and caregivers see the value of exercise but need more help, especially during treatment. They are interested in using a mobile app if it includes features like exercise videos, progress tracking, and options for different fitness levels. A well-designed app could reduce health gaps. A cancer-specific exercise app that is easy to use and fits individual needs could help close the gap in care for people living in remote or underserved areas.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Development of an AI-Driven MRI Method for Hypoxia Mapping In-Vitro - Poster #67

Presenter: Maryam Abarashi, University of calgary

Other Authors: Ali Golestani, Clara Fallone

ABSTRACT_ID#3331

ABSTRACT

Hypoxia is a common feature of solid tumors and is linked to poor response to treatment, faster tumor spread, and worse outcomes for patients. There is a strong need for non-invasive methods that can identify and measure hypoxia inside tumors. Magnetic Resonance Imaging (MRI) is a safe, non-invasive imaging technique that creates detailed images of soft tissues without radiation. It is especially useful for hypoxia studies because oxygen levels change the MRI signal. This project aims to develop an artificial intelligence (AI) method that can use MRI data to map hypoxia levels. Human colon cancer cells (HCT116) will be grown into 3D clusters called spheroids inside microfluidic chips, which allow control of oxygen levels. Some spheroids will be kept in normal oxygen (normoxic) conditions, and others in low oxygen (hypoxic) conditions. To confirm which spheroids are truly hypoxic, we will measure the levels of a gene called carbonic anhydrase IX (CA-IX) using a technique called qPCR. The MRI images will be collected using a 3T Siemens scanner, and the average of different parameters will be driven. Then the correlation between different parameters and CA-IX results is investigated to train a machine learning model. This model will learn how to predict hypoxia based on MRI signals. The ultimate goal is to translate this AI-enhanced MRI workflow into clinical practice.

LAY SUMMARY

We develop a system to grow tumor models in microfluidic chips and scan them with MRI. We matched MRI data with gene markers (CA-IX) of hypoxia to train an AI model. This method supports future use of AI and MRI to detect cancer hypoxia and guide treatment.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

A narrative review of digital health interventions for the perioperative care of older cancer patients - Poster #68

Presenter: Michelle Pham, University of Calgary

Other Authors: Madeline McDonald

ABSTRACT_ID#3128

ABSTRACT

Background: Older adults experience the highest cancer burden and are at increased risk for postoperative complications, highlighting the need for improved perioperative care. Digital health interventions may address challenges, such as mobility and care coordination, yet their role in geriatric oncologic surgery is understudied. Objective: To synthesize evidence on digital perioperative interventions for older cancer patients, evaluate clinical and system-level outcomes, and identify considerations for equitable global implementation. Methods: A narrative review of EMBASE, Web of Science, MEDLINE, and CINAHL (to December 30, 2024), supplemented by Google and citation searches, included studies on digital perioperative interventions in cancer patients aged ≥ 65 . Data on study design, population, intervention type, outcomes, implementation facilitators/barriers, and global relevance were extracted. Results: Eight studies (2018–2023), all from high-income countries (US, Netherlands, Australia, Taiwan), met inclusion. Interventions included telemedicine ($n=3$), mobile or smart device-enabled symptom tracking ($n=7$), and wearable biometric monitoring ($n=7$). Reported benefits included improved physical activity, functional recovery, and complication detection. High adherence and acceptance were noted among all studies that measured them. However, systemic barriers were indicated, including limited internet access, device management issues, and digital literacy gaps. No studies examined cost-effectiveness, feasibility in LMICs, or cultural adaptability. Conclusions: Digital interventions may improve perioperative outcomes in older cancer patients, but current evidence is geographically limited and overlooks equity and contextual fit. Future research should apply implementation science to assess cost, cultural relevance, and system integration, especially in LMICs, to inform scalable, equitable models in global oncologic surgery.

LAY SUMMARY

Digital health interventions show promise in enhancing perioperative care for older adults with cancer, supporting improved physical activity, functional recovery, and early identification of complications. There is potential for digital health to support patient engagement and care coordination, as studies report high levels of adherence and acceptance among older patients when using tools such as telemedicine, mobile apps, and wearable devices. However, current evidence is limited to high-income countries and highlights barriers such as internet access, digital literacy, and device usability. The gap emphasizes the need for research focused on equitable, culturally adaptable implementation in diverse global settings.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Hybrid Approach for Enhancing Lesion Segmentation in Fundus Images - Poster #69

Presenter: Mohammadmahdi Eshragh, University of Calgary

Other Authors: Ezekiel Weis, Emad Mohammed, Behrouz Far, Trafford Crump

ABSTRACT_ID#2487

ABSTRACT

Purpose Uveal melanoma (UM) is a rare, but potentially fatal, form of eye cancer. Up to 50% of patients who receive a diagnosis of UM will die of metastasis after treatment of the tumor. The earlier UM is identified and treated the lower the risk of metastasis and the better the outcomes. However, identifying and monitoring lesions using diagnostic imaging is difficult. Machine learning (ML) aids lesion detection but faces challenges such as color similarity, shape asymmetry, and limited labeled data. A hybrid approach combining ML with traditional segmentation improves accuracy. **Objective** The purpose of this study was to develop and compare hybrid model against a conventional ML model for detecting UM in fundus images. **Methods** Fundus images from the Alberta Ocular Brachytherapy Program were utilized, with ground truth masks generated under the supervision of an ocular oncologist. The hybrid model was compared to Attention U-Net model. Traditional segmentation methods rely on object characteristics like color or intensity and require manual settings. Our hybrid approach trains ML models to provide those settings. **Results** The dataset included 253 RGB fundus, split into 183 (training), 46 (validation), and 24 (testing). The hybrid model achieved a Dice coefficient of 89.7% and an IOU of 80.1%, outperforming Attention U-Net, which achieved 51.3% and 34.2%, respectively. **Conclusions** The proposed hybrid model significantly improves segmentation accuracy for fundus images, outperforming a conventional ML model and enhancing the identification of choroidal nevus/UM.

LAY SUMMARY

Early detection of eye cancer is critical: Uveal melanoma is a rare but serious eye cancer. Half of the patients may develop fatal metastasis after treatment, so finding tumors early greatly improves survival chances. Hybrid AI model improves accuracy: We developed a hybrid model that combines traditional image analysis with machine learning. This approach helps overcome issues like limited training data and visual similarities between healthy and cancerous tissue. Our model outperformed existing AI tools: Using real patient images from the Alberta Ocular Brachytherapy Program, our hybrid model identified tumors much more accurately than a popular AI model (Attention U-Net), achieving nearly 90% accuracy compared to just 51%.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Expanding the definition of MHC Class I peptide binding promiscuity to support vaccine discovery across cancers with CARMEN - Poster #71

Presenter: Michal Waleron, University of Calgary

Other Authors: Ashwin Kallor, Aleksander Palkowski, Emilia Daghir-Wojtkowiak, Mikolaj Kocikowski, Fabio Marino, Beatriz Martinez, Javier Alfaro, Fabio Zanzotto, The KATY Consortium

ABSTRACT_ID#3074

ABSTRACT

Promiscuity in T-cell antigen landscapes refers to the dual flexibility of peptides binding multiple MHC alleles and MHC alleles presenting diverse arrays of peptides. By understanding how neoantigens are shared across varied HLA backgrounds, promiscuity analysis can inform the selection of cancer-vaccine targets that reach a wider segment of the population and help refine patient stratification for diverse immunotherapies. We expand the concept of promiscuity to encompass peptides, MHC alleles, individuals, populations, and genomic regions. Our CARMEN database release harmonizes data from 72 publications (2,323 samples) across tissue types, with a focus on cancer. Using Gibbs clustering and dimensionality reduction (UMAP), we systematically map promiscuity and immunological versatility across these biological levels. Gene and mutation analysis reveals recurrent cancer mutations in highly promiscuous genomic regions, highly mutated cancer genes that avoid presented regions, and sheds light on genomic regions important to response to immunotherapy.

LAY SUMMARY

We developed a comprehensive motif-landscape by analyzing 72 mass spectrometry datasets, using an unsupervised method to map peptide motifs across HLA alleles. This landscape visualizes similarities and differences in peptide clustering and forms the basis for a novel promiscuity score quantifying peptide diversity and recurrence across individuals, populations, and genomic regions. Mapping peptides to the genome revealed "hotspots" of antigen presentation—with a bias toward genomic/proteomic clustering, which we called epitope contigs. Using COSMIC data, we show that recurrent cancer mutations often occur outside these contigs. However, some highly immune-visible cancer genes overlap with promiscuous contigs, offering potential for vaccine targeting. We also extend this analysis to the dark proteome, examining promiscuity among non-canonical peptides. To assess clinical relevance, we use machine learning to predict immunotherapy response based on mutations within promiscuous peptides or contigs. We further investigate how predictive mutations differ in their contig promiscuity across coding and intronic regions.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Systematic Assessment of Prognostic Gene Signatures in Ovarian Cancer - Poster #72

Presenter: Mars Wichmann-Young, University of Ottawa

Other Authors: Ben Patrick, Arvind Mer

ABSTRACT_ID#3005

ABSTRACT

Introduction: Ovarian cancer (OC) remains a leading gynecological malignancy, characterized by poor clinical outcomes and biological heterogeneity. Many prognostic gene signatures have been proposed, yet their reliability across large-scale, heterogeneous patient cohorts remains unclear. **Methods:** We established a comprehensive OC transcriptomic atlas from 61 studies, encompassing over 7,000 samples with clinical metadata. This was used to systematically evaluate the prognostic efficacy of 6 well-established gene signatures for both overall survival (OS) and progression-free survival (PFS). Signature-based scores for each patient used mean gene expression, differentiating protective from risk-associated genes where applicable, and survival differences were determined using log-rank tests. We also identified prognostic genes across all datasets by conducting univariate and multivariate Cox regression followed by random-effects meta-analysis. **Results:** Overlap among the 6 gene signatures was minimal, with the highest overlap observed between two gene signatures under 4%. Univariate analyses revealed that the CLOVAR signature had the highest proportion of genes whose hazard ratios aligned with the direction of our meta-analysis findings. However, these signatures demonstrated limited prognostic power across datasets; the best-performing signature (CLOVAR), stratified OS and PFS in only 50% of datasets. Considering these limitations, we utilized our atlas and machine-learning methodologies to develop a novel prognostic gene signature with excellent predictive stratification. **Conclusions:** Existing prognostic gene signatures for OC exhibit limited generalizability and inconsistent performance across diverse cohorts. Leveraging our OC atlas and advanced machine-learning approaches, we developed a novel gene signature that shows promising reliability for risk stratification and may enhance prognostic assessment across diverse cohorts.

LAY SUMMARY

We analyzed existing gene signature-based biomarker panels for predicting ovarian cancer outcomes and found that they are unreliable across diverse patient populations. Using machine learning approach, we developed a new, more robust gene signature for improved prognostic accuracy. We also created a new and enhanced resource by curating data from over 7,000 ovarian cancer patients, helping to better understand link between genes and patient outcome.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Building an interoperable radiomics-dosimetrics knowledge base via mCODE for radiotherapy - Poster #73

Presenter: Odette Rios-Ibacache, McGill University

Other Authors: Amal Zouaq, John Kildea

ABSTRACT_ID#2378

ABSTRACT

Background: Glioma patients often experience tumor recurrence even after surgical resection, radiotherapy, and chemotherapy. A supporting AI radiomics-based algorithm for predicting glioma recurrence could potentially monitor patient evolution and follow-up care. However, the unstructured nature of real-world data limits model multicentre validation and clinical translation. Therefore, a strategy to structure relevant radiation medicine patient data is essential. **Methods:** This study proposes a strategy to structure patient data, encoding radiomics and dosimetrics, developing an ontology, and developing a knowledge base (KB). Building upon Minimal Common Oncology Data Elements (mCODE), we propose an extension to link patients' medical image data, radiomics, and dosimetrics with their electronic health records in a standardized way. A review of the existing literature on the harmonization and standardization of radiomics and dosimetrics methods was conducted to include the minimum parameters that would impact their extraction. We are also developing a feature-extractor module to automatically extract and integrate the elements and features needed for our extension, enriching the KB. **Results and Conclusions:** Our proposed extension currently includes 14 data sets with 99 attributes, incorporating an Imaging Study Profile, a Radiomics Report, and a Dosimetrics Report for each Cancer Patient Profile in mCODE. We developed the feature-extractor module mCODE-MOSAICO, which converts and stores the elements for our extension, automatically extracting the radiomics and dosimetrics features. This work introduces a novel framework for structured cancer data sharing, being an initial step in developing a KB. These results set the path for our research study in tumor recurrence prediction using multicentre datasets.

LAY SUMMARY

Glioma patients often experience tumor recurrence. A supporting AI model based on radiomics (quantitative data from images beyond human perception) for predicting this adverse outcome could potentially enhance follow-up care. However, health data in the real world is often disorganized and dispersed, limiting AI research and clinical translation. Our research proposes a strategy to structure real-world radiation oncology data, encoding radiomics and dosimetrics features with standardized elements to build a knowledge base (a coherent data repository) to enhance data interoperability among health centres. We developed a feature-extractor python module called mCODE-MOSAICO to automatically extract the elements for our extension and populate the knowledge base. This approach will facilitate our research on tumor recurrence prediction in glioma patients using multicentre datasets. Our work will serve as a template for future similar initiatives.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

A Radiomics-Based Breast Cancer Detection Model - Poster #75

Presenter: Ravi Bullock, BC Cancer Kelowna

Other Authors: Yiwen Xu, Rasika Rajapakshe

ABSTRACT_ID#2403

ABSTRACT

Purpose: This study uses radiologist annotations on screening mammograms to develop radiomics-based machine-learning models for early breast cancer detection. We trained models to differentiate breast tissue with abnormalities from normal breast tissue. **Methods:** A total of 1,604 screening mammograms (n=1,294 participants) were used in this retrospective study. Each mammogram included an original capture image, and a secondary capture image with one radiologist annotation outlining a region of interest (ROI) with an abnormality. The annotation on each secondary capture image was used to select an ROI in the original image. An ROI with normal tissue was automatically selected from the remaining breast tissue for comparison. Radiomics features were extracted from the ROIs with abnormalities and normal tissue. Feature selection was conducted using the Scikit-Learn SelectKBest method with chi-squared, ANOVA F-, and mutual information score functions. Six binary classifiers, logistic regression, random forest, XGBoost, bagging, discriminant analysis, and support vector machines were trained on the selected features. The model performance was assessed with the area under the receiver operating characteristic curve (AUC), sensitivity, and specificity on a holdout dataset. **Results:** The models achieved AUC values of 0.69-0.73 on the holdout dataset. The best model combined ANOVA F-score feature selection with a discriminant analysis classifier, achieving AUC=0.73[0.70, 0.77]. **Conclusion:** Radiomics-based models may distinguish ROIs with abnormalities from those with normal tissue on 2D full-resolution non-contrast enhanced mammograms. This pipeline offers an accurate, computationally-inexpensive, and interpretable solution for early detection. It is a step towards more complex algorithms being integrated into screening programs.

LAY SUMMARY

Breast cancer remains the leading cause of cancer-related death among women worldwide, with incidence rates projected to increase by 38% by 2050. Mortality is particularly high in areas with limited access to screening, such as rural and underserved communities, highlighting the necessity for affordable and accessible approaches to streamline the breast screening process. One promising approach is called radiomics. Radiomics is a technique that involves extracting hand-crafted features from medical images to train machine learning models. It has been successfully applied across various medical disciplines to enhance diagnostic accuracy and prognostic assessment. Radiomics models can be developed using low-cost hardware and are easily deployable on standard consumer-grade computers commonly found in healthcare settings. Moreover, radiomics models are interpretable by nature due to the manually-defined features, allowing for clinicians to easily understand their decision process. This study aimed to develop radiomics-based machine learning models to distinguish breast tissue with abnormalities and normal breast tissue on screening mammograms, thereby facilitating early breast cancer detection. The models demonstrated strong performance based on the area under the receiver operating characteristic curve values, sensitivity, and specificity. This study is intended to provide a foundation for the integration of more sophisticated algorithms into breast screening programs.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

A scoping review of AI-in-the-loop for decision-making in lung cancer screening - Poster #76

Presenter: Riley Ngo, University of Calgary

Other Authors: Alain Tremblay, Darren Brenner, Matt Warkentin

ABSTRACT_ID#2792

ABSTRACT

Background: Lung cancer is the leading cause of cancer-related mortality globally. Screening can detect cancers earlier, but optimization of workflows remains challenging. Artificial intelligence (AI) is being investigated as a complementary tool to improve detection and reduce radiologist workload (i.e., AI-in-the-loop), but its clinical performance across various workflows remains understudied. The objective of this study was to synthesize recent literature evaluating AI tools in lung cancer screening, focusing on diagnostic performance and workflow integration. **Methods:** We performed a scoping review using PRISMA-ScR in MEDLINE Ovid, Embase, and Scopus, including studies from 2020 to 2025 that evaluated AI performance in lung cancer screening using computed tomography (CT) or chest X-rays (CXR). Eligible studies used clinical data, measured quantitative performance, and compared AI to radiologist performance. We screened 101 studies in total and included 16 after applying inclusion criteria. **Results:** Studies evaluated AI as standalone (n=9), second-reader (n=5), first-reader (n=4), and concurrent (n=3), with some evaluating multiple. AI consistently improved sensitivity, particularly in first-reader and second-reader roles, but often increased false positive rates. One limitation is that all studies were retrospective and there was considerable heterogeneity in patient populations and algorithms, limiting generalizability. Several AI tools showed promise in reducing workload while maintaining diagnostic performance. **Conclusion:** AI can enhance sensitivity in lung cancer screening, particularly when integrated into hybrid workflows. However, gains may come at the cost of increased false positives. Our findings will inform microsimulation modeling using the OncoSim-Lung platform to inform lung cancer screening programs and AI integration in Canada.

LAY SUMMARY

Artificial intelligence (AI) can improve sensitivity in lung cancer screening when used as a tool alongside radiologists to analyze CT/CXR scans. This may help detect cancers that would otherwise be missed and reduce workload, though it may also increase false positives. AI showed promise as a first reader (i.e., used before radiologists) to rule out negative scans, while AI as a second or concurrent reader showed increased sensitivity compared to either AI or radiologists alone. This highlights AI's potential to triage negative scans and streamline diagnostic workflows. Data collected from this review will inform scenarios for the OncoSim-Lung model, allowing us to simulate the impact of AI integration in Canadian lung cancer screening programs.

Classifying MGMT Promoter Methylation Status using Vision Transformers and Radiomics - Poster #77

Presenter: Sana Mahmood, Toronto Metropolitan University

Other Authors: Nathan Churchill, Elodie Lugez

ABSTRACT_ID#2895

ABSTRACT

Purpose: Glioblastoma is an aggressive cancer that occurs in the brain and spinal cord. Treatment options include chemotherapy, most commonly in the form of temozolomide, and radiotherapy. Responsivity to temozolomide has been correlated with the methylation of the MGMT (O6-methylguanine-DNA methyltransferase) gene. However, obtaining a patient's MGMT promoter methylation status requires an invasive biopsy. In this study, non-invasive machine learning solutions to classify MGMT promoter methylation status in glioblastoma patients are developed. **Methods:** A machine learning model using Vision Transformers (ViT) was developed. The LUMIERE dataset was used to create this solution, which consists of multi-modal MRI scans of glioblastoma patients, and corresponding Radiomics features. To select relevant Radiomics features for this task, a feature selection model was constructed. The selected Radiomics features are embedded to corresponding T1c or T2-weighted MRI scans and inputted to the ViT for classification. The model classifies the patient corresponding to the MRI and Radiomics input as methylated or unmethylated for the MGMT gene. **Results:** Results of the feature selection show that the number of Radiomics features needed significantly reduce from 106 to roughly 21-35. The number of selected features varied with the complexity of the feature selection model used. Furthermore, the ViT performed the classification on MRI input alone at an average accuracy of 80%. **Conclusion:** The results of the Radiomics analysis and ViT suggest that the integration of the carefully selected Radiomics features to the ViT will significantly increase the accuracy of classifying MGMT promoter methylation status.

LAY SUMMARY

Machine learning models can be used to assist in treatment planning for glioblastoma cancer patients by classifying a key biomarker known as MGMT promoter methylation status. Our research shows that Vision Transformers, a type of machine learning model, can classify MGMT promoter methylation status using MRI input at an average accuracy of 80%. Our study is investigating the use of Radiomics, features extracted from medical imaging, as additional input to a Vision Transformer model to classify MGMT promoter methylation status in glioblastoma patients. Through a feature selection pipeline incorporating multiple models, we have reduced the number of Radiomics features needed for this task by up to 80%, from 106 features to a range between 21-35. Our study aims to leverage MRI scans and their corresponding selected Radiomics features as input to a Vision Transformer in order to improve the accuracy of classifying MGMT promoter methylation status.

Quantifying and Connecting Nuclear Atypia with Rapid Recurrence to Improve Grading in Non-Muscle Invasive Bladder Cancer - Poster #80

Presenter: Kathrin Tyryshkin, Queen's University

Other Authors: Ana Maria Vera Rodriguez, Sunghyun Kim, Katherine Lindale, Ava Slotman, Evelyn Yach, Kathrin Tyryshkin, David Berman

ABSTRACT_ID#3159

ABSTRACT

Non-muscle invasive bladder cancer (NMIBC) grading follows two systems: WHO 1973 (Grade 1, 2, 3) and WHO 2004 (High Grade [HG], Low Grade [LG]). The WHO 1973 system lacks clear criteria, leading to pathologist disagreements, while the simplified two-tier WHO 2004 system improves consistency but omits the Grade 3 category, limiting the detection of aggressive cancers. Prior studies using mean nuclear measurements obscured rare, atypical characteristics of Grade 3 nuclei linked to worse prognosis. We developed an interquartile range (IQR)-based outlier detector to quantify these abnormalities and validate the merged LG category, aiming to reduce variability and predict recurrence risk. We analyzed tissue cores from 255 stage Ta, BCG-naïve NMIBC patients. Visiopharm image analysis software extracted 11 quantitative nuclear size and shape measurements. An IQR-based outlier detection was stratified using a reference cohort of 5 Grade 2-HG samples. The detector computed the percentage of outliers based on nuclear size. To validate the merged LG category, each feature's mean and standard deviation (SD) were compared across LG tumours to characterize low-risk nuclear morphology. Kaplan-Meier analysis assessed recurrence-free survival. Outlier percentage for larger diameter best identifies rapidly recurring cancers ($p = 0.024$), carrying greater prognostic power than mean ($p = 0.82$) or SD ($p = 0.44$). Low variance in nuclear shape (convexity) best identified the slowest-to-recur cancers ($p < 0.001$). Quantifying atypical nuclei enables more standardized grading, reducing interobserver variability while identifying rapidly recurring cancers. This approach could enhance diagnostic accuracy, ensuring appropriate treatment for high-risk cancers and reducing progression risk.

LAY SUMMARY

Cancer grading reflects how much a tumour cell resembles a normal cell. Higher-grade cancer cells deviate further from normal cells, resisting treatment and recurring more often. Bladder cancer grading follows two systems: WHO 1973 (Grade 1/2/3 [G1/2/3]) and WHO 2004 (High Grade [HG], Low Grade [LG]). Both rely on qualitative characteristics, causing subjective grading and unclear boundaries. G3 is more aggressive, requiring intensive treatments. With recent advancements, it is now possible to quantify and analyze the shape and size of cell nuclei. We aim to use these quantifiable features to reduce variability, predict recurrence risk, and avoid unnecessary treatments to overall improve patient outcomes. Tumours with more irregular nuclear shapes were found to have higher recurrence risk in the LG cohort. Tumours with abnormally large nuclear size also showed faster recurrence within the G2-HG/G3 cohort. Thus, treating all LG or HG tumours similarly is problematic due to the prominent differences in recurrence risk. These results support defining quantitative thresholds for grading to improve the reliability and prognostic power of BC grading in clinical practice. Our findings show promise in increasing standardization, demonstrating how more objective, quantitative-based metrics can be implemented into pathology workflows to improve patient care.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Establishing an Artificial Intelligence (AI) Based Tool for Clinical Trial Matching: A Canadian Study Comparing Manual and Automated Approaches - Poster #81

Presenter: Depen Sharma, University of Windsor & Windsor Regional Hospital

Other Authors: Christina Trieu, Swati Kalia, Laurice Arayan, Megan Delisle, Utkarsh Akhouri, Eva Lorena Moreira Vieira, Renee Nassar, Ria Patel, Salah Alhajsaleh, Anaa Jaet, Anthony Luginaah, Milica Paunic, Pratham Gupta, Olla Hilal, Mahmoud Hossami, Roaa Hirmiz, Michae

ABSTRACT_ID#2496

ABSTRACT

Background: Since 2023, the Clinical Trials Navigator (CTN) program has doubled the rates of referrals to trials at Windsor Regional Hospital. Ongoing efforts are focused on expediting this process, including the use of artificial intelligence (AI). The proposed study will compare the clinical trial matching ability of AI versus the ongoing CTN program. **Methods:** The pilot project will use mock HER2-positive breast cancer cases to train, optimize and compare multiple AI algorithms. Inclusion/exclusion criteria will be manually extracted from actively recruiting Canadian trials. Our Clinical Trial Matching System (CTMS) combines natural language processing (NLP)-based trial matching with rule-based eligibility logic, integrating structured patient data and post-processed unstructured clinical notes. Patient-trial compatibility will be assessed using inclusion semantic similarity scores and categorical hard-matching. The resulting framework integrates rule-based filtering with NLP to automate early exclusion and surface potential trial matches. Eligibility assessments from the CTN, determined by consensus, will be used as the comparative arm. We will evaluate metrics (e.g., time and sensitivity) across both methods to further train and enhance our AI's performance. **Results:** CTMS currently matches one patient to multiple trials in eleven seconds, compared to the three-day average processing time manually by CTN. Preliminary results indicate that a GPT-based model can provide more targeted trial recommendations compared to non-GPT-based models. **Conclusion:** Optimization is necessary to evaluate whether the tool effectively matches patients to clinical trials. If successful, the results will be used prospectively with other tumour sites to help integrate AI into the CTN workflow.

LAY SUMMARY

AI can dramatically reduce trial matching time An internal study of our program found that patients who enrolled in clinical trials had a significantly shorter CTN processing time of 4.1 days compared to 12.5 days for patients who were not enrolled. Our Clinical Trials Matching System (CTMS) is able to match patients to trials in seconds, compared to days with the current manual process. Faster matching leverage with AI could therefore play a vital role in improving trial access and enrollment outcomes. Supports healthcare teams by reducing administrative burden Reviewing patient charts and trial eligibility criteria is time-consuming and needs to be done to ensure the patient is the right fit for a trial. Using a matchmaking tool can streamline this process and allow healthcare professionals more time to focus on patient care. Scaling to other cancers While still being refined, our similarity threshold has constantly improved and now identifies potential matches at a 0.6 similarity threshold, showing it's capable of learning and improving with more data. As this number increases, we will be able to expand beyond HER2-positive breast cancer and tailor it to work for many other disease sites, thus benefiting more patients over time.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

Mining the Medical Record: AI-Based Curation of Electronic Medical Records for Cancer Risk Prediction - Poster #82

Presenter: Tony Yu, Ontario Institute for Cancer Research/University of Toronto

Other Authors: Julia Sobotka, Jochen Weile, Trevor Pugh, Melanie Courtot

ABSTRACT_ID#3361

ABSTRACT

Introduction: Electronic medical records (EMRs) hold valuable longitudinal information that can inform cancer screening and risk stratification. However, patient data is often collected and stored as unstructured text; in order to meaningfully use this data, manual extraction and curation into standardized, interoperable data formats is necessary, a time-intensive process that is unfeasible at scale. Advancements in AI and Large Language Models (LLMs) have shown promising text processing and information extraction abilities for medical texts, providing a potential avenue for scalable data harmonization. Methods: Demonstrate accurate data curation into a common data model with LLMs: We will use unstructured EMR data from the CHARM study, a patient cohort with hereditary cancer syndrome. LLMs will be prompted to extract clinical entities from the unstructured clinical texts and format them into permissible vocabularies in the correct data fields. Preliminary results highlight the tool's time savings, with the automated LLM-based pipeline expected to significantly reduce time necessary for data curation compared to manual methods. Enable improved risk prediction of cancer disease-onset: Using data extracted and structured by the LLM-based pipeline, the clinical parameters will be used to train cancer risk prediction models, for outcome classification, risk stratification and risk score prediction. The accuracy of risk modelling will be assessed using real world clinical outcomes from the CHARM cohort. Integrating additional data modalities such as liquid biopsy features (fragmentomic features) will also be explored for its potential to improve risk modelling in this scalable, automated proof-of-concept.

LAY SUMMARY

AI for Medical Data Extraction: Medical notes constitute a massive source of patient data, yet analysis of this data at scale remains a challenge. AI tools such as Large Language Models (LLMs) can facilitate the automated extraction and curation of medical notes, streamlining the process of converting unstructured text data into standardized structured data for downstream analysis. Data Curation at Scale: LLMs enable the efficient processing and organization of vast amounts of medical data, enabling large-scale curation for research and clinical purposes, ultimately improving data usability and quality. Cancer Risk Prediction: By extracting key clinical features from ever abundant medical notes, LLMs could enhance predictive models for cancer risk, supporting earlier detection, more personalized treatment plans, and improved patient outcomes.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

Investigating EGFRvIII mRNA LNP vaccine-induced re-programming of the GBM TME using the 10x Genomics Visium HD platform - Poster #83

Presenter: Varsha Thoppey Manoharan, University of Calgary

Other Authors: Shannon Snelling, Gurveer Gill, Robert Nechanitzky, Xueqing Lun, Kristofer Ellestad, Joanna Pyczek, Kirsten Olsen, Yu Wu, Yury Karpov, Jun Liu, Matthew Gold, Haritha Menon, Yilin Tan, Rajesh Krishnan, Douglas Mahoney, Natalia Martin-Orozco, Jennifer Chan,

ABSTRACT_ID#3219

ABSTRACT

Glioblastoma (GBM) is a lethal brain tumor refractory to standard therapies, highlighting the need for alternative treatments. Immunotherapies, particularly cancer vaccines targeting neoantigens, offer a compelling approach to enhance anti-tumor immunity. To this end, we developed a preclinical mRNA lipid nanoparticle (LNP) vaccine targeting human EGFRvIII, a GBM-specific oncogenic variant, and evaluated its impact on survival in an EGFRvIII-driven tumor model, utilizing EGFRvIII-OE/CDKN2A-KO/PTEN-KO syngeneic mice. Following vaccine administration on days 7, 10, 14, 21, and 36 post-tumor cell implantations, vaccinated mice showed complete tumor clearance by day 30, whereas controls exhibited significant tumor burden. To elucidate the vaccine's therapeutic mechanisms and impact on the tumor microenvironment (TME), we performed VisiumHD spatial transcriptomic profiling on coronal brain sections from four mouse groups: buffer control and luciferase vaccine at day 21, and EGFRvIII mRNA-LNP vaccine-treated tumors at days 21 and 32. Using an unsupervised deconvolution approach, we identified 29 distinct transcriptional programs localized within the tumor lesion, comprising diverse glial, myeloid, and lymphocyte signatures. Notably, EGFRvIII vaccine-treated tumors exhibited a significant enrichment of pro-inflammatory and antigen-processing tumor-associated macrophage populations, alongside activated cytotoxic T-cells compared to controls. Furthermore, a differential neighborhood analysis between the EGFRvIII vaccine-treated and control samples revealed a close spatial association of these activated immune signatures, indicating a vaccine-induced re-programming of the TME to facilitate a coordinated anti-tumor response. Our results thus demonstrate the EGFRvIII mRNA-LNP vaccine as a potent therapeutic that drives GBM regression in preclinical models by modulating the TME, paving the way for its clinical translation.

LAY SUMMARY

Background: An mRNA lipid nanoparticle (LNP) vaccine targeting EGFRvIII - a mutation specific to GBM cancer cells - induces complete tumor clearance by day 30 following vaccine administration in an EGFRvIII-driven GBM mouse model. **Data:** To investigate mechanisms underlying the vaccine's therapeutic efficacy, spatial transcriptomics (10x Genomics Visium HD) was used to profile mouse brain sections from four experimental groups: buffer control and luciferase vaccine (day 21), and EGFRvIII vaccine-treated mice at days 21 and 32 post-treatment. **Methodology:** Spatial gene expression data were analyzed using an unsupervised machine learning algorithm to identify cell types, cellular activities, and spatial niches across the four conditions. **Results:** Treatment with the EGFRvIII vaccine led to a significant increase in tumor-suppressive macrophages and activated cytotoxic T cells in mice. Notably, these immune cells were found in closer proximity to each other, compared to control groups. **Conclusion:** The EGFRvIII vaccine reprograms the GBM tumor microenvironment (TME), promoting a coordinated anti-tumor immune response.

Bioinformatics, Big Data, AI, and Technology

Monday, November 3, 2025

But What Exactly Is Wrong with My Organoids? High-throughput Computer-Vision Pipeline for Organoid Developmental Trajectory Analysis - Poster #84

Presenter: Yiran Shao, Ontario Institute for Cancer Research

Other Authors: Jenna Haverfield, Sihem Sellami, Aimee K. Ryan, Koren Mann, Indra Gupta, Philip Awadalla

ABSTRACT_ID#2965

ABSTRACT

Organoids are widely used to model tissue development, disease progression, and therapeutic response. However, their broader application is hindered by high variability, unclear developmental endpoints, and reliance on subjective visual evaluation. We present a high-throughput computational pipeline for objective, interpretable analysis of organoid morphology and developmental dynamics using brightfield microscopy images. The pipeline performs robust image preprocessing, segmentation, and feature extraction, combining classical morphological descriptors with image embeddings from a self-supervised vision transformer model optimized for microscopy. These features enable the reconstruction of developmental trajectories across time and treatment conditions, allowing for the identification of branching events, aberrant subpopulations, and treatment-induced divergence. We demonstrated the utility of this framework across multiple datasets comprising diverse organoid types and perturbation conditions, including environmental toxins and genetic modifications. By integrating pseudotemporal ordering with feature attribution, our approach links complex image-derived features back to biologically interpretable phenotypes. This trajectory-aware framework accounts for both temporal and experimental metadata, offering a scalable and generalizable tool for assessing organoid culture quality, identifying phenotypic transitions, and modelling in vitro developmental trajectories relevant to cancer research.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

A Deep Learning System Classifies Cancer Origins Using Somatic Mutation Patterns Detected by Plasma Whole-Genome Sequencing - Poster #85

Presenter: Xindi Zhang, Ontario Institute for Cancer Research

Other Authors: Wei Jiao, Gurnit Atwal, Ander Díaz Navarro, Gregory Hogue, Quaid Morris, Lincoln Stein

ABSTRACT_ID#3140

ABSTRACT

A tumour's cell of origin is one of the most critical predictors of its natural history and response to therapy. However, identifying tissue origins can be challenging in patients with early-stage or metastatic cancer, particularly when biopsy is not feasible due to tumour size, anatomical location, or disease stage. Somatic passenger mutations captured by tumour whole-genome sequencing (tWGS) reflect cell-type-specific chromatin accessibility. Building on this insight, our lab developed DeepTumour, a deep neural network (DNN) that classifies 24 common tumour types with 91% accuracy. This project has two main aims: first, to expand DeepTumour using additional tWGS samples to improve cancer type resolution; and second, to adapt the model for plasma whole-genome sequencing (pWGS) to enable non-invasive tumour classification. We evaluated several normalization strategies and model architectures. The best-performing approaches were a DNN using mutation load-adjusted z-score-transformed features and a transformer-based model with multi-head attention. As both yielded comparable accuracy, we proceeded with the DNN approach. Expanding the training dataset with additional tWGS samples enabled classification of 31 tumour types with 90% accuracy. To simulate the lower mutation burden of pWGS, we subsampled somatic mutations from tWGS. The model maintained over 78% accuracy when more than 25% of mutations were retained—an encouraging result, as real pWGS samples can show up to >90% concordance with matched tWGS, though typically at lower levels. Testing the model on 178 pancreatic cancer patients' pWGS data yielded 76% accuracy, demonstrating DeepTumour's potential to support diagnosis, guide treatment, inform retrospective research, and adapt to pWGS.

LAY SUMMARY

Understanding Cancer Origins: Knowing where a tumour started in the body (its "cell of origin") is crucial for selecting the right treatment. This is typically determined by the tumour's organ and histology. However, identifying the origin can be challenging—especially when the tumour is difficult to access due to its size, location, or stage. **A New Non-Invasive Approach:** This research builds on our lab's tool, DeepTumour, which uses tumour DNA mutations to predict cancer type. We are now expanding it to work with plasma DNA collected from a simple blood draw—providing a safer, more accessible option for patients who cannot undergo a biopsy. **Encouraging Preliminary Results:** After expansion, the model accurately identified 31 cancer types with up to 90% accuracy using solid tumour sequencing. When tested on blood samples from 178 pancreatic cancer patients, it achieved 76% accuracy—highlighting its potential to support diagnosis, inform treatment decisions, and advance cancer research.

Pancreatic neuroendocrine tumour heterogeneity and metastatic machinery revealed by single-cell transcriptomic profiling - Poster #86

Presenter: Zoey Wang, Sunnybrook Research Institute

Other Authors: Nilakshi Kulathunga, Areeba Qureshi, Rusha Chakraborty, Lina Chen, Weei-Yuarn Huang, Hansen He, Iacovos Michael

ABSTRACT_ID#3141

ABSTRACT

Pancreatic neuroendocrine tumours (PanNETs) are increasing in incidence and highly lethal, with metastasis being a major negative prognostic factor. Current clinical grading offers limited guidance for disease management, as tumours of the same grade can range from indolent to highly metastatic and treatment-resistant. There is thus an urgent need to elucidate the molecular features underlying PanNET aggressiveness, particularly through resolving intra-tumour heterogeneity. Here, we performed single-cell RNA sequencing on 24 archived PanNET samples from 14 patients (9 female and 5 male), including matched primary and liver metastatic tumours, using the 10x Genomics Flex technology. This yielded a total of 124,416 high-quality single cells, comprising 65,594 cancer cells in addition to microenvironmental cell types. To uncover the mechanisms of PanNET progression, we performed pseudo-time analysis which identified a distinct subset of primary cancer cells primed for metastasis. Interestingly, gene set enrichment analysis revealed a coordinated upregulation of cilia- and hypoxia-related genes in these primary PanNET cells as well as in metastases. Hypoxia correlates with more aggressive cancer characteristics, and has been shown to modulate cilia function leading to the dysregulation of diverse signalling pathways. Our results suggest a potential role for the hypoxia-cilia axis in metastasis. Altogether, we have delineated a high-resolution landscape of PanNET molecular heterogeneity and a correlation of cilia and hypoxia with metastasis, a predictor of poor patient outcome. Our findings lay the groundwork for developing novel prognostic biomarkers and therapeutic targets to improve the clinical management of PanNET.

LAY SUMMARY

Patients with pancreatic neuroendocrine tumour display unpredictable clinical behaviour and high mortality. A subset of cancer cells acquires the ability to metastasise and drive disease progression. Hypoxia response and dysregulated cilia function are associated with the transition of cancer cells to more aggressive states.

Bioinformatics, Big Data, AI, and Technology

Sunday, November 2, 2025

AI for Single-cell Multi-modal Biology - Poster #87

Presenter: Xin Tang, University of British Columbia

ABSTRACT_ID#3182

ABSTRACT

Current biotechnologies can simultaneously measure multiple high-dimensional modalities (e.g., RNA, DNA accessibility, and protein) from the same cells. A combination of different analytical tasks (e.g., multi-modal integration and cross-modal analysis) is required to comprehensively understand such data, inferring how gene regulation drives biological diversity and functions. However, current analytical methods are designed to perform a single task, only providing a partial picture of the multi-modal data. Here, we present UnitedNet, an explainable multi-task deep neural network capable of integrating different tasks to analyze single-cell multi-modality data. Applied to various multi-modality datasets (e.g., Patch-seq, multiome ATAC + gene expression, and spatial transcriptomics), UnitedNet demonstrates similar or better accuracy in multi-modal integration and cross-modal prediction compared with state-of-the-art methods. Moreover, by dissecting the trained UnitedNet with the explainable machine learning algorithm, we can directly quantify the relationship between gene expression and other modalities with cell-type specificity. UnitedNet is a comprehensive end-to-end framework that could be broadly applicable to single-cell multi-modality biology. This framework has the potential to facilitate the discovery of cell-type-specific regulation kinetics across transcriptomics and other modalities.

LAY SUMMARY

Bulk, single-cell, and spatial genomics have improved our ability to dissect tumor ecosystems. Computationally, if we are to rely on AI to understand Cancer Genomics, we must know when to trust the results. We developed an explainable machine learning method to dissect gene regulation in single-cell multi-omics.

Docosahexaenoic acid supplementation in women with breast cancer undergoing neoadjuvant chemotherapy: assessment of efficacy and immune function in the DHA WIN trial - Poster #88

Presenter: Jaqueline Munhoz, University of Alberta

Other Authors: Marnie Newell, Gilbert Bigras, Susan Goruk, Anil Abraham Joy, Sunita Ghosh, Vera Mazurak, Kerry S. Courneya, Catherine J. Field

ABSTRACT_ID#2334

ABSTRACT

Background: The anti-cancer effects of docosahexaenoic acid (DHA) have been demonstrated in rodent models of breast cancer treated with neoadjuvant chemotherapy (NAC). However, clinical evidence is limited. This randomized, double-blind, placebo-controlled trial aimed to determine DHA supplementation's efficacy and immune effects during NAC. **Methods:** Participants received either DHA 4.4 g/day orally (n=23) or a placebo (n=26) over 18 weeks of NAC. The primary outcome was treatment efficacy, measured as changes in the percentage of the proliferation marker Ki-67, assessed by immunohistochemistry analysis. Secondary outcomes included changes in the DHA content of plasma phospholipids quantified by gas-liquid chromatography and the ability of immune cells to produce cytokines in response to ex vivo antigen challenge. **Results:** The % of DHA in plasma phospholipids was increased twofold at 9 and 15 weeks of NAC in the DHA group ($P<0.001$). The expression of Ki-67 decreased in both groups ($P<0.001$), with no effect of DHA. However, when stratified by breast cancer subtype, there was a trend to a greater reduction of Ki-67 in the human epidermal growth receptor 2 positive (HER2+) subtype in the DHA group ($P=0.1$). After 15 weeks of therapy, the DHA-supplemented group produced more IL-10 and IFN- γ after phytohemagglutinin challenge, and more TNF- α and IFN- γ after lipopolysaccharide ($P<0.05$). **Conclusion:** NAC significantly decreased the expression of Ki-67. The efficacy of DHA on the HER2+ subtype remains unclear. Immune findings suggest that DHA may help protect against the NAC's negative immune effects. The impact of these immune differences on health outcomes requires further research.

LAY SUMMARY

Docosahexaenoic acid supplementation during chemotherapy does not appear to enhance overall treatment outcomes; however, breast cancer subtypes may be an important factor. Docosahexaenoic acid supplementation during chemotherapy may help preserve the immune system.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Trial design with an equity lens: A focus on improving access to cancer clinical trials - Poster #89

Presenter: Anna Johnson, Canadian Cancer Trials Group

Other Authors: Mariam Jafri, Ian Poon, Michael Kolinsky, Wei Tu, Keyue Ding, Willard Richardson, Erwin Wanderer, Sarah Radcliffe, David Boren, Ryan Kelly, Wendy Parulekar, Janet Dancey

ABSTRACT_ID#2654

ABSTRACT

Clinical trials are essential for establishing new standards of care, yet trial participants often do not reflect the diverse populations who will ultimately use these treatments. To address this gap, the Canadian Cancer Trials Group (CCTG) selected two randomized trials—HN13 (NCT06641791), a head and neck cancer trial, and PR25 (NCT06439225), a prostate cancer trial—to implement targeted initiatives aimed at improving trial equity, diversity, and accessibility. A full-day workshop brought together trial teams, CCTG leaders, patient partners, and equity and clinical trial experts to review the demographics of prostate and head and neck cancer patients in Canada and identify strategies to enhance inclusion. Key priorities emerging from the workshop included: 1) Embedding equity, diversity, and inclusion (EDI) training into trial start-up webinars; 2) Establishing a trial reimbursement policy for participant expenses; 3) Developing Trial Diversity Action Plans with communication, education, and outreach components; 4) Translating patient-facing materials into additional languages; and 5) Creating resources to support culturally safe, trauma-informed, and inclusive trial delivery. Implementation of specific tools to assist with equitable recruitment, protocol changes to improve access, and monitoring plans are also underway. Prioritizing health equity for trial participants has led to systematic improvements in trial planning, conduct, and communication strategies. CCTG is a National Program of the Canadian Cancer Society (grant number: 707213).

LAY SUMMARY

Striving for diversity in clinical trial participants is essential for achieving health equity. Improving equity, diversity, and access in clinical trials requires a collaborative and multi-pronged approach. A focus on health equity can systematically improve trial planning, conduct, and communication strategies.

A Phase I-II Study of Niacin in Patients with Newly Diagnosed Glioblastoma Receiving Radiotherapy and Temozolomide: Phase I Tolerability Results and Phase II Interim Analysis - Poster #90

Presenter: Gloria Roldan Urgoiti, Arthur Child Comprehensive Cancer Centre

Other Authors: Paula de Robles, Roger Tsang, Morgan Willson, Sunita Ghosh, Muhammad Faruqi, Gerald Lim, Shaun Loewen, Robert Nordal, Gregory Cairncross, Catriona Leckie, Candice Poon, Wee Yong

ABSTRACT_ID#2939

ABSTRACT

INTRODUCTION Median survival of patients with newly diagnosed glioblastoma (GB) treated with standard of care (SOC) surgery, radiotherapy, and temozolomide is 15 months with progression free survival at 6 months (PFS-6M) of 53.9%. In vivo studies demonstrated increased survival in mice with GB treated with niacin. **METHODS** This is a first in human Phase I-II study of controlled-release niacin (Niacin CRT™) added to SOC. Phase I evaluated intra-patient dose escalation of niacin (500 - 3000 mg/d) to determine dose limiting toxicity (DLT), maximum tolerated dose (MTD) and recommended phase II dose (RP2D). Phase II aims to determine if niacin adds $\geq 20\%$ absolute increase in PFS-6M over historical controls. Interim/futility analysis was planned when 24 patients became evaluable for PFS-6M. The study would stop if the conditional power (one-sided Z test) $< 20\%$ or futility index $> 80\%$. **RESULTS** Phase I was completed with 15 patients of median age: 57 years (37-68), 40% women, and 47% with MGMT promoter methylated. The most common side effect was flushing (10/15; 9 grade 1). Two DLTs occurred at 2,500 mg/d niacin (grade 3 thrombocytopenia and hyperbilirubinemia). Niacin dose escalated up to 2000 mg/d is the ongoing RP2D. Twenty-four patients became evaluable for PFS-6M on May 28, 2025 and central radiology review reported PFS-6M of 82.3%. Monitoring Board review is scheduled for June 18, 2025. **CONCLUSIONS** The MTD dose of Niacin CRT™ added to first line treatment in patients with GB is 2000 mg/d. The interim analysis already showed an absolute increase in PFS-6M of 28%.

LAY SUMMARY

Glioblastoma (GB) is an aggressive brain cancer and despite surgery, radiotherapy and chemotherapy, only 53.9% of the patients are free of progression of the tumor 6 months after diagnoses. Researchers showed that in mice with glioblastoma, adding niacin improved survival. This is the first study in humans testing if controlled-released niacin [Niacin CRT (TM)] can be added to the treatment safely (phase 1) and if it improves the control of the tumor (Phase 2); we considered that an improvement in disease control from 53.9% to 73.9% at 6 months would be significant. In the phase I, fifteen patients planned to escalate the dose of niacin from 500 mg to 3000 mg/d but we found that the maximum safe dose was 2000 mg/d with liver damage and low platelets at higher doses. The phase II was planned with 48 patients and it had an analysis to know if the study was worth continuing when 24 patients were on treatment for 6 months (May 28/2025). The statistician reported that 82.3% of the patients did not have tumor progression at the 6-month mark. This is better than we had expected. On June 18, 2025 an independent committee will review these results.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

A National Clinical Trial Finder for all Canadians: Insights from CCS's Feasibility Study - Poster #91

Presenter: Alana Chalmers, Canadian Cancer Society

Other Authors: Tracy Torchetti, Devin Gauthier

ABSTRACT_ID#2799

ABSTRACT

The Canadian Cancer Society (CCS) conducted a feasibility study to assess the potential of developing a national cancer trial finder program after the pending discontinuation of CanadianCancerTrials.ca (CCT). CCT was Canada's only bilingual, pan-Canadian cancer trials database. Three key findings emerged from our study which engaged patients and advocates, industry, researchers and other cancer system stakeholders. First, there was strong consensus among stakeholders on the urgent need for a patient-centred Canadian source of clinical trial records. Canadians are overly reliant on ClinicalTrials.gov which lacks patient-friendly content, limiting accessibility and understanding among patients. Second, stakeholders viewed CCS as a natural steward for a next-generation, patient-centric trial finder. Many felt CCS was uniquely positioned to lead this initiative due to its trusted reputation among cancer patients, its reach, and its established role in cancer research. The study also highlighted the opportunity for CCS to enhance trial awareness and access through such a program. Third, the study established that for CCS to be successful with this program, it would require collaboration with partners across the cancer system. This would be necessary to help centralize trial record data, ensure trial record accuracy with timely updates, and reach patients where they are. Based on the findings of this study, CCS built a partnership with Quebec Clinical Research in Oncology (Q-CROC) to develop the Cancer Trials Canada (CTC) program, with funding support from Canadian Partnership Against Cancer.

LAY SUMMARY

1. Canadians need better access to cancer clinical trial information. With the discontinuation of CanadianCancerTrials.ca, patients and caregivers face challenges in finding trial information. Many rely on ClinicalTrials.gov, which lacks patient-friendly content, making it difficult to navigate. A national, patient-centered trial finder is urgently needed.
2. The Canadian Cancer Society is positioned to lead this initiative. Stakeholders—including patients, advocates, industry, and researchers—agree that CCS is a trusted source of cancer information. With its established role in research and patient support, CCS is well-suited to create a next-generation trial-finding platform that improves awareness and access.
3. Collaboration is key to success. To build an effective cancer trial finder, CCS must work with partners across the cancer system. Centralizing trial data, ensuring record accuracy, and reaching patients through diverse channels will be essential. With support from Quebec Clinical Research in Oncology and Canadian Partnership Against Cancer, CCS is making this vision a reality.

Cancer Clinical Research and Trials

Monday, November 3, 2025

The SEAMLESS Trial: A Randomized Waitlist-Controlled Study of a Mindfulness App for Psychosocial Symptom Management in People with Cancer - Poster #92

Presenter: Hanna Conradi, University of Calgary

ABSTRACT_ID#2661

ABSTRACT

Background: Many people living with cancer (PWC) experience persistent psychosocial symptoms post-treatment, which can increase healthcare use, impact treatment adherence, and reduce quality of life. Mindfulness-Based Interventions (MBIs) are effective in reducing such symptoms, but there is a reported need for more accessible delivery formats, such as mobile app-based MBIs. Although in-person MBIs appear to improve symptoms by targeting mechanisms like rumination and experiential avoidance, it remains unclear whether app-based MBIs work through the same pathways. Moreover, the potential moderating role of demographic and disease factors has not been adequately explored. **Methods:** This multi-site randomized, waitlist-controlled clinical trial is enrolling 345 Canadian PWC post-treatment (n=202 to date; Immediate: 98, Waitlist: 104). Participants complete validated measures of mindfulness, rumination, experiential avoidance, and psychosocial symptoms (e.g., depression, anxiety, fear of recurrence, fatigue, physical function, quality of life). Two-level hierarchical linear modeling will test symptom changes between groups at 3-month follow-up. Structural equation modeling will evaluate mediation and moderation. **Results:** The sample includes 50% females and a mean age of 59 [29–84]). Diagnoses include breast (36–39%), prostate (20–22%), lung (2%), head & neck (2–4%), and “other” (36–38%). 78% were diagnosed at early stage and 22% at late-stage. Preliminary results will describe baseline symptom levels and adherence rates. **Conclusions:** Findings will inform the development of accessible, mechanism-informed MBIs tailored to diverse cancer survivors to improve psychosocial outcomes post-treatment.

Outcomes of Temozolomide and Capecitabine chemotherapy used in Neuroendocrine and Endocrine Tumours: A Real-World Comparison of evidence in Alberta, Canada, from 2011-2021 - Poster #93

Presenter: Alda Aleksy, Arthur Child Comprehensive Cancer Centre

ABSTRACT_ID#2599

ABSTRACT

The combination of capecitabine and temozolomide (CAPTEM) showed PFS benefits in pancreatic neuroendocrine tumours (NETs). We aimed to assess its effectiveness in the broader NETs(neuroendocrine and endocrine tumors) population. We identified all patients with NETs treated with CAPTEM (at least 1 cycle) in Alberta from 2011-2021. PFS and OS from start of CAPTEM were compared between groups stratified by primary: Pancreatic NETs (PNETs); gastrointestinal NETs (GINETs); Pulmonary NETs (PuNET); other NETs (ONETs). PFS and OS were compared between NETs when CAPTEM was used as first vs. later- line therapy and patients receiving ≥ 6 vs. < 6 cycles. Among 159 patients identified, there were 67 PNETs, 38 GINETs, 35 PuNETs, 19 ONETs. Compared to PNETs, GINETs were not associated with statistically significant difference in median PFS (10.5 vs 9 m, $p=.11$; HR=1.14). Compared to PNETs, PuNETs and ONETs were both associated with statistically significant lower median PFS (PuNETs 3vs9m, $p=.02$, HR=1.6 95; ONETs 2vs9m, $p=.01$, HR= 1.56. OS analyses revealed similar results. When CAPTEM was used as first vs. later-line therapy, there was higher median PFS (10vs3.5m, $P=.008$; HR=0.56) and OS (23vs4.7m, $P<.0001$; HR=0.42). Patients receiving ≥ 6 vs < 6 cycles of CAPTEM had higher median PFS (18vs2m, $P<.0001$; HR=0.22) and OS (29vs4.2m, $P<.0001$; HR=0.22). CAPTEM shows similar efficacy in GINETs as in PNETs but might not be as effective in PuNETs and ONETs. Notably, use of CAPTEM in a first-line setting and with an extended cycle regimen significantly correlates with improved OS across the broad spectrum of NETs.

LAY SUMMARY

Capecitabine/temozolomide (CAPTEM) showed PFS benefits in PNETs. We assessed its effectiveness in the broader neuroendocrine and endocrine tumor population. We identified patients with NETs/ETs treated with CAPTEM (≤ 1 cycle) in Alberta from 2011-2021. PFS and OS from start of CAPTEM were compared between groups stratified by primary NETs: Pancreatic (PNETs); gastrointestinal (GINETs); Pulmonary (PuNET); other (ONETs). PFS, OS were compared when CAPTEM was used as first vs. later- line and in patients receiving ≥ 6 vs. < 6 cycles. Compared to PNETs, GINETs were not associated with statistically significant difference in mPFS (10.5 vs 9 m, $p=.11$). Compared to PNETs, PuNETs and ONETs were both associated with SS lower mPFS (PuNETs 3vs9m, $p=.02$; ONETs 2vs9m, $p=.01$,). OS analyses revealed similar results. When CAPTEM was used as first vs. later-line therapy, there was higher mPFS (10vs3.5m, $P=.008$;) and OS (23vs4.7m, $P<.0001$;). Patients receiving ≥ 6 vs < 6 cycles had higher mPFS (18vs2m, $P<.0001$) and OS (29vs4.2m, $P<.0001$). CAPTEM shows similar efficacy in GINETs as in PNETs but is not as effective in PuNETs and ONETs. Notably, use of CAPTEM in a first-line setting and with an extended cycle regimen significantly correlates with improved OS across the broad spectrum of NETs.

Effects of exercise on body composition and sarcopenia in people with colon cancer: a CHALLENGE sub-study protocol - Poster #94

Presenter: Ignacio Catala Vilaplana, University of British Columbia

Other Authors: Howard J. Lim, Kerry S. Courneya, Christopher M. Booth, Anthony Reiman, Derek J. Jonker, Haji Ibraheem Chalchal, Kamal Haider, Kelvin K.W. Chan, Mahmoud Abdelsalam, Michael Sanatani, Neil S. Chua, Ralph P.W Wong, Ronald L. Burkes, Sasha Lupichuk, Stacey Hubay, Cameron J. Mitchell, Colin Mar, Elizabeth M. Cespedes Feliciano, Gillian Hanley, Grant R. Williams, Christopher J. O'Callaghan, Kristin L. Campbell

ABSTRACT_ID#2880

ABSTRACT

The CHALLENGE Trial has recently demonstrated longer 5-year disease-free survival (Exercise group (EG): 80.3% vs Health-Education group (HEG): 73.9%) and 8-year overall survival (EG: 90.3% vs HEG: 83.2%) in colon cancer patients after a 3-year structured exercise program. Purpose: This sub-study will leverage data from a large randomized clinical trial (CHALLENGE Trial) to test the effect of the first 12-months of the 3-year long exercise intervention compared to health-education group on muscle health and body composition in adults with colon cancer using computed tomography (CT) images. Methods: Following ethics approval at each site in Canada who has agreed to participate, CT scans at the time of diagnosis and 12-month follow up visit for the CO.21 will be de-identified. Whole body tissue volumes will be estimated based on CT images at the level of the third lumbar vertebra using DAFS Express (Voronoi Health Analytics Inc., Vancouver BC). Height-adjusted muscle quantity will be measured as skeletal muscle index (SMI), muscle quality will be measured as skeletal muscle density (SMD) and skeletal muscle gauge (SMG). Quantity of visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), intermuscular adipose tissue (IMAT), and total adipose tissue (TAT) will also be measured. Results: At the 14 Canadian sites of the CO.21 trial, there are a potential of 503 participants. Final results are expected in 2026. Conclusion: This work will provide insight into mechanisms linking physical activity to cancer outcomes and inform future studies of optimal lifestyle interventions to improve cancer outcomes.

LAY SUMMARY

- The CHALLENGE Trial has recently demonstrated longer 5-year disease-free survival (Exercise group (EG): 80.3% vs Health-Education group (HEG): 73.9%) and 8-year overall survival (EG: 90.3% vs HEG: 83.2%) in colon cancer patients after a 3-year structured exercise program.
- Observational evidence consistently shows that poorer muscle quality, lower muscle mass, and higher adiposity are associated with an increased risk of cancer recurrence, cancer-specific mortality, and all-cause mortality. Lifestyle interventions, particularly physical activity, hold the most promise as a treatment approach in cancer care for improving muscle quality, muscle quantity, and adiposity.
- This sub-study will leverage data from a large randomized clinical trial (CHALLENGE Trial) to test the effect of the first 12-months of the 3-year long exercise intervention compared to health-education group on muscle health and body composition in adults with colon cancer using computed tomography (CT) images to provide insight into mechanisms linking physical activity to cancer outcomes and inform future studies of optimal lifestyle interventions to improve cancer outcomes.

Exploring the impact of Canadian propolis during cancer therapy-induced cell fate decisions - Poster #95

Presenter: Andrea Manjarrez Treo, CRCHUM et Institut du cancer de Montréal

Other Authors: Nicolas Malaquin, Francis Rodier, Yann Loranger, Isabelle Rabbat

ABSTRACT_ID#3309

ABSTRACT

Cell senescence is a critical tumor suppression mechanism, yet the accumulation of senescent cells contributes to aging and tissue dysfunction. Particularly, senescent cells accumulate more rapidly during cancer treatment, and targeting senescence-associated phenotypes is a therapeutic opportunity. Unfortunately, current senotherapeutics are often toxic or ineffective in vivo highlighting the need for alternatives. Propolis, a bee-derived natural product high in polyphenols and flavonoids, has demonstrated anti-inflammatory, antioxidant and anticancer properties, suggesting potential to modulate senescence and other cell fate decisions, such as apoptosis during anticancer therapies. We hypothesize that propolis will alter senescence- and cell survival-associated phenotypes during anticancer therapies in normal and cancer cells, including the pro-inflammatory senescence secretome, which interface with the immune system. To test this, we acquired high-quality propolis extracts from Canada and Brazil with compound traceability and chemical content characterization, supplied through a collaboration with the Canadian beekeepers and researchers at Bee-O-Pharm. Using a versatile real-time single-cell imaging pipeline enabling simultaneous detection of cell proliferation, death and senescence, we defined the cytotoxic profile of red, green, and brown propolis in normal human cells. We are currently investigating and will discuss the effects of propolis on cell death, senescence and inflammation in multiple cancer subtypes. Actions of propolis identified in this context will be further tested in advanced 3D pre-clinical models. As a non-toxic and accessible compound from Canadian sources, propolis may enable the development of inhalable or topical therapeutics suitable for specific cancer contexts, offering novel strategies to exploit cell fate modulation for clinical benefit.

LAY SUMMARY

Propolis shows promise in fighting senescence cells and cancer: Propolis, a natural bee product, contains different chemical compounds like polyphenols and flavonoids, that confers anti-inflammatory, antioxidant and antimicrobial properties along with antiaging, antisenescence and antiproliferative activity. These helps slow down cancer cell growth, trigger cancer cell death and block tumor spread by targeting several critical pathways in the cell. Different types of propolis target senescence and cancer in unique way: Green, red and brown propolis extracts and their own chemical compound quantity for flavonoids or polyphenols such as CAPE and CA, according to our preliminary laboratory findings, act in different ways (depending on the type and concentration) in the human normal cells. Safer cancer treatments may be supported by propolis with fewer side effects: Although current drugs that target senescence cells are effective and beneficial (sometimes), they can cause serious side effects. Propolis may increase their effectiveness while lowering negative side effects including immune suppression or tissue damage, offering a non-toxic, low-cost option for patients with suppressed immune system, providing a more secure, natural alternative or complement to harsh treatments.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Assessment of OncoQuébec, an Oncology Clinical Trials Search Engine, and Onco+, a Personalized Navigation Support Tool, in Quebec - Poster #97

Presenter: Mariam Mehran, Quebec – Clinical Research Organization in Cancer (Q-CROC)

Other Authors: Lucie D'Amours, Nawal Haddadi, Amélie Bérubé, Catherine Vayssier, Marie-Claude Guertin, Jean-Paul Bahary, Gerald Batist

ABSTRACT_ID#2509

ABSTRACT

Background: OncoQuébec is Quebec's trial search application in oncology, featuring a free navigation service, Onco+. Clinical trial search tools are widely available, but data on their use and impact remain limited. Here, we describe the impact of OncoQuébec on patient recruitment and the use of Onco+ service in Quebec. Methods: OncoQuébec's impact was evaluated retrospectively using data from three major recruiting centers. Number of patients recruited was examined across studies that were categorized as "higher traffic" vs. "lower traffic" studies according to the median number of users, views and clicks to study contact. Contact details were accessible for all studies. Onco+ use was assessed from May 2022 to April 2025. Results: For OncoQuébec's evaluation, a total of 312 studies was analyzed. These studies resulted in a cumulative total of 1,683 users, 2,747 views and 429 clicks. A total of 662 patients were recruited. Studies with higher traffic (n=97) recruited significantly more patients, 2.0 times (95% CI: 1.3, 3.3) more, than studies with lower traffic (n=215). Onco+ was accessed by an average of 9 users/month through direct searches and referrals. Most requests were from patients or caregivers (90%), for advanced cancers (79%), and over half were from outside Quebec's major centers. Survey results showed 85% of users were satisfied with the service. Conclusions: OncoQuébec's assessment suggests that this platform drives traffic and positively influences recruitment. Onco+ is used throughout Quebec with a high satisfaction rate. The plan is to expand the use of these search and navigation tools across Canada.

LAY SUMMARY

Quebec-Clinical Research Organization in Cancer (Q-CROC), a not-for-profit organization, has the mission to improve accessibility to oncology clinical trials in Quebec. Q-CROC's platform, OncoQuébec, is Quebec's trial search application in oncology, featuring a free navigation service, Onco+, available for both patients and healthcare professionals. The objective of this research is to describe the impact of OncoQuébec on patient recruitment and the use of Onco+ service. Studies with higher traffic recruited twice as many patients as those with lower traffic. This statistically significant finding suggests that OncoQuébec could positively influence recruitment. Onco+ is used throughout Quebec with a high satisfaction rate. Most requests were from patients/caregivers, for advanced cancers, and over half were from outside Quebec's major centers. It is to the benefit of all sponsors and recruiting sites to have their clinical studies listed on OncoQuébec with the most up-to-date and accurate clinical trial information. The plan is to expand the use of these search and navigation tools across Canada.

Cancer Clinical Research and Trials

Monday, November 3, 2025

Identification and Comparison of Novel Interventions for Stages IIIB and IIIC Non-Small-Cell Lung Cancer - Poster #98

Presenter: Marc Kerba, Alberta Health Services

Other Authors: Lauren Tam

ABSTRACT_ID#3114

ABSTRACT

Non-small cell lung cancer is one of the most common types of cancers and the current standard of care for stages 3B/C is chemoradiation and immunotherapy. A large proportion of patients experience significant side effects after chemoradiation, such as pain, fatigue, and overall decreased quality of life. These patients are unable to proceed to consolidation with immunotherapy as a result. To counteract these symptoms, adjuvant interventions must be effective and financially accessible. A systematic review was conducted to evaluate interventions so as to compare their efficacy, cost-effectiveness, and feasibility. The databases of the National Center for Complementary and Integrative Health, Natural Medicines, MEDLINE (Ovid), PubMed, Cochrane Library, CINAHL, and MEDLINE (Ebsco) were searched. Four categories of interventions were found and evaluated; being mind-body exercises, breathing exercises, dietary advice on caloric intake, and fatty acid supplements. Each intervention was analyzed for effects on quality of life, physical function, and emotional wellbeing using a scoring system to evaluate the interventions. It was found that mind-body exercises are most likely to be beneficial as they were most effective at reducing cancer symptoms and improving physical function. Breathing exercises, fatty acid supplements, and dietary advice with calorie supplements were noted to be less effective. There were significant limitations with this review as there is limited literature on adjuvant interventions that satisfied the exclusion criteria of this study. The findings from this review will improve our understanding of how to better address the symptom control needs for patients with cancer using complementary and integrative medicine.

LAY SUMMARY

1. Patients with lung cancer may benefit from complementary and integrative medicine to address significant side-effects after chemoradiation, such as pain, fatigue, and overall decreased quality of life.
2. Mind-body exercises are most likely to be beneficial as they were most effective at reducing cancer symptoms and improving physical function.
3. Breathing exercises, fatty acid supplements, and dietary advice with calorie supplements were noted to be less effective at symptom control.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Multionics profiling of apheresis and CAR T cells - Poster #99

Presenter: Evelyn Teh, National Research Council of Canada

Other Authors: Susanne Penny, Kenneth Chisholm, Natasha Kekre, Kevin Hay, Brad Nelson, Robert Holt, Rob Laister, Samuel Saibil, Devanand Pinto

ABSTRACT_ID#3281

ABSTRACT

aCD19 chimeric antigen receptor (CAR) T cell therapy is highly efficacious for B-cell malignancies, but 30-60% of patients eventually relapse. Presently, parameters such as T-cell phenotypes and polyfunctionality, metabolic features (OXPHOS vs glycolysis) and in-vitro expansion during manufacturing used to predict CAR-T efficacy and persistence are generated during or post manufacturing of CAR-T cells. However, to maximize the cost of effectiveness of the therapy, it would be more desirable to have predictive biomarkers of response and survival before CAR-T cell manufacturing such as the apheresis material used for CAR-T production. To this end, we performed metabolomics and proteomics of bulk apheresis and post-manufactured CART cells from 30 patients with B-cell malignancies enrolled in the CD-19 directed CAR-T trial (CLIC-01) to identify correlates of response and understand how the manufacturing processes alter the immune-metabolome and impact T-cell fitness. Metabolomics of the bulk apheresis showed elevated acylcarnitines in non-responders, highlighting mitochondria dysfunction and a potential marker for T-cell exhaustion. Proteomics of CAR-T cells post production further revealed an increase in large neutral amino acid transporters, and increased nucleotide synthesis pathway suggesting increased T-cell proliferation in non-responders. An exhausted cell phenotype with proliferative capabilities but lacking effector function hints at a subset of T-cells known as precursor exhausted T-cell (Tpex). Furthermore, production of CAR-T cells in a replete environment failed to rescue these cells in non-responders. Targeted metabolic interventions during production could potentially unlock both proliferation and effector function and warrants further investigation.

LAY SUMMARY

T-cells can adjust how they use energy and nutrients, which helps them survive and function better in changing environments. Adjusting the nutrients and conditions during T-cell production might be crucial for boosting and improving their health and performance.

Cancer Clinical Research and Trials

Monday, November 3, 2025

Integrating the Patient Voice in Early Training for the Next Generation of Scientists - Poster #100

Presenter: Megan Sewell, Independent

Other Authors: Sonia del Rincon

ABSTRACT_ID#2476

ABSTRACT

Integrating the patient voice in cancer research is a transformative approach that fosters collaboration between researchers and individuals directly impacted by the disease. This inclusive model enhances research quality, ensures relevance to real-world needs, and promotes mutual learning. The Patient Partner Engagement Day at the Lady Davis Institute exemplifies this approach by creating a space for dialogue and relationship-building between researchers and patient partners. Similarly, the CIHR “Bringing Biology to Cancer Survivorship” workshop highlighted the importance of aligning research with survivorship needs, blending scientific knowledge with lived experiences for more impactful outcomes. Patient engagement fosters empowerment and a sense of value, while also promoting more accurate and meaningful research. Early involvement helps build trust and lays the groundwork for sustainable, long-term collaboration. Events like the 13th Annual RRCancer Symposium provide vital platforms for networking and shared learning, with contributions from patient partners such as Megan, whose candid interactions with research trainees bridged the gap between lab science and human impact. At McGill University, the Integrated Cancer Science Program (ICSP) is embedding patient-centered principles into early scientific education. This ensures that future researchers are equipped with a deep understanding of patient perspectives, ethics, and collaborative practices. Looking ahead, cancer research will increasingly move toward co-leadership with patient partners, driving innovative methodologies and more personalized treatments. The integration of patient voices not only strengthens research design but also improves healthcare outcomes, reinforcing the critical role of patients as equal contributors in the scientific process.

LAY SUMMARY

1. **Holistic Research Grounded in Lived Experience** Integrating the patient voice creates a more holistic research model, one that goes beyond biology to include emotional, social, and practical dimensions of the cancer journey. This ensures that research is not only scientifically sound but also deeply relevant to patients’ real-world challenges.
2. **Trust Through Early, Meaningful Engagement** Early and continuous involvement of patient partners builds trust and long-term collaboration, transforming research into a shared journey. Dialogues at events like engagement days and symposiums help bridge the gap between scientific inquiry and the human impact of cancer.
3. **Advancing Survivorship-Centered Innovation** By focusing on survivorship and embedding patient-centered thinking into scientific training, like in McGill’s ICSP, researchers are being prepared to co-create solutions that prioritize quality of life, long-term outcomes, and personalized care.

Translating Trials to Practice: A Real World Analysis of Effectiveness and Safety of Neoadjuvant Chemoimmunotherapy in Resectable Non-Small Cell Lung Cancer - Poster #101

Presenter: Siddhartha Goutam, University of Calgary

Other Authors: Michelle Dean, Amanda JW Gibson, Vishal Navani

ABSTRACT_ID#2832

ABSTRACT

Introduction: Neoadjuvant chemoimmunotherapy (NCIT) has proven effective in improving overall survival (OS) in patients with resectable non-small cell lung cancer (NSCLC) in pivotal phase III clinical trials. The extent to which trial outcomes reflect real world experience is unknown. We aimed to characterize the complete (PCR) and major pathologic response (MPR) rates of patients treated with NCIT, quantify NCIT completion, adverse events and surgical outcomes of patients treated in routine practice. **Methods:** We utilized the Glans-Look Lung Cancer Research Repository (GLR), a database of all patients with lung cancer in Alberta, Canada. Demographic, clinical, treatment and outcomes data were extracted from the GLR and characterized using univariate methods. **Results:** 64 patients initiated NCIT, in whom median age was 64. 36(56%) were of male sex and 61(95%) were ECOG ≤ 1 . 25(43%) had PD-L1 $> 50\%$ and 18(28.1%) had a tier-1 mutation. 57 (89.0%) completed all prescribed cycles, and 55 (85.9%) proceeded to surgical completion. The objective response rate at pre-operative CT was 70.3%. 54 (98.1%) patients had R0 margins. 19 (34.5%) patients had PCR, and 28 (50.9%) of patients had MPR. 23 (35.9%) of patients had serious adverse events ($\geq$ Grade 3, or requiring ED visit/hospitalization/treatment delay). Operative complications occurred in 20 patients, most commonly air leak/pneumothorax (6), atrial dysrhythmia (4), and kidney injury (2), with median hospital stay of 6 days. **Conclusion:** NCIT is similarly effective and safe in a real world cohort of patients compared to the population in Checkmate 816. Incidence of surgical complications and complications mimicked trial data.

LAY SUMMARY

1. The combination of chemo- and immunotherapy administered preoperatively has proven effective in improving surgical outcomes and overall survival in patients with surgically resectable non-small cell lung cancer. Our study showed that 1/4 patients achieved complete response (i.e. no visible cancer cells in resected tumor specimen) after chemo-immunotherapy prior to surgery.
2. Patients developed toxicities of systemic therapy and surgical complications, however the rate of these were similar to what was described in the trial population.
3. Our findings suggest that the combination chemoimmunotherapy in a real cohort of patients in Alberta, Canada has similar efficacy in achieving tumor killing, and safety compared to the pivotal clinical trials. However we have yet to characterize how long our patients survive, which will require a longer duration of follow up.

Connecting Clinical Trials with Patients using Patient Navigation: A Scoping Review - Poster #102

Presenter: Olla Hilal, Western University

Other Authors: Yvonne Paglicauan, Ria Patel, Pratham Gupta, Leena Moshref, Nicole Askin, Carla Epp, Anthony Luginaah, Renee Nassar, Roaa Hirmiz, Milica Paunic, Mahmoud Hossami, Rhonda Abdel-Nabi, Kayla Touma, Depen Sharma, Rija Fatima, Emmanuel Akingbade, Ibrahim Mohamed, Swati Kalia, Michael Touma, Govana Sadik, Laurice Arayan, Salah Alhajsaleh, Anaam Jaet, Christina Trieu, Megan Delisle, Caroline Hamm

ABSTRACT_ID#2756

ABSTRACT

Introduction: Patient navigation is a promising strategy to improve access to cancer care, but its role in increasing access to cancer clinical trials has not been systematically evaluated. This scoping review aims to critically appraise, synthesize, and present the available evidence on the use of patient navigation to increase cancer clinical trial enrollment. **Methods:** Nine databases were searched for English peer-reviewed articles from inception through December 21, 2023, and the search was updated on March 5, 2025. Two independent researchers screened titles, abstracts, and full texts and extracted data using standardized forms. **Results:** Among the 23 included articles, 18 (78.3%) were observational studies and 5 (21.7%) were randomized trials. Thirteen (56.5%) focused on equity, all addressing racial/ethnic groups. Seven (30.4%) articles used patient navigation for clinical trials for all cancer types; 14 (60.9%) focused on specific cancers, with 12 (85.7%) primarily addressing breast cancer. Among 21 studies describing navigator qualifications, 4 (17.4%) required professional training, while 17 (73.9%) used community representatives. The interventions used most frequently by navigators included education in 19 articles (82.6%) and care coordination in 17 articles (73.9%). Direct clinical trial referrals were unmentioned; logistical and financial assistance appeared in only 2 articles each (8.7%). In 7 (30.4%) studies, navigators directed patients to trials within and outside their center; 16 (69.6%) navigated patients only within their center. **Conclusions:** Future research should employ more rigorous designs to evaluate different patient navigation approaches, assessing their impact on clinical trial enrollment across a wider range of cancers and patient populations.

LAY SUMMARY

- **Less patient navigation interventions focused on all cancer types:** Majority of existing patient navigation interventions focused on connecting patients with clinical trials with a specific type of cancer rather than considering patients with any cancer. This demonstrates the difficulty for patients with other types of cancers that may not be considered in patient navigation interventions to be connected to a clinical trial.
- **Lack of diversity in types of navigation interventions used:** The most common interventions used by patient navigators were surrounding educating patients and care coordination (e.g., facilitating linkages to other services). However, existing patient navigation interventions demonstrate the lack of focus on financial assistance and logistical assistance. Therefore, patient navigation programs should investigate expanding their program to consider other navigation interventions that patients may find beneficial.
- **Equity limitations:** Ten (43.5%) articles did not focus on any form of equity, diversity, or inclusion characteristics. Of the articles that did (56.5%), they only focused on racial/ethnic groups, demonstrating the need to examine a wider range of patient populations.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Impact of Clinical Trial Navigators on Clinical Trial Accrual through Multidisciplinary Case Conferences: A Pre- & Post-Implementation Study - Poster #103

Presenter: Anaam Jaet, Windsor Regional Hospital

Other Authors: Christina Trieu, Salah Alhajsaleh, Swati Kalia, Renee Nassar, Mahmoud Hossami, Laurice Togonon Arayan, Depen Sharma, Roaa Hirmiz, Milica Paunic, Olla Hilal, Anthony Luginaah, Michael Tuma, Govana Sadik, Ria Patel, Pratham Gupta, Megan Delisle, Lee McGrath, Caroline Hamm

ABSTRACT_ID#2409

ABSTRACT

Clinical trial accrual improves cancer outcomes but remains limited due to systemic barriers, including lack of awareness, time constraints, and inadequate referral processes, with only ~3% of adult cancer patients participating. Integrating Clinical Trial Navigators (CTNs), who provide tailored trial recommendations based on treatment history and eligibility, into Multidisciplinary Case Conferences (MCCs) has shown potential to enhance trial discussions, increase referrals, and streamline enrollment. This study evaluates the impact of CTNs on trial accrual, implementation effectiveness, and workflow optimization. We hypothesize that CTN integration into MCCs will increase referral and accrual rates while improving clinical trial integration efficiency. This hybrid effectiveness-implementation study focuses on breast, glioblastoma, and colorectal cancers at the Windsor Cancer Centre. Baseline referral and enrollment rates are established through observational chart reviews, while CTNs use updated Master Lists and the “Look Up Trials” app during MCCs to identify trial options. Surveys and structured interviews using the Consolidated Framework for Implementation Research (CFIR) and a “Think Aloud” process provide qualitative feedback on barriers, facilitators, and workflow changes. Data are analyzed using descriptive statistics and thematic analysis. In phase I of the project, 24% of patients discussed in the MCCs had potential trials. Anticipated outcomes from prior studies include a 25% referral rate and an 8% accrual rate among ~168 patients and ~60 physicians. Secondary outcomes target iterative process improvements and evaluate the app’s effectiveness in streamlining trial matching. Preliminary feedback suggests potential for enhanced physician satisfaction and optimized workflows. Completed results are expected by time of conference.

LAY SUMMARY

More Patients Matched to Clinical Trials: When Clinical Trial Navigators (CTNs) join tumor boards or what’s known as Multidisciplinary Case Conferences (MCCs), where a team of oncologists, pathologists, radiologists and other healthcare professionals meet to discuss cancer cases. They help doctors quickly find clinical trials that might fit a patient’s treatment plan. In early results, nearly 1 in 4 patients discussed had a potential trial option. **Better Referrals and Enrollment:** By using tools like updated trial lists and a “Look Up Trials” app, CTNs are expected to increase the number of patients referred to—and actually joining—clinical trials. The goal is to reach a 25% referral rate and 8% enrollment rate, improving access to cutting-edge treatment options. **Improved Workflow and Physician Satisfaction:** Doctors and healthcare teams reported that having a CTN present made discussions more efficient and helped reduce the time and effort needed to match patients with trials. Early feedback also suggests it makes the process more satisfying and effective for everyone involved. These insights show promise in making cancer clinical trials more accessible, which can lead to better outcomes for patients and more efficient healthcare delivery.

Cancer Clinical Research and Trials

Monday, November 3, 2025

Sponsor Implementation of Decentralized Clinical Trial Elements to Improve Access to Clinical Trials: The Canadian Cancer Trials Group Experience - Poster #104

Presenter: Patti OBrien, Canadian Cancer Trials Group

Other Authors: Dora Nomikos, Michelle Savoie, Melanie Leitch, Scott Wilkes, Alison Urton, Janet Dancey

ABSTRACT_ID#2908

ABSTRACT

The Canadian Cancer Trials Group (CCTG) is adopting decentralized clinical trial (DCT) strategies to reduce participation burdens, expand access and reduce carbon footprint by enabling some or all trial activities outside traditional sites. DCTs support more equitable and representative participation, improve trial efficiency, and may accelerate recruitment. CCTG is implementing DCT elements while ensuring compliance with regulations and alignment with Health Canada's position on DCTs, the FDA guidance, and input from clinical research partners in Canada and the U.S. CCTG's DCT strategies include: 1) Embedding DCT elements in protocols (e.g. remote consent, virtual visits, off-site standard of care (SOC) procedures, decentralized drug delivery); 2) Defining when non-SOC activities can occur off-site; 3) Standardizing site inquiries with a centralized DCT request form; 4) Applying a decision matrix to assess participant-specific scenarios; and 5) Providing site guidance to maintain oversight and trial integrity. To date, CCTG has sponsored over 20 trials with DCT participation and is receiving an increasing number of site requests to conduct trials with DCT elements. Lessons learned from leading DCTs have informed CCTG's current approach to trial conduct. With partners and stakeholders, CCTG will continue to refine and expand its DCT framework to increase accessibility and equity in its clinical trials and assess impact on participation of patients from rural/remote and traditionally underrepresented groups. CCTG is a National Program of the Canadian Cancer Society (grant number: 707213).

LAY SUMMARY

1. The Canadian Cancer Trials Group (CCTG) is improving trial access and reducing participant burden by implementing decentralized clinical trial (DCT) strategies while upholding sponsor oversight and regulatory compliance.
2. DCT elements integrated into trial protocols include remote consent, virtual visits, SOC procedures and follow-up by local providers, and decentralized drug delivery.
3. CCTG supports sites with a standardized inquiry process, a decision matrix to evaluate DCT requests, and practical guidance to ensure consistent and compliant implementation.

Cancer Clinical Research and Trials

Monday, November 3, 2025

Characterizing Patient-Tailored Gene Signatures at each Clinical Stage of Prostate Cancer as Precision Oncology - Poster #106

Presenter: Priyankana Banerjee, McGill University Health Center - Research Institute at Glen

Other Authors: Seta Derderian, Alexis Rompré-Brodeur, Rafael Sanchez-Salas, James Tsui, Rajan Raghu, Aprikian Armen, Simone Chevalier

ABSTRACT_ID#3289

ABSTRACT

Prostate Cancer (PCa) harbours inter-patient and intra-tumoral heterogeneity challenging risk stratification and optimal disease management. In addition to blood PSA, multiparametric-MRI-biopsy provides subjective initial screening. However, lack of specificity fails to overcome undertreatment and overtreatment of indolent disease. Most low-risk cases are managed by active surveillance and/or focal therapy, while intermediate to high-risk cases require curative therapies involving radical prostatectomy and/or radiation therapy, often combined with androgen-deprivation therapy (ADT). About 25-35% of patients experience recurrence and require ADT for sensitive (HSPC) until progression to metastases (m), followed by 2nd generation Androgen Receptor Inhibitors and taxanes for mHSPC and mcastrate-resistant (mCRPC), respectively. Molecular testing using liquid biopsies has advanced as a modality to identify both prognostic and predictive biomarkers. Amongst circulating bio-analytes explored, little is known on blood gene transcripts (mRNA) profiling and extracellular vesicles (EVs) signatures to monitor PCa treatment response/resistance and lethality. We hypothesize that comparative clinical characterization of such signatures during the disease trajectory will open new possibilities to offer patient-tailored therapeutic options. The host lab recently tested 57 genes representing cell subtypes, drug resistance, and drug targets in diverse categories of patients, and confirmed clinical significance and high inter-patient heterogeneity. We are optimizing multiplex double digital PCR for most promising genes in blood RNA and EVs from patients at different clinical phases. This precision oncology approach will identify novel candidates for new therapeutic modalities, establish if existing drugs could be part of clinical trials to better stratify patients, customize disease management and impact on death from PCa.

LAY SUMMARY

Prostate cancer (PCa) is a major cause of cancer deaths worldwide, ranking 3rd in Canadian men. The precision-oncology based project provides the following key insights on prostate cancer management as the patients grow old with the disease. Measuring response to treatment strategies is crucial to better managing the progression of cancer. In this project, we analyze the over-expression of mRNA in patient blood as a factor for determining a) treatment sensitivity, b) establishing resistance towards the current treatment, c) progressing towards metastases and, d) new targets for prostate specific drug-development. The project also introduces mRNA and extracellular vesicles (containing mRNA and proteins) as potential blood-biomarkers in PCa patients, representing the different cell subtypes that contribute to the aggressiveness of the disease as well as patient-to-patient heterogeneity. Establishing patient-specific prognostic and predictive biomarkers in the blood is a non-invasive method of diagnosis and follow-up testing for prostate cancer patients. The panel of mRNA in the blood correlates with the dynamics of cellular and systemic response towards existing treatment modalities.

Cancer Clinical Research and Trials

Monday, November 3, 2025

Barriers and Facilitators to Participation in Cancer Clinical Trials: Perspectives from South Asian Communities - Poster #108

Presenter: Patil Mksyartinian, Colorectal Cancer Canada

Other Authors: Iris Karry, Natasha Bassett-Saltarelli, Arun Garg

ABSTRACT_ID#2871

ABSTRACT

Cancer clinical trials are essential for discovering better ways to prevent, find, treat and manage cancer. In Canada, less than 5% of cancer patients participate in clinical trials, with rates as low as 1% in some provinces. Ethnic minority populations remain underrepresented in trials, highlighting an important gap that must be addressed for scientific advances in health to be benefitted by all populations. This quality improvement study aimed to identify the barriers and facilitators influencing trial participation among South Asian Canadians, who represent the country's largest minor ethnic group (7.1% of the population). From January to May 2025, Colorectal Cancer Canada collaborated with the South Asian Health Institute to conduct fifteen semi-structured interviews with South Asian individuals to explore their perceptions of cancer clinical trials. The qualitative data from interviews is currently undergoing a thematic analysis to identify key themes. Ongoing analysis will guide the co-development of culturally tailored materials and outreach strategies, in collaboration with South Asian community representatives, to raise awareness, build trust, and strive to ultimately increase cancer clinical trial participation. This work is an important step toward fostering more inclusive health research through meaningful community engagement and collaboration.

LAY SUMMARY

1. **Misconceptions and Personal Experience Shape Individual Perspectives and Health Decisions** The study participants expressed limited awareness of cancer clinical trials, and misconceptions were common. Their perspectives on cancer and cancer clinical trials were shaped by sociocultural beliefs and personal and family experiences, which strongly influenced their health decision-making. These findings highlight the need for clear, tailored, and culturally relevant information that speaks directly to people's real concerns about cancer and clinical trials.
2. **Family and Community Networks Are Key Influencers in Health Navigation** The study participants shared that health decisions are often made with input from family members. Outreach efforts that involve families, respect cultural values, and use trusted community messengers, rather than focusing only on individuals, can help build trust, support shared decision-making, and improve participation.
3. **Trusted Community Channels Help Reach Underserved Populations** Study participants indicated that sharing information through familiar community spaces, such as places of worship, cultural centers, and local gathering places, and trusted communication channels like community radio, local leaders, and storytelling by community members can effectively reach individuals who may not typically engage with the healthcare system. These approaches help build trust and make information about cancer and clinical trials more accessible.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Therapeutic Clinical Trial Enrollment in Children with Cancer in Canada: An Analysis of CYP-C Data - Poster #109

Presenter: Randy Barber, C17 Council

Other Authors: Vera Grywacheski, Lin Xie, Parisa Khodabandehloo, Samina Aziz, Mylene Frechette, Katherine McKenzie, Christina Ricci, Nicole Winch, Alex Airhart, Paul Gibson

ABSTRACT_ID#3060

ABSTRACT

Background In Canada, nearly 1,000 children under age 15 are diagnosed with cancer annually. While five-year survival rates now exceed 86%, ongoing progress depends on access to therapeutic clinical trials. These trials aim to advance treatment efficacy and reduce treatment-related harm, yet enrollment remains limited for many children. **Methods** Using data from the Cancer in Young People in Canada (CYP-C) program, this poster describes enrollment in therapeutic clinical trials for initial cancer treatment among children diagnosed between 2001 and 2021. Analyses include children under age 15 with complete treatment plan information and exclude relapsed or unclassifiable ICCC-3 cases. **Results** Overall enrollment for initial treatment varied by cancer type: 48% of children with leukemia enrolled on a trial, compared to 14% with non-central nervous system (non-CNS) solid tumours, 13% with lymphoma, and 7% with CNS tumours. Over time, enrollment across all cancer types declined, though leukemia enrollment showed some rebound in 2020–2021. Regional disparities were evident with the highest enrollment across all cancer types observed in Quebec. Among non-enrolled children, the most common reason was no available trial; this was reported for 62% of leukemia, 70% of lymphoma, 63% of non-CNS solid tumour, and 50% of CNS tumour cases. **Conclusions** This analysis highlights variation in trial enrollment by cancer type, time, and region. Clinical trials are important to improving outcomes for children with cancer, and ongoing surveillance of their availability and enrollment is essential to ensure equitable access and maximize their impact.

LAY SUMMARY

Enrollment in clinical trials has declined over time across most cancer types, with some rebound for leukemia observed in 2020–2021. There are regional differences in enrollment, with children in Quebec more likely to enroll in clinical trials than those in other provinces, highlighting inequities in access to research opportunities. The main reason children were not enrolled was the lack of available trials, underscoring the need to expand trial availability to improve equitable access.

Adjuvant chemotherapy outcomes among patients with stage II early-onset colon cancer in Alberta - Poster #111

Presenter: Robert B. Basmadjian, University of Calgary

Other Authors: Robert B. Basmadjian, Dylan E. O'Sullivan, Tamer N. Jarada, Winson Y. Cheung, Patricia A. Tang, Sharlene Gill, Safiya Karim, Robert Hilsden, Colleen Ann Cuthbert, Khara Sauro, Joon Lee, Christie Farrer, Barry D. Stein, Darren R. Brenner

ABSTRACT_ID#2890

ABSTRACT

Background: Early-onset colorectal cancer (eoCRC) among adults under 50 years of age are underrepresented in randomized trials of novel therapies. Consequently, evidence to inform treatment, particularly the appropriateness of adjuvant chemotherapy in stage II colon cancer, remains limited and controversial. Objective: To evaluate the real-world effectiveness of initiating adjuvant chemotherapy vs. observation on recurrence-free (RFS) and overall survival (OS) in stage II eoCRC in Alberta, Canada. Methods: We conducted a population-based, retrospective cohort study including all patients aged 18 to 49 years in Alberta diagnosed with invasive colon cancer (2004-2020). Observational data were used to emulate a target trial comparing RFS and OS between patients initiating adjuvant chemotherapy (fluorouracil/capecitabine-based) within 12 weeks of surgery vs. observation (no chemotherapy). Follow-up began at surgery and continued until recurrence, death, last healthcare contact, or administrative end of follow-up (April 2022). Marginal Cox models with inverse-probability censoring weights and artificial cloning were employed to estimate hazard ratios (HR) and 95% confidence intervals (95% CI). Results: Among 230 patients, 94 (41%) initiated chemotherapy. Patients initiating chemotherapy were more likely to have T4 and high grade tumours, and fewer than 12 examined lymph nodes. The risks of recurrence and death were 21% (HR:0.79; 95%CI:0.41-1.51) and 20% (HR:0.80; 95%CI:0.40-1.61) lower among those who initiated chemotherapy versus observation, but statistical significance was not achieved. Conclusion: Our study was limited in sample size, outcome events, and data regarding microsatellite instability. Future studies should explore prediction of high-risk status to identify patients more likely to benefit from adjuvant chemotherapy.

LAY SUMMARY

Key Messages:

- Some younger adults with stage II colon cancer receive chemotherapy after surgery, but its benefit is unclear. In this Alberta-based study, 41% of early-onset patients received chemotherapy, often those with higher-risk tumor features.
- Patients who received chemotherapy had modestly better outcomes, but the benefit was uncertain. While those treated with chemotherapy had a slightly lower risk of recurrence or death, the differences were not statistically significant.
- More research is needed to identify which younger patients truly benefit from chemotherapy. Current treatment decisions may be influenced by age or tumour characteristics without strong supporting evidence. Larger studies and better risk prediction tools are needed to avoid unnecessary treatment and guide care more precisely.

Impact de l'estradiol dans le microenvironnement immunitaire du cancer de la vessie - Poster #112

Presenter: Geneviève Trépanier, CRCHU de Québec - Université Laval

Other Authors: Typhaine Gris, Rose Lemelin, Paul Toren

ABSTRACT_ID#2536

ABSTRACT

Le cancer de la vessie (CV) est le 7^e cancer le plus fréquent au Canada. L'immunothérapie par instillation vésicale de BCG est utilisée pour traiter le cancer de la vessie non infiltrant le muscle (CVNIM), mais les récurrences restent fréquentes, nécessitant de nouvelles stratégies pour améliorer la réponse au BCG. Les influences hormonales sur la réponse immunitaire au CV semblent expliquer les différences d'incidence et de pronostic entre sexes : l'incidence est 3 à 4 fois plus élevée chez les hommes, tandis que les femmes présentent des tumeurs plus agressives et une réponse au BCG plus faible, sans différence liée au sexe pour la chimiothérapie. Les stéroïdes sexuels jouent un rôle dans ces différences immunologiques sexuelles mais leur impact sur le microenvironnement immunitaire tumoral (TIME) et la réponse au BCG reste à préciser. Nous avons étudié l'effet de l'estradiol sur le TIME du CV en utilisant notre modèle murin MBT-2 publié, traité ou non par BCG, chez des souris mâles castrées et femelles ovariectomisées. L'estradiol augmente la croissance tumorale et le volume tumoral au moment du sacrifice chez les deux sexes. Les analyses de cytométrie en flux du TIME et du sang ont révélé une augmentation de l'infiltration des cellules immunitaires dans les tumeurs, mais avec une activité diminuée, favorisant un cancer plus agressif et une moindre réponse au traitement. Notre objectif est de développer des stratégies exploitant ces différences hormonales pour améliorer l'efficacité de l'immunothérapie du cancer de la vessie.

LAY SUMMARY

Le cancer de la vessie est plus agressif chez les femmes et elles répondent moins bien à l'immunothérapie par le BCG. Nous cherchons à comprendre le rôle des hormones sexuelles sur le développement du cancer de la vessie. Nous avons montré que l'estradiol augmentait la croissance tumorale et l'infiltration de cellules immunitaires non fonctionnelles quel que soit le sexe biologique des souris.

The addition of monoclonal antibodies to chemotherapy in metastatic early-onset colorectal cancer – a comparative effectiveness study using real-world data - Poster #113

Presenter: Robert B. Basmadjian, University of Calgary

Other Authors: Robert B. Basmadjian, Dylan E. O'Sullivan, Tamer N. Jarada, Winson Y. Cheung, Patricia A. Tang, Sharlene Gill, Safiya Karim, Robert Hilsden, Colleen Ann Cuthbert, Khara Sauro, Joon Lee, Christie Farrer, Barry D. Stein, Darren R. Brenner

ABSTRACT_ID#2894

ABSTRACT

Background: Early-onset colorectal cancer (eoCRC) among adults under age 50 are underrepresented in randomized trials of novel therapies. The benefit of adding monoclonal antibodies (MAB) to chemotherapy for stage IV disease remains uncertain, with limited knowledge on factors beyond RAS genes that influence treatment response. **Objective:** To assess the real-world effectiveness of initiating first-line combination chemotherapy vs. combination chemotherapy plus MAB on overall survival (OS) in stage IV eoCRC in Alberta, Canada. **Methods:** We conducted a population-based, retrospective cohort study including all patients aged 18 to 49 years in Alberta diagnosed with metastatic CRC (2004-2020). Observational data were used to emulate a target trial comparing OS between patients initiating combination chemotherapy (fluorouracil/capecitabine plus oxaliplatin) vs. combination chemotherapy plus MAB (bevacizumab/cetuximab/panitumumab) within 16 weeks of diagnosis. Follow-up began at diagnosis and continued until death, last healthcare contact, or administrative end of follow-up (April 2022). Marginal Cox models with inverse-probability censoring weights and artificial cloning were employed to estimate hazard ratios (HR) and 95% confidence intervals (95% CI). **Results:** Among 674 patients, 146 (22%) received chemotherapy plus MAB. These patients were more likely to have proximal tumours and less likely to undergo surgery or radiation. The risk of death was 5% higher (HR: 1.05; 95% CI: 0.88–1.26) in the MAB group compared to chemotherapy alone, though not statistically significant. **Conclusion:** We did not observe survival benefits of initiating first-line MAB in stage IV eoCRC. This may reflect treatment compliance, subsequent treatment lines, and missing RAS data, a key predictor of treatment response.

LAY SUMMARY

Key Messages:

- Real-world data showed no clear survival benefit from adding monoclonal antibody (MAB) therapies to standard chemotherapy for patients under 50 with stage IV colorectal cancer. While increasingly used and contemporary, our study found no significant difference in survival between patients who received MABs and those who did not.
- Patients who received MABs differed in important ways: they were more likely to have proximal tumours and less likely to undergo surgery or radiation. These differences suggest MABs in Alberta are more often initiated when prognosis is unfavourable and surgery is not an option or preferred.
- Lack of key molecular data limits interpretation. We could not account for RAS gene status, which are known to predict how well MABs work. Without this information, it is difficult to determine whether the absence of benefit was due to treatment ineffectiveness or poor selection of patients likely to respond.

Cancer Clinical Research and Trials

Monday, November 3, 2025

A Path Forward: Advancing Canada's clinical trials ecosystem through the development of a cancer clinical trials action plan - Poster #114

Presenter: Vanessa Silano, Canadian Cancer Society

Other Authors: Helena Sonea, Don Wood, Heather Mulligan, Laura Orsini, Stephen Piazza, Elizabeth Holmes, Tracy Torchetti, Stuart Edmonds

ABSTRACT_ID#3240

ABSTRACT

The Canadian Cancer Society's (CCS) Cancer Clinical Trials Action Plan (CTAP) identifies ways to build a coordinated, sustainable and inclusive national cancer clinical trials ecosystem that improves access for all Canadians. The CTAP captures voices from across the country and aligns outreach efforts for various champions. From May 2024 to February 2025, CCS facilitated a series of forums to consult with individuals across the cancer community, including people with cancer and caregivers, to address pressing challenges in clinical trials. Each forum focused on key themes including best practices and standardized tools, legislative and regulatory opportunities, building capacity, and funding. After each forum, CCS synthesized input, shared it for validation, and conducted a thematic analysis of all forum content. Perspectives aligned strongly across sectors. A literature review and summarized recommendations from previous reports further informed this process. Seven priority areas were identified: 1) Make cancer clinical trials accessible to everyone in Canada; 2) Streamline regulatory and administrative processes for cancer clinical trials; 3) Enhance public awareness about cancer clinical trials; 4) Improve healthcare workforce challenges; 5) Transform cancer clinical trials through integrated data collection and coordination; 6) Improve Canada's position as a global centre of excellence for cancer clinical trials; and 7) Establish robust and sustainable clinical trials funding in Canada. Actioning these priority areas will require champions across at least 7 sectors. Future efforts will focus on implementing the CTAP through assigning outcomes and establishing timelines for each priority area.

LAY SUMMARY

Clinical trials are essential for advancing medical knowledge, improving patient care, and can offer hope when standard treatments are no longer viable. Yet in Canada, people with cancer and their families often face barriers when accessing clinical trials. The Cancer Clinical Trials Action Plan was developed to give clinical trials stakeholders a vision for a national cancer clinical trials system in Canada. Engaging stakeholders in the development of the Cancer Clinical Trials Action Plan ensures that the perspectives and voices of individuals from across the cancer community, including people with cancer and caregivers, are meaningfully integrated, helping to create a more equitable and accessible cancer clinical trials ecosystem in Canada.

Cancer Clinical Research and Trials

Sunday, November 2, 2025

Geographic Patterns of Breast Cancer Clinical Trial Enrollment in Canada: An Analysis of CCTG-Registered Trials - Poster #115

Presenter: Robert Olson, UBC and BC Cancer

Other Authors: Curtis Leclerc

ABSTRACT_ID#2382

ABSTRACT

Objective: To understand the geographical landscape of Canadian clinical trials using a breast cancer tumour site model.

Methods: We conducted a retrospective observational analysis of 7,005 participants enrolled in 16 CCTG-registered breast cancer clinical trials between 2000 and 2017. Participant accrual was assessed by province, city, and major cancer center. Census Metropolitan Areas (CMAs), as defined by Statistics Canada, were used to identify urban recruitment patterns. Data were visualized using geographic information systems and analyzed descriptively to assess regional differences. **Result:** Clinical trial recruitment was highly centralized. 99.9% of participants were enrolled from CMA-designated urban centers, with only three patients recruited from non-CMA regions. Ontario, Quebec, and British Columbia accounted for over 80% of national accrual, with Ontario alone contributing 52.6%. The majority of trial enrollments originated from a small number of large urban academic centers, with minimal participation from rural or remote regions and none from the territories.

Conclusion: Breast cancer clinical trial enrollment in Canada is highly concentrated in urban academic centers, leaving rural, remote, and northern populations significantly underrepresented. These findings underscore the urgent need for decentralized trial models, regional recruitment networks, and targeted investments in rural research infrastructure to ensure equitable access to clinical research across Canada.

LAY SUMMARY

Breast cancer clinical trials in Canada are overwhelmingly centralized: over 99% of participants in national trials were enrolled at large urban cancer centers, with nearly no accrual from semi-urban or rural centres. Trial enrollment is highly uneven across provinces. Ontario, Quebec, and British Columbia accounted for more than 80% of national participants, while some provinces and all three territories had no trial enrollees at all. Now is a key opportunity to expand access to clinical trials. Our findings support the urgent need to engage funders and trialists in advancing decentralized models (such as virtual enrollment and follow-up), to ensure all Canadians can participate in cancer research, regardless of geography.

Survival outcomes for patients with extensive stage small cell lung cancer (ES- SCLC) treated with consolidative thoracic radiation therapy (cTRT) and chemoimmunotherapy (CIT): a population level study -

Poster #116

Presenter: Yizhuo Kelly Gao, University of Calgary

Other Authors: Amanda Willams Gibson, Michelle Liane Dean, Marc Kerba, Vishal Navani

ABSTRACT_ID#2915

ABSTRACT

Background: A synergistic effect of cTRT+CIT has been theorized to enhance survival outcomes in patients with ES-SCLC. We aims to investigate the safety and effectiveness of patients with ES-SCLC treated with CIT+cTRT in routine clinical practice. **Methods:** We identified a cohort of ES-SCLC patients treated with CIT+cTRT from Sept 2019 to Nov 2023, using a province-wide lung cancer registry, from which baseline, treatment, and outcome information were derived. **Results:** 50 patients were included. At CIT initiation, 34 patients had ECOG $\leq$ 1 and 38 had baseline brain metastasis. 35 patients received durvalumab, 11 atezolizumab, 4 alternate immune checkpoint inhibitors (ICI). Median time from CIT initiation to cTRT was 4.7 months. Total dose of cTRT varied, with most patients (n= 23) receiving 30Gy in 10 fractions. The incidence of post-cTRT pneumonitis was n=9, induced by radiation (n=4), ICI (n=3) or mixed etiology (n=2); the majority were low grade (n=8) and managed with outpatient steroids, only 1 patient had grade 3 pneumonitis required hospitalization. Median OS and PFS post-CIT initiation were 21.3 and 11.7 months. Baseline brain metastasis was associated with both worse OS (HR = 3.0, p = 0.015) and PFS (HR = 2.9, p = 0.008). The type of ICI, atezolizumab vs durvalumab, did not significantly impact OS or PFS. **Conclusion:** We observed a high incidence of pneumonitis (18%) in this cohort of ES-SCLC patients receiving CIT+cTRT. Most of our pneumonitis cases were low-grade and did not require hospitalization. Overall, CIT+cTRT appears to be well tolerated in this cohort of real world patients.

LAY SUMMARY

Our study observed a high incidence of pneumonitis (18%) in this largest North American cohort of real-life ES-SCLC patients receiving CIT + cTRT. Most of our pneumonitis cases were low-grade and did not require hospitalization. Overall, CIT + cTRT appears to be well tolerated in this cohort of real world patients.

Cancer Risk and Prevention

Monday, November 3, 2025

Investigating the Biological Impact of Wildfire Smoke on Lung Cancer Risk Using a Fruit Fly Model System - Poster #122

Presenter: Nawanjani Pathinayake, Queens University

Other Authors: Maria Aristizabal, Benedikt Gregor

ABSTRACT_ID#3017

ABSTRACT

Wildfire smoke is an emerging health threat that contains harmful substances particulate matter (PM2.5, PM10), gases (CO, SO₂), and polycyclic aromatic hydrocarbons, that are known or suspected carcinogens. Nevertheless, how wildfire smoke contributes to cancer biology remains poorly understood. Using an in-house custom made woodfire smoke exposure chamber our goal is to understand the smoke conditions and real-time monitoring, ensuring reproducible and relevant exposures. We are using *Drosophila melanogaster* model system in which flies share 60% genetic homology with humans and have respiratory system with key similarities to the human lung; airways composed of single epithelial cells that form branched tube networks for gas exchange. As such fruit flies can be exposed via inhalation the main route of exposure in humans. Exposing flies to 800ug/m³ of PM2.5 for 6 hours a day for 5 days significantly decreased longevity in males but not females, with similar but more pronounced effects seen in unexposed male but not female progeny. Given that progeny may have been exposed as embryos, our findings suggest potential age-at-exposure dependent effects on longevity, although transgenerational effects are also possible. As a complement, we are examining the impact of wildfire smoke on genome integrity, transcription, or the epigenome and whether these effects are transmitted to subsequent generations and are shaped by sex, age-at-exposure, and dose. As such our work in the well-established fruit fly model system presents a new means to gain much needed insight into the mechanisms by which wildfire smoke, a growing health threat, contributes to lung cancer biology.

LAY SUMMARY

Wildfire smoke contains harmful substances that may contribute to lung cancer, but how this happens at the biological level is not well understood. We developed a specialized chamber that creates controlled wildfire smoke conditions, allowing us to study its effects using fruit flies, which have genetic and respiratory similarities to humans¹. Our research found that smoke exposure led to sex-specific changes in lifespan in both parent flies and their offspring, even when the offspring were not directly exposed. This suggests possible heritable effects, which we are investigating further by tracking lifespan across generations and gene expression changes.

Cancer Risk and Prevention

Sunday, November 2, 2025

Lifetime occupational exposure to endocrine-disrupting chemicals, genetic susceptibility, and postmenopausal breast cancer risk - Poster #123

Presenter: Vikki Ho, Queen's University

Other Authors: Christina Reeves, Harriet Richardson, France Labrèche, Mark S. Goldberg, Jacques Simard, Laura Pelland-St-Pierre

ABSTRACT_ID#3146

ABSTRACT

Postmenopausal breast cancer (BC) risk may be affected by exposure to endocrine disrupting chemical (EDC) mixtures. Such exposures are common in workplaces, but research has focused on individual chemicals. This study aims to: Estimate the association between lifetime occupational exposure to EDCs, grouped by mode of action, and postmenopausal BC risk. Determine potential gene-environment interaction using a 313-variant polygenic risk score (PRS313). Data from a Montreal-based case-control study (2008-2011) was used. Cases included 695 women with malignant BC frequency-matched to 608 controls by age. Lifetime occupational history and risk factor data were collected by interview. Experts assigned lifetime occupational exposure to 24 EDCs classified by their mode of action as estrogenic, anti-estrogenic, and anti-androgenic. Exposure metrics included: ever/never exposure, duration of exposure, and cumulative exposure. Unconditional logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals [95% CI] for the association between BC and EDC exposure, plus EDC-PRS313 interaction. Ever exposure to EDCs occurred in 19% of cases and 15% of controls. Adjusted results for ever/never exposed to estrogenic (OR=1.51 [0.97, 2.37]) and anti-androgenic (OR=1.55 [1.11, 2.16]) EDCs increased risk more than anti-estrogenic (OR=1.13 [0.77, 1.68]). Similar results were found for duration; while cumulative exposure to anti-androgenic EDCs was associated with BC (OR=1.19 [1.01, 1.46]), but not estrogenic (OR=1.13 [0.97, 1.46]) and anti-estrogenic (OR=1.05 [0.84, 1.33]) EDCs. No important gene-environment interactions were detected, irrespective of mode of action. Preliminary findings suggest exposure to estrogenic and anti-androgenic EDCs may increase BC risk independently of PRS313.

LAY SUMMARY

Occupational exposure to endocrine-disrupting chemicals (EDCs) increases the risk of postmenopausal breast cancer. Workplace exposure to the selected 24 EDCs in our study, especially those that mimic the effects of estrogen (estrogenic) and oppose the effects of androgens (anti-androgenic), increased the risk of postmenopausal breast cancer. In our study, no important interaction between EDC exposure and genetic susceptibility was found. A validated polygenic risk score derived from 313 gene variants that collectively predicts breast cancer risk was used to determine if genetic susceptibility alters the risk of postmenopausal breast cancer in workers exposed to EDCs. There was no evidence that genetic susceptibility, as estimated from the polygenic risk score, modified the relationship between exposure to EDCs at the workplace and postmenopausal breast cancer risk.

Cancer Risk and Prevention

Monday, November 3, 2025

Canada's population attributable risk of lung cancer from residential radon gas exposure based on census-weighted indoor air radon levels and environmental time-use patterns - Poster #124

Presenter: Aaron A. Goodarzi, University of Calgary

Other Authors: John M Danforth, Dustin Pearson, Monica Caicedo Roa, Matthew Warkentin, Cheryl Peters, Darren Brenner

ABSTRACT_ID#3259

ABSTRACT

Increased risk of lung cancer from an environmental carcinogen exposure such as radon gas is a function of how much carcinogen is in a building, and how much time a person spends in that building over time. Over the past decade, a Canadian alliance of research and public health partners, including those involved in the Evict Radon National Study, Health Canada, and others, have integrated residential radon gas readings with housing, community, and population information to establish a symmetric understanding of radon exposure within the residential built environment. These outcomes were released as the “Cross-Canada Survey of Radon Exposure in the Residential Buildings of Urban and Rural Communities” in late 2024, and reported that 17.8% of houses in Canada exceed 200 Bq/m³, with profound differences based on region, urban-to-rural community, and residential building design type within Canada. I will provide an update on national radon exposure statistics for 2025, and describe the quantitative impact that these have had on Canadian health-seeking behaviour such as lung cancer risk awareness. I will also describe how we have leveraged radon report outcomes with an equally symmetric understanding of patterns of time-use between different environments to calculate Canadian population-attributable risk of lung cancer from residential radon exposure. We find that at least 1 in 7 lung cancers diagnosed in Canada are due to radon exposure from the residential environment. Based on current cancer statistics, at least 4,000 new lung cancer diagnoses per year are attributable to radon, highlighting the urgent need for primary prevention.

LAY SUMMARY

Lung cancer risk from cancer-causing environmental exposures such as radon is a function of how much of radon is in a building, and how much time a person spends in that building over time. 17.8% of houses in Canada exceed maximum tolerated radon exposure thresholds, with profound differences in how people are exposed based on region, whether they live in an urban-to-rural community, and the building design type of their primary house within Canada. New estimates find that 1 in 7 lung cancers that are diagnosed in Canada today are due to people breathing high radon levels within the residential environment.

Cancer Risk and Prevention

Sunday, November 2, 2025

Mother-daughter HPV screening and vaccination, a scoping review - Poster #125

Presenter: Alexandra Miller, Women's Health Research Institute

Other Authors: Grace McCloskey, Candice Ruck, Amy Booth, Laurie Smith, Gina Ogilvie

ABSTRACT_ID#2833

ABSTRACT

Introduction: Cervical cancer (CC) is the fourth most common cancer in women worldwide, with the highest incidence rates in low- and middle-income countries (LMIC). This concentration is largely driven by low uptake of CC screening and HPV vaccinations, both of which are effective in preventing CC. To address low uptake, combining cervical screening for mothers and HPV vaccination for daughters into one service has been suggested, however there is limited existing research. This scoping review aims to explore the evidence on the integration of maternal CC screening with adolescent HPV vaccination and its potential influence on engagement with CC prevention. **Methods:** PubMed was systematically searched. For inclusion, studies needed to be published in English between 2000-2025, include adolescents or pre-adolescents, and involve mother-daughter integrated services. Two authors independently screened title, abstract, and full-texts. Relevant papers were then extracted. **Results:** From 151 papers initially identified, 11 papers were identified for full-text review, and 6 papers, all from LMICs, were extracted. Local healthcare staff and community health leaders were integral in the implementation of all vaccination-screening programs. Most studies were conducted in urban or semi-urban areas. All studies suggested that combined mother-daughter service provision may increase engagement in CC prevention measures. **Discussion:** There is a limited but growing body of evidence on mother-daughter strategies to enhance engagement in CC prevention in LMICs. Despite heterogeneity in outcome measures, findings suggest that integrated mother-daughter service delivery may enhance engagement in CC prevention. Further research using standardized evaluation methods are needed to better assess effectiveness.

LAY SUMMARY

Combining HPV vaccination for adolescent girls with cervical screening for their mothers can be an effective approach in the prevention of cervical cancer, especially in low- and middle-income countries (LMIC). Most studies were in urban and semi-urban settings, with limited research in rural areas. Further research is needed to compare the mother-daughter approach to the standard approach of independent vaccination and screenings.

Cancer Risk and Prevention

Monday, November 3, 2025

Understanding behavioural, built environmental and socio-demographic influences on lung cancer risk from residential radon exposure in Canada - Poster #126

Presenter: Dustin D. Pearson, University of Calgary

Other Authors: Grace Johnson, John Danforth, Monica Caicedo Roa, Markus Nielson, Josh Taron, Aaron Goodarzi

ABSTRACT_ID#3267

ABSTRACT

Residential radon exposure is a leading cause of lung cancer in Canada, and is an anthropogenic environmental cause of cancer largely arising from how we have and are building residential properties. The 2024 Cross Canada Radon Report demonstrated that over 10.8 million Canadians live in an indoor environment containing levels of the radioactive gas, radon (^{222}Rn), that increases their lifetime relative risk of lung cancer by at least 16%. Moreover, 1 in 5 homes have levels of radon that are above Health Canada's limit of 200 Bq/m³. I will present the outcomes from >600,000 continuous hourly radon readings collected across years, from residential houses from multiple Canadian cities. I will describe the influence of residential building indoor air dynamics on Canadian radon exposure, presenting data as a function of variables such as building type (single detached bungalows, split levels, multi-storeys, and semi detached and row style houses), ventilation and heating features, seasonal fluctuations, regional differences, and human behaviours such working life patterns, occupation type, and more. I will also present our new multivariate, predictive models that describe the Canadian residential building characteristics that are most associated with high indoor air radon levels. Our models demonstrate more than 80% capability of predicting a home containing radon above 200 Bq/m³ (Health Canada's exposure limit), with building age and type being the greatest contributing factors. Finally, these models underline how, within one region, the built environment is the primary factor driving high radon and underlies Canada's substantial and worsening radon gas exposure problem.

LAY SUMMARY

One of the largest sources of environmental lung cancer, comes from our homes, with 1 in 5 Canadian residential properties having high, cancer causing levels of radioactive radon gas. High radon in Canada is primarily a human created issue, with how we build and use our residential buildings being the largest factors in having high radon. By using continuous hourly radon data, along side more than 66,000 long term radon results from Canadian residential properties, my work shows how factors such as the type of residential property and its age are the most significant factors in understanding risk to high radon exposure. This work is important as it highlights how Canadian residential properties are being built (unintentionally) to standards that increase lung cancer risk, from radon and that if left unaddressed, will increase the burden of non-tobacco related lung cancers in Canada.

Cancer Risk and Prevention

Sunday, November 2, 2025

Changes in Cancer-Causing Substance Use During COVID-19 in Gender Minority Populations: an analysis from the CanPath cohort - Poster #127

Presenter: Ace Chan, University of British Columbia

Other Authors: Hannah Kia, Travis Salway, Trevor Dummer

ABSTRACT_ID#2985

ABSTRACT

Background: Gender minority (GM) people are those whose gender identity is – either fully or in part – different from their biological sex assigned at birth (e.g., transgender or non-binary people). Unique stressors relative to cisgender (CG) populations, including stigma and discrimination, are experienced by GM people which may lead to using cancer-causing substances as means of coping. This study aimed to assess if alcohol, smoking, e-cigarettes, and cannabis use in the GM population, changed during the COVID-19 pandemic. Methods: The Canadian Partnership for Tomorrow's Health (CanPath), a national longitudinal cohort, aims to better understand cancer risk factors. Three survey cycles of CanPath were used to answer the aims of this study. There were 76,795 CG people and 513 GM people in the study sample. GEE analyses were conducted to determine changes in alcohol, smoking, and vaping between 2008 and 2020. Logistic regression analyses were conducted to determine pre/post-COVID-19 change alcohol, smoking, e-cigarette, and cannabis use, in 2020. Results: Longitudinal analyses found no significant differences in alcohol, smoking, and vaping use between CG and GM people. Logistic regression analyses found that GM people had higher odds of reporting more cannabis use than before the pandemic, compared to CG people (adjusted odds ratio (aOR): 1.85; 95% confidence interval (CI): 1.11-3.09). Conclusion: We detected a temporal increase in cannabis use among GM adults in Canada in 2020 when participants reported their use before and during the beginning of the pandemic. The increase may be explained by how cannabis use provides gender-affirmation.

LAY SUMMARY

Between 2008 and 2020, there were no differences in alcohol use, smoking, and vaping when gender minority people were compared to cisgender people. However in 2020, gender minority participants who reported their cannabis use before and during the pandemic showed that they had higher cannabis use compared to cisgender people. Increases in cannabis use during the pandemic can possibly be explained by how the literature documents the relationship between cannabis use and gender affirmation.

Cancer Risk and Prevention

Monday, November 3, 2025

Cervical cancer elimination in Manitoba: An exploration of the current state and opportunities for successful elimination in the future - Poster #128

Presenter: Carrie O'Conaill, CancerCare Manitoba

Other Authors: Carla Loeppky, Alyson Mahar, Erin Dean, Donna Turner

ABSTRACT_ID#2702

ABSTRACT

Background. Cervical cancer can be eliminated through human papillomavirus (HPV) vaccination and cervical cancer screening, both of which significantly reduce disease burden. The World Health Organization (WHO) has set a global elimination target of 4 cases per 100,000 women by 2040, emphasizing prevention, early detection, and equitable healthcare. Manitoba has an opportunity to advance this goal by strengthening prevention strategies, addressing screening disparities, and ensuring equitable vaccine coverage. **Methods:** Objectives fall into three sections. First, we examine HPV vaccination rates, cervical cancer screening participation, and cervical cancer incidence in Manitoba over time and across geography, sex, and income quintiles using GIS mapping to help identify unmet needs. Second, a retrospective cohort study assesses whether HPV vaccination influences screening participation for a vaccinated cohort of individuals and matched unvaccinated controls. Finally, a microsimulation platform (OncoSim-Cervical by Canadian Partnership Against Cancer) compares over 1,000 scenarios to estimate future cervical cancer incidence, mortality, program volumes and costs, and outcomes to define the best path forwards for elimination of cervical cancer in Manitoba. **Results:** Preliminary findings indicate rising cervical cancer diagnoses in Manitoba (+9.14 annual percent change between 2018-2023, $p < 0.05$) with an age-standardized incidence rate of 9.4 per 100,000 women in 2023. Ongoing analyses of vaccination uptake and screening trends will be presented along with microsimulation results to create a Manitoba-specific roadmap. **Conclusion:** Cervical cancer elimination in Manitoba is achievable through data-driven strategies, targeted interventions, and evidence-based healthcare planning. This research will define current prevention efforts, highlight gaps, and establish a roadmap for elimination.

LAY SUMMARY

Cervical cancer can be eliminated in Manitoba. The province's current incidence rate remains higher than the World Health Organization's target of 4 per 100,000 for elimination by 2040. Strengthening HPV vaccination and screening programs can help reach this goal. Data-driven strategies will guide elimination efforts. This research examines cervical cancer epidemiology, HPV vaccination rates, and cervical cancer screening participation in Manitoba, identifying populations at higher risk due to low vaccine uptake or screening gaps. The findings will support targeted health interventions to improve access. A clear roadmap for elimination will be developed. Using insights from Manitoba's cancer data and the OncoSim-Cervical model, the study will identify optimal prevention and treatment strategies, guiding a high-quality implementation plan to eliminate cervical cancer in the province.

Cancer Risk and Prevention

Monday, November 3, 2025

HiChIP-Based Epigenomic Footprinting Identifies a Promoter Variant of UXS1 That Confers Genetic Susceptibility to Gastroesophageal Cancer - Poster #130

Presenter: Ansley Gnanapragasam, McGill

Other Authors: Eftyhios Kirbizakis, Anna Li, Kyle White, Katelyn Mortenson, Colleen Russett, Juliana Cavalcante de Moura, Wajih Jawhar, Yifei Yan, Betty Giannias, Sophie Camilleri-Broet, Nicholas Bertos, Jonathan Cools-Lartigue, Livia Garzia, Veena Sangwan, Lorenzo Ferr

ABSTRACT_ID#2299

ABSTRACT

Genome-wide association studies (GWAS) have identified more than a hundred single nucleotide variants (SNV) associated with the risk of gastroesophageal cancer (GEC). The majority of the identified SNVs map to noncoding regions of the genome. Uncovering the causal SNVs and genes they modulate could help improve GEC prevention and treatment. Herein, we used HiChIP against histone 3 lysine 27 acetylation (H3K27ac) to simultaneously annotate active promoters and enhancers, identify the interactions between them, and detect nucleosome-free regions (NFR) harboring potential causal SNVs in a single assay. The application of H3K27ac HiChIP in GEC relevant models identified 61 potential functional SNVs that reside in NFRs and interact with 49 genes at 17 loci. The approach led to a 67% reduction in the number of SNVs in linkage disequilibrium at these 17 loci, and at 7 loci, a single putative causal SNV was identified. One SNV, rs147518036, located within the promoter of the UDP-glucuronate decarboxylase 1 (UXS1) gene, seemed to underlie the GEC risk association captured by the rs75460256 index SNV. The rs147518036 SNV creates a GABPA DNA recognition motif, resulting in increased promoter activity, and CRISPR-mediated inhibition of the UXS1 promoter reduced the viability of the GEC cells. These findings provide a framework that simplifies the identification of potentially functional regulatory SNVs and target genes underlying risk-associated loci. In addition, the study implicates increased expression of the enzyme UXS1 and activation of its metabolic pathway as a predisposition to gastric cancer, which highlights potential therapeutic avenues to treat this disease.

LAY SUMMARY

Researchers have found over 100 genetic variants linked to the risk of gastroesophageal cancer but most of these variants are located in parts of the genome that don't directly code for proteins, making it hard to understand how they cause disease. To facilitate the identification of variants that directly play a role in this pathology, we devised a method that incorporates a technique called HiChIP to map which parts of the genome regulate gene activity and how these parts physically interact. This alongside side a tool we designed, Histrader, to identify nucleosome free regions (NFRs), allowed us to dramatically reduce the number of SNVs and pinpoint potentially causal SNVs. We focused on one such variant, called rs147518036, which increases the activity of a gene called UXS1. Blocking this gene reduced cancer cell survival, suggesting it plays a role in cancer development. Our work here provides a new way to uncover hidden genetic risks for cancers and opens the door to potential new treatment strategies targeting genes like UXS1.

Cancer Risk and Prevention

Sunday, November 2, 2025

Thinking style disposition and identification of cancer misinformation in a population-based survey - Poster #131

Presenter: Alexandra Walker, University of British Columbia

Other Authors: Emily Heer, Samina Hossain, Alessandro Marcon, Marco Zenone, Melanie Matte, Cheryl Peters

ABSTRACT_ID#3011

ABSTRACT

Background: Most people believe information sometimes, but there may be factors that make some more open to these beliefs than others. Dispositional thinking style, the tendency toward open-minded, close-minded, intuitive, or effortful thinking, is one theory of susceptibility that has been proposed. We used a validated questionnaire to examine whether there is an association between individual thinking styles and belief in cancer misinformation. Methods: Through an online survey, we asked participants how much they agreed with both true statements about cancer risk factors and cancer misinformation. At the end of the survey, participants completed a questionnaire to assess four distinct thinking styles. We analyzed results descriptively and conducted regression models to identify possible associations. Results: A total of 2,003 respondents completed the survey. Thinking style score was correlated with correctness in all cases: active open-minded thinking (AOT), close-minded thinking (CMT), preference for effortful thinking (PET), and preference for intuitive thinking (PIT). In general, those with more alignment with AOT or preference for PET was associated with higher correctness of cancer myths and close alignment with CMT or PIT was associated with lower correctness. Additionally, higher self-reported confidence was associated with worse myth detection for those who were more closely aligned with CMT and PIT, suggesting an overconfidence effect. Conclusion: Greater alignment with close-minded and intuitive thinking styles predicted less accuracy in detecting cancer misinformation, suggesting greater susceptibility to belief in misinformation. Individuals with these thinking styles may also have misaligned confidence in their ability to detect misinformation.

LAY SUMMARY

Thinking style is linked to how well individuals can spot cancer misinformation. Close-minded thinkers and people who rely on their intuition are less able to identify misinformation than open-minded or effortful thinkers. For close-minded and intuitive thinkers, having higher confidence in health knowledge further harmed their ability to detect misinformation. Some thinking styles might be more vulnerable to misinformation, especially when coupled with higher confidence. Encouraging analytical thinking might be a strategy to address misinformation.

Cancer Risk and Prevention

Monday, November 3, 2025

Unlock new cancer and chronic disease insights: How researchers can leverage Alberta's Tomorrow Project's longitudinal data and biosamples - Poster #134

Presenter: Grace Shen-Tu, Alberta Health Services

Other Authors: Laura Grant, Shandra Harman, Gillian MacNevin, Sara Nejatnamini, Will Rosner, Cassidy See, Bud Skiffington, Jennifer Vena

ABSTRACT_ID#2994

ABSTRACT

Background: Alberta's Tomorrow Project (ATP) is a prospective cohort study established in 2000 to provide researchers and other data-users with a platform for investigating the etiology of cancer and chronic diseases. **Methods:** Between 2000 and 2015, ATP recruited 55,000 Albertans aged 35–69 with no previous cancer diagnosis (except non-melanoma skin cancer). At recruitment, ATP collected comprehensive health and lifestyle data from all participants and biosamples (blood, urine, and saliva) from the 30,000 who attended a study centre. Since then, all participants have been invited to complete numerous follow-up health and lifestyle questionnaires. In 2008, ATP joined the Canadian Partnership for Tomorrow's Health (CanPath), a consortium of similar prospective cohort studies across Canada which is compiled of more than 330,000 Canadians. **Results:** ATP is as a well-established repository of longitudinal health and lifestyle data and biosamples. Participant data can also be linked with administrative health data, including tumour staging, screening records, environmental exposures, and more. As of 2025, over 6,000 participants have experienced cancer. ATP's participation in CanPath, Canada's largest population health research platform, enables data pooling with other regional studies across Canada, enhancing research possibilities. **Conclusion:** Through our comprehensive longitudinal information on participant demographics, healthcare utilization, and modifiable risk factors—including how these have changed over time—along with linkage capabilities to external datasets, ATP can support a variety of cancer and chronic disease research. For more information, please visit <https://myatpresearch.ca/>.

LAY SUMMARY

Since 2000, ATP has been following the health of almost 55,000 Albertans to better understand why some people get cancer while others do not. ATP collects health and lifestyle information through questionnaires, and also links to medical records, cancer registries, and environmental data, giving us a comprehensive view of participants' health over time. ATP is a rich resource for researchers looking at the health of Albertans.

Cancer Risk and Prevention

Sunday, November 2, 2025

Trends in smoking and obesity prevalence at breast cancer diagnosis: Results from a 35-year retrospective study - Poster #135

Presenter: Flora Nguyen Van Long, Centre de Recherche du CHU de Québec - Université Laval

Other Authors: Ruth Doriane Sèhivè Gbessemehlan, Nyamador Komla David Adenyo, Ouerdia Haroun, Annie Turgeon, Asma Boubaker, Christian Laflamme, Julie Lemieux, Hermann Nabi

ABSTRACT_ID#2863

ABSTRACT

Background: Breast cancer (BC) is the most commonly diagnosed cancer among women. Monitoring long-term trends in modifiable risk factors at the time of diagnosis is critical for guiding and evaluating prevention strategies. This study examined 35-year trends in the prevalence of two modifiable risk factors—obesity and smoking—among women diagnosed with BC. **Methods:** We conducted a retrospective cohort study of 14,595 BC women diagnosed at the Centre des maladies du sein in Quebec City, between 1987 and 2021. Prevalence ratios (PRs) for obesity and smoking were estimated using multivariable log-binomial regression models, adjusting for demographic and clinical characteristics. **Results:** Over the 35-year period, the prevalence of obesity at diagnosis significantly increased (PR = 1.19; 95% CI 1.15–1.24; $p < 0.0001$), while smoking prevalence declined (PR = 0.92; 95% CI 0.89–0.95; $p < 0.0001$). Stratified analyses revealed a more pronounced increase in obesity (PR = 1.40; 95% CI 1.25–1.57; $p < 0.0001$) and a sharper decline in smoking (PR = 0.82; 95% CI 0.78–0.86; $p < 0.0001$) among premenopausal women compared to postmenopausal women (obesity: PR = 1.16; 95% CI 1.11–1.21; $p < 0.0001$; smoking: PR = 0.97; 95% CI 0.93–1.01; $p = 0.1555$). **Conclusions:** The rising prevalence of obesity among BC patients at diagnosis highlights the need to reinforce public health anti-obesity efforts. While reductions in smoking suggest the success of anti-smoking measures, additional targeted strategies are necessary to mitigate the growing obesity epidemic and its potential impact on BC incidence in Canada.

LAY SUMMARY

Breast cancer cases are still on the rise in Canada. Many factors can increase the risk of developing breast cancer, including lifestyle habits. By looking at how common these habits are among people diagnosed with breast cancer over time, we can better understand whether current prevention efforts are working. This information can also help shape future public health campaigns. In this study, we focused on two lifestyle risk factors: obesity and smoking. We found that, over the past 35 years, fewer people diagnosed with breast cancer in Quebec City were smoking at the time of their diagnosis. At the same time, more of them were living with obesity. These changes were even more noticeable in women who had not yet reached menopause. These results suggest that public health efforts to reduce smoking have worked well. However, current strategies to prevent and manage obesity do not seem to be as effective. In fact, obesity may now be a more important risk factor for breast cancer than smoking—especially for younger women. To help reduce future cases of breast cancer, it is important to improve and focus obesity prevention programs, particularly for women who have not yet reached menopause.

Cancer Risk and Prevention

Monday, November 3, 2025

Fish Oil Enhances the Efficacy of a Ketogenic Diet in Lowering Tobacco Carcinogen-induced Lung Cancer - Poster #136

Presenter: Ingrid Elisia, BC Cancer Research Center

Other Authors: Michelle Yeung, Sara Kowalski, Taras Shyp, Jason Tee, Serena Hollman, Amy Wong, Janette King, Roger Dyer, Poul Sorensen, Gerald Krystal

ABSTRACT_ID#2546

ABSTRACT

Since cancer cells typically take up and need more glucose than normal cells, we hypothesized that a ketogenic diet, which is very low in carbohydrates, might slow tumor growth. However, since ketogenic diets are very high in fat, and different fats might have different biological effects, we investigated whether fat types influenced the efficacy of a ketogenic diet to lower tobacco carcinogen-induced lung cancer in mice. We placed mice on a Western diet, or ketogenic diet comprised of either palm oil, olive oil, corn oil, milk fat, fish oil, medium chain triglyceride oil, or a blend of fats typically found in a Western diet. We then intraperitoneally injected the tobacco-specific carcinogen into these mice. Mice that were fed the ketogenic diets had significantly ($P < 0.05$) lower lung nodule numbers compared to the Western diet, suggesting that glucose restriction is indeed a feasible strategy to slow tumor formation. Comparing the different ketogenic diets, we found the ketogenic diet containing fish oil was significantly ($P < 0.05$) more effective than the rest of the ketogenic diets in reducing lung tumor formation. This increased efficacy was associated with higher plasma β -hydroxybutyrate, lower blood glucose, and liver fatty acid expression. Taken together, fish oil appears to be unique in its ability to promote ketosis in mice fed a ketogenic diet. We conclude that incorporation of fish oil, which is high in the omega-3 fatty acids, EPA, and DHA, can increase the efficacy of a ketogenic diet in preventing the formation of NNK-induced lung tumors.

LAY SUMMARY

We evaluated whether low-carbohydrate, high-fat ketogenic diets can influence lung cancer formation in mice treated with a cigarette carcinogen. Since a ketogenic diet typically consists of at least 70-80% of total calories as fat, and some fats are reported to promote cancer, we also wanted to determine if the type of fat used in these ketogenic diets matters. We formulated ketogenic diets enriched in various types of fats, including palm oil, olive oil, corn oil, milk fat, fish oil, medium chain triglyceride oil, or a blend of fats typically found in a Western diet. We then tested whether this series of ketogenic diets could prevent lung tumour growth in mice injected with the cigarette smoke carcinogen. We found that while most of these ketogenic diets were able to reduce lung tumours in these mice, compared to a Western diet, incorporating fish oil into the ketogenic diet provided the greatest suppression of lung tumour nodules. This suggests that incorporation of fish oil, which is high in the omega-3 fatty acids, EPA, and DHA, can potentially increase the effectiveness of a ketogenic diet in preventing lung cancer.

Cancer Risk and Prevention

Sunday, November 2, 2025

Leveraging Large Language Models for Real-Time Detection and Classification of HPV Vaccine Misinformation to Inform Targeted Public Health Interventions in Canada - Poster #137

Presenter: Lisa YW Tang, University of British Columbia

ABSTRACT_ID#2530

ABSTRACT

Human Papillomavirus (HPV) vaccination, with nearly 90% efficacy against cancer-causing infections, is critical for preventing cervical and other HPV-related cancers. However, Canada's goal of achieving a 90% HPV vaccination rate among youth by 2025 is at risk, with vaccination rates plummeting (e.g., 5.2% in Ontario during 2019–2020), jeopardizing cancer prevention efforts. A significant driver of this decline is the spread of HPV vaccine misinformation on social media. To address this, we propose LISTEN (Leveraging Intelligence from Social networks to promote health Equity and Neutralize harm), a digital surveillance tool powered by Large Language Models (LLMs). LISTEN enables real-time detection, classification, and trend analysis of vaccine misinformation across Canadian social media platforms. It identifies false narratives—such as misinformation about vaccine side effects, efficacy, or necessity—while monitoring public engagement indicators. Leveraging in-context learning and multilingual processing, LISTEN achieves detection accuracy exceeding 80% F1-macro, as validated by experimental evaluations with open-source datasets. With stakeholder consultation and co-development underway, LISTEN is the potential to equip public health agencies to: Develop evidence-based counter-messaging to refute misinformation; Target interventions by pinpointing where specific falsehoods are gaining traction; Inform platform-specific communication strategies; Allocate resources effectively against the most impactful misinformation. By delivering actionable, LLM-driven intelligence, LISTEN empowers public health officials to implement precise, timely interventions, rebuild vaccine confidence, improve HPV vaccination uptake, and achieve Canada's 2025 immunization targets, ultimately reducing the burden of HPV-related cancers.

LAY SUMMARY

HPV vaccination rates in Canada are declining, putting cancer prevention at risk. Despite the vaccine being nearly 90% effective in preventing HPV-related cancers, recent vaccination rates (e.g., only 5.2% in Ontario in 2019–2020) are far below the national goal of 90% by 2025. This decline threatens efforts to prevent cervical and other HPV-related cancers. Social media misinformation is a major factor driving vaccine hesitancy. False claims about HPV vaccines—such as misinformation about safety, effectiveness, or necessity—spread quickly on social media, influencing public perceptions and decision-making. Our LISTEN framework proposes a cutting-edge solution to detect and neutralize HPV vaccine misinformation. Powered by well-tested large language models, LISTEN can monitor misinformation trends on social media in real-time, identify false narratives, and track public engagement with this content. With detection accuracy above 80%, LISTEN is designed to help public health agencies create rapid, targeted responses to misinformation and allocate resources efficiently to improve HPV vaccination rates and rebuild public trust.

Cancer Risk and Prevention

Monday, November 3, 2025

A Low-Carbohydrate Diet High in Soy Protein and Fish Oil Reduces Lung, Breast, Myelodysplastic, and Colon Cancer in Mouse Models - Poster #138

Presenter: Ingrid Elisia, BC Cancer Research Center

Other Authors: Michelle Yeung, Gerald Krystal

ABSTRACT_ID#2560

ABSTRACT

Based on Otto Warburg's finding that cancer cells typically take up and need more glucose than normal cells, we hypothesized that a glucose-restricted diet might be effective in both preventing and treating cancer. We formulated different low-carbohydrate (CHO) diets and showed that a low-CHO diet containing the resistant starch, amylose, soy protein and fish oil (i.e., a 15% Amylose/Soy/FO diet), was most effective in preventing the development of tobacco carcinogen-induced lung cancer in A/J mice. We further showed that this same low-CHO diet, when compared to a Western diet, was highly effective in reducing mammary tumour burden in C3(1)/SV40 mice, colitis-associated colon cancer in BALB/c mice, and in extending the lifespan of miR-146a KO mice that typically succumb to myelomalignancy-related deaths. This 15% Amylose/Soy/FO diet shifted the metabolism of these mice from a reliance on glycolysis, which is commonly upregulated in cancer cells, to using fats as an energy source. At the same time, this 15% Amylose/Soy/FO diet reduced chronic inflammation, an important cancer-promoting factor in many of our mouse models. In the case of colon cancer, we also showed that this 15% Amylose/Soy/FO diet induced a marked change in the fecal microbiome and an increase in cecal and fecal acetate, as well as a reduction in interleukin-17A expressing cells in colonic tumors, typically associated with a poor prognosis in colon cancer patients. Taken together, we suggest that consuming a 15% Amylose/Soy/FO diet may be effective in preventing various cancers and that clinical trials to confirm its efficacy in humans are warranted.

LAY SUMMARY

Since cancer cells typically need more glucose than normal cells to survive and proliferate, we hypothesized that a glucose-restricted diet, such as a low-carbohydrate diet, might be effective in preventing or treating cancers. We formulated a series of low-carbohydrate diets varying in type and level of carbohydrate, protein, and fat and found that a low-carbohydrate diet high in resistant starch, soy protein, and fish oil (15% Amylose/Soy/FO) was most effective in preventing cigarette smoke-induced lung tumours in mice. Since no two cancers are the same, we tested the impact of this diet on different types of cancer and found that the 15% Amylose/Soy/FO was also effective in myelodysplasia, breast and colon cancer, but not in prostate cancer in mouse models. This suggests that consuming a 15% Amylose/Soy/FO diet may be effective in preventing various cancers and that human trials are warranted.

Cancer Risk and Prevention

Sunday, November 2, 2025

Investigating the molecular programs involved in metastasis initiation in human triple-negative breast cancer - Poster #139

Presenter: Hee Jin (Hayley) Shin, University of Toronto/Princess Margaret Cancer Research Centre

Other Authors: Sarah Kronheim, Darryl Chan, Sahil Sharma, Robert Vanner, Laurie Ailles, Long Nguyen

ABSTRACT_ID#2984

ABSTRACT

Triple-negative breast cancer (TNBC) is the subtype with the worst 5-year survival rate especially in the metastatic setting. These cancers display significant cellular and phenotypic heterogeneity, where metastatic disease is often attributed to a minority of clones. Therefore, we aim to identify the underlying molecular programs that regulate metastasis initiation in TNBC. To achieve this, we developed a lentiviral-based cellular barcoding system where TNBC cells are labeled uniquely and permanently with a DNA-based barcode sequence that is detectable with both DNA amplicon and single cell RNA sequencing (scRNAseq). This will allow us to associate individual TNBC clones with their transcriptomic profiles. This system was also constructed with both EGFP for in vitro tracking and Luciferase for in vivo tracking. We used this to track clonal growth and metastases from 2 cell lines and 2 patient-derived xenograft (PDX) models injected into immunodeficient NSG mice. Metastases were primarily detected in the lungs for all models, followed by liver for all except 1 PDX model. Metastatic burden in the lungs was quantified using flow cytometry, with up to 15% CD298+ human tumour cells measured in dissociated mouse lung tissue thus validating the feasibility of quantifying and isolating cells from metastatic clones from mouse organs. From scRNAseq, we will perform differential gene expression analyses comparing clones that metastasize to those that do not. This work will elucidate key molecular pathways involved in metastasis initiation in TNBC with the potential to guide future drug development efforts aimed at improving outcomes for patients with TNBC.

LAY SUMMARY

- Triple negative breast cancer (TNBC) is the most aggressive breast cancer subtype especially when it has spread to other organs – termed metastasis. Our lab has shown that not all cancer cells give rise to progeny that spread to other organs. Therefore, I aim to investigate why some TNBC cells spread to other organs while others do not.
- We developed a new tool called cellular barcoding to label TNBC cells with unique barcodes such that the barcodes will act as a label for all the cells representing the progeny of a single initially and uniquely barcoded cell. Using this approach we can determine which cells give rise to progeny that will spread to other organs, and which will not.
- We show that TNBC tumour cells spread primarily to the lungs and liver. We have developed an approach to quantify the number of tumour cells detected in these organs and show in some cases up to 15% of cells in the mouse lung were made up of tumour cells. I aim to isolate and profile these tumour cells to understand which genes are turned on and off that allow them to spread.

Cancer Risk and Prevention

Monday, November 3, 2025

Saponins in Soy Reduce Lung Cancer in a Mouse Model by Increasing Plasma Isoflavone Levels - Poster #140

Presenter: Ingrid Elisia, BC Cancer Research Center

Other Authors: Michelle Yeung, Sara Kowalski, Amy Wong, Colton Dietrich, Samantha Wu, Vianne Chang, Hans Adomat, Gerald Krystal

ABSTRACT_ID#2570

ABSTRACT

We recently found that soy protein was superior to casein in preventing cigarette carcinogen-induced lung cancer in mice. Specifically, we showed that diets containing 35% of total calories as soy protein isolate (SPI), which is partially delipidated, concentrated soybean protein, reduced the number of lung nodules induced by the nicotine-derived nitrosamine ketone (NNK) by half, compared to 35% casein. To understand why SPI, which contains several potential bioactive components, such as isoflavones, bioactive peptides, saponins, and proteins was superior to the milk protein, casein, we tested each of these potentially bioactive components individually or in combinations and found from in vivo studies that only diets containing saponins in combination with isoflavones significantly lowered lung nodule formation. We further showed that this combination resulted in an increased level of isoflavones in the plasma. We conclude that saponins together with isoflavones are responsible for the ability of SPI to reduce lung cancer formation in mice and that this is due, at least in part, to the saponins increasing the in vivo levels of isoflavones.

LAY SUMMARY

We recently found that soy protein isolate (SPI) was more effective than casein at preventing cigarette carcinogen-induced lung cancer in mice. SPI, however, contains several potential components that could be effective in preventing cancer, such as isoflavones, saponins, peptides and proteins. To identify the components within SPI responsible for its cancer prevention properties we tested each of these potentially bioactive components individually or in combinations and found that only diets containing saponins in combination with isoflavones significantly lowered lung nodule formation in these cigarette carcinogen-treated mice. We found that while isoflavones alone could not lower lung tumour formation, combining saponins and isoflavones increased the level of isoflavones in plasma, suggesting that saponins act, at least in part, by increasing in vivo isoflavone levels and that this is responsible for the ability of SPI to reduce lung cancer formation in mice.

Cancer Risk and Prevention

Sunday, November 2, 2025

Integrating PM2.5 Physical Characterization with In Vitro Models to Investigate Lung Epithelial Cell Responses to Air Pollution - Poster #141

Presenter: Isabella Mendoza Montealegre, Dalhousie

Other Authors: Jayme Salsman, Graham Dellaire, Geoffrey Maksym

ABSTRACT_ID#3034

ABSTRACT

Environmental pollutants generated by urban expansion, climate change, and forest fires produce airborne particles that contribute to rising rates of cardiorespiratory disease. Particulate matter equal to or smaller than 2.5 microns (PM2.5) can penetrate deep into the lungs and damage the alveolar epithelium (AE). Depending on their source, these particles can contain different chemical profiles that can contribute to how toxic they are. In addition, it is likely that their physical properties, including size, morphology, and aggregation state, can also cause damage independent of their chemical composition. Thus, characterizing the physical properties of PM2.5 prior to in vitro exposure is important for understanding how particle structure influences deposition, cellular uptake, and biological responses. We are characterizing the physical properties of PM2.5 using scanning electron microscopy (SEM), transmission electron microscopy (TEM), and Morphologi imaging to assess particle shape, size distribution, and surface features. A549 alveolar type-II cells and small airway epithelial cells (SAEC) were exposed to PM2.5, revealing differences in particle association and internalization. TEM confirmed intracellular uptake, while SEM showed variable clustering patterns across cell types, suggesting that uptake depends on epithelial phenotype and surface structures. To model airway exposure more accurately, we are developing an air-liquid interface (ALI) system using SAECs and A549s to investigate how tight junction formation, ciliation and production of surfactant and mucin alter PM2.5 interactions with cells. By integrating physical particle characterization with in vitro modelling, this work aims to clarify how PM2.5 disrupts epithelial homeostasis and contributes to early events in lung carcinogenesis.

LAY SUMMARY

Tiny air pollution particles (PM2.5) can reach deep into the lungs and harm the cells that line our airways, potentially contributing to lung diseases, including cancer. These particles vary in shape and size depending on where they come from, which may affect how toxic they are. We found that different lung cell types interact with PM2.5 in different ways. Some cells internalize the particles, while others show clusters of particles on their surface. This suggests that the structure and function of each cell type influence how particles are taken up or deposited. To better mimic how lungs encounter pollution in real life, we're building a 3D lab model of the airway using human lung cells. This model includes features like mucus, cilia, and surfactant that help us study how particles behave at the air-cell interface, improving our understanding of early lung damage from pollution exposure.

Cancer Risk and Prevention

Monday, November 3, 2025

Evaluating the Relationship Between Glucagon-Like Peptide-1 Receptor Agonists and Colorectal Cancer Risk

- Poster #142

Presenter: Isha Lalani, University of Calgary

Other Authors: Reynaldo Nambayan, Chantelle Carbonell, Yibing Ruan, Robert J. Hilsden, Darren R. Brenner

ABSTRACT_ID#3016

ABSTRACT

Background: People living with obesity or type 2 diabetes are at a higher risk of developing various chronic conditions, including at least 13 obesity-associated cancers. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are commonly used to treat type 2 diabetes, offering benefits for glycemic control and weight loss. As use expands, interest is growing in their effects on cancer outcomes. Although GLP-1RAs may help reduce obesity-related cancer risk, more data are needed to understand their association with site-specific cancers such as colorectal cancer (CRC). **Objective:** To systematically assess the association between GLP-1RA use and risk of CRC in adults with type 2 diabetes and/or obesity. **Methods:** A comprehensive search of EMBASE and MEDLINE was conducted through April 2025 to identify cohort and case-control studies reporting cancer risk among GLP-1RA users compared to non-users or users of other anti-diabetic agents. Randomized controlled trials and case reports were excluded. **Results:** Of 2,114 studies identified, 1,379 were screened after removing duplicates. 73 studies met inclusion criteria, 10 of which specifically examined CRC outcomes in observational studies of real-world data. GLP-1RAs assessed included semaglutide, dulaglutide, liraglutide with comparators such as insulin, metformin, and SGLT-2 inhibitors. A pooled analysis showed a statistically significant reduction in CRC risk associated with GLP-1RA use (RR 0.63, 95% CI 0.53–0.74). **Conclusion:** GLP-1RA use was associated with a lower risk of CRC compared to other antidiabetic agents in observational studies. Further research is needed to assess the clinical relevance and impact of GLP-1RAs on CRC risk reduction.

LAY SUMMARY

People with obesity or type 2 diabetes are more likely to develop certain cancers, including colorectal cancer. This review examined existing research to assess if glucagon-like peptide-1 receptor agonists reduce the risk of colorectal cancer. Findings suggest that people using glucagon-like peptide-1 receptor agonists had a lower risk of developing colorectal cancer compared to those not using them or using other diabetes medications. More research is needed to confirm and understand this potential benefit.

Cancer Risk and Prevention

Sunday, November 2, 2025

Active surveillance of RAS-positive indeterminate thyroid nodules suggests that observation is a viable option - Poster #143

Presenter: Jackson Qi, University of Calgary

Other Authors: Jiahui Wu, Moosa Khalil, Sana Ghaznavi, Caitlin Yeo, Erik Nohr, Adrian Box, Mark Eszlinger, Ralf Paschke

ABSTRACT_ID#2720

ABSTRACT

Background: Resection of all RAS-positive nodules is a low-yield strategy resulting in overtreatment for up to 50% of patients. Our approach incorporates clinical findings, standardized cytology and ultrasound results into shared decision-making. Methods: Reflex ThyroSPEC molecular testing (MT) for Bethesda III and IV indeterminate thyroid nodules was implemented in July 2020. The study included 233 consecutive patients (200 Bethesda III, 33 Bethesda IV). 162 (70%) underwent surgery while 71 (30%) surveillance. Nodule stability was evaluated in 47 surveillance patients with at least one post-MT ultrasound and one-year follow-up. Results: Median maximum nodule size was larger in surgery group than surveillance group (2.9 cm vs. 2.4 cm, $p = 0.003$). Bethesda III nodules were more common in surveillance patients than surgery patients (94% vs. 82%, $p = 0.023$). Nuclear atypia was reported in 52% of surgery patients and 38% of surveillance patients ($p = 0.059$). Seven patients changed to surgery due to patient preference, nodule growth, or symptoms; 4 patients had malignant histology of ATA low-risk cancers. Using a 72% volume increase threshold, 2/47 (4%) nodules exhibited growth, 29/47 (62%) were stable or decreased. Using a 50% threshold, 3/47 (6%) exhibited growth, with a cumulative incidence of growth of 2% at 1 year, 2% at 2 years, and 24% at 4 years. Conclusion: RAS mutation should not be the sole determinant for surgery. Clinical factors such as Bethesda category and cytologic atypia should also guide management decisions. Observation of RAS-positive patients is a viable option, though further long-term data are needed.

LAY SUMMARY

Active surveillance of RAS-positive indeterminate thyroid nodules suggests that observation is a viable option

Cancer Risk and Prevention

Monday, November 3, 2025

Increasing years of night shift work and the risk of multiple cancers using a systematic review and dose-response meta-analysis - Poster #144

Presenter: Jennifer Ritonja, BC Cancer Research Institute

Other Authors: Kyriaki Papantoniou, Lisa Leung, Pareesa Kassam, Agnes Ebenberger, Gernot Wagner, Gerald Gartlehner, Irma Klerings, Rebecca Morgan, Harald Herkner, Kristan Aronson, Eva Schernhammer

ABSTRACT_ID#2776

ABSTRACT

Objective: Although the International Agency for Research on Cancer classified night shift work as a probable carcinogen (Group 2A), the evidence for its carcinogenicity in humans remains "limited", partly due to heterogeneity observed across epidemiological studies. Using a systematic review and dose-response meta-analysis, we aimed to estimate the association between years of night shift work and incidence of various cancer types, while also exploring sources of heterogeneity.

Methods: Multiple electronic databases were systematically searched up to September 8, 2024. Incremental risk ratios (RRs) and corresponding 95% confidence intervals (95% CIs) were calculated for each study and pooled using a linear random-effects dose-response meta-analysis, conducted separately for each cancer outcome. **Results:** Seventy-two studies were included encompassing multiple cancer sites. While positive associations were observed for some cancers (pooled RRs and 95% CIs per 5-year increment in night shift work for cancers of the breast [1.03, 1.00-1.07]; prostate [1.06, 0.99-1.14]; colorectum [1.02, 0.99-1.05]; and lung [1.13, 1.00-1.28]), null associations were seen for others. For breast cancer, statistically significant positive associations were found only among case-control studies (1.05, 1.00-1.11) and studies capturing lifetime occupational history (1.07, 1.03-1.11), while cohort studies and studies not capturing lifetime history showed null associations. Studies with a higher risk of bias, particularly in exposure classification, tended to report null associations. **Conclusion:** Our results suggest that longer years of night shift work is associated with an increased risk of multiple cancer types in a dose-response manner. Heterogeneity across studies is apparent due to variations in exposure measures.

LAY SUMMARY

Key Messages: The relationship between working night shifts and developing cancer is not well understood, as studies have found mixed results. Our review found that people who work more years of night shifts may have a higher risk of certain cancers – especially breast, colorectal and prostate cancers. Our results suggest that capturing night shift work over a person's whole career and using detailed information may help identify workers at a higher cancer risk, which could guide workplace health policies and targeted cancer screening programs.

Cancer Risk and Prevention

Sunday, November 2, 2025

Environmental Carcinogenesis in Lung Cancer: Sodium Arsenite-Induced PML Dysregulation and LINE-1 Activation - Poster #147

Presenter: Kennedy Whelan, Dalhousie University

Other Authors: Sabateeshan Mathavarajah, Jayme Salsman, Victor Martinez, Alison Wallace, Graham Dellaire

ABSTRACT_ID#2632

ABSTRACT

Sodium arsenite (NaAsO₂) is a known environmental carcinogen linked to the development of lung cancer (LC), potentially through activation of LINE-1 retrotransposons (L1). The tumour suppressor promyelocytic leukemia protein (PML) inhibits L1 activity and the subsequent L1-induced inflammatory response of the cGAS-STING pathway. We are interested in exploring the role of PML and L1 expression as potential biomarkers for NaAsO₂ exposure and understanding how these factors contribute to the development of LC. Using immunofluorescence microscopy, we determined that human bronchial epithelial (HBE3-KT) cells and small airway epithelial (HSAEC1-KT) cells exposed to NaAsO₂ accumulated cytoplasmic PML-containing structures and decreased PML nuclear body (PML NB) abundance. We used RT-qPCR to assess L1 activation and found that NaAsO₂ treatment increased expression of endogenous L1. In addition, a L1 reporter assay confirmed that NaAsO₂ treatment upregulated L1 integration, and that this activity was suppressed by PML over expression. Finally, using a cGAMP ELISA assay we assessed the cGAS-STING pathway and found increased cGAS activity following NaAsO₂ treatment, which was reversed with PML overexpression. Our results indicate that NaAsO₂ activates L1 and degrades PML NBs, which play a role in L1 and tumor suppression. Activation of L1s by NaAsO₂ exposure, could lead to an increased risk of genomic instability via L1 retrotransposition in the nucleus, and chronic inflammation via cytoplasmic activation of the cGAS-STING pathway in response to L1 cDNA. Thus, we hypothesize that chronic exposure to NaAsO₂ in drinking water could increase LC risk by degrading PML and activating L1 retrotransposition.

LAY SUMMARY

We are exploring the protein PML and the LINE-1 retrotransposon as markers for arsenic exposure and how their relationship with arsenic leads to the development of lung cancer.

Cancer Risk and Prevention

Sunday, November 2, 2025

From Bench to Better Screening: Reference Gene Validation for a Blood-Based Gene Panel in Colorectal Cancer Diagnostics - Poster #149

Presenter: Hailey Langford, University of Winnipeg

Other Authors: Abinash Meher, Shiby Kuriakose, Mariam Ragheb, Harminder Singh, Anuraag Shrivastav

ABSTRACT_ID#2897

ABSTRACT

Reliable normalization using stable reference genes is essential for accurate gene expression analysis. Therefore, reference gene stability should be assessed for each disease state and experimental context, especially for complex diseases such as cancer. Colorectal cancer (CRC) remains a major global health concern and is ranked in the top three leading causes of cancer deaths worldwide. Previous findings from our laboratory demonstrated elevated expression levels of the two N-myristoyltransferase (NMT) proteins, NMT1 and NMT2, in the peripheral blood mononuclear cells (PBMC) of individuals with colorectal adenomatous polyps (AP) or cancer (CRC). Guided by these results, we initiated a study to determine whether NMT1 and NMT2 gene expression mirrors protein expression to design a less complex CRC screening test. Therefore, we sought to first identify stable reference genes across various colorectal pathologies, including CRC, AP, serrated polyps, hyperplastic polyps, and no evidence of disease (NED) samples. An initial screening evaluated the stability of a panel of 32 candidate reference genes in PBMC samples from the four pathophysiological and NED groups. From this, eight genes were selected and further validated in a larger cohort. Stability was assessed using multiple algorithms, including, geNorm, NormFinder, BestKeeper, and DCT. Our results identified four of the most stable genes (PSMC4, GUSB, PGK1, and EIF2B1) across four colorectal pathologies to be used for normalization in future gene expression studies. This study also highlights context specific differences in reference gene stability and supports the use of validated reference genes over conventional choices such as GAPDH and ACTB.

LAY SUMMARY

Finding stable reference genes is an important step toward a simple blood test to help detect early colorectal cancer by measuring specific gene activity. Reference (control) genes help ensure the results for disease markers are accurate by correcting for differences such as sample amount or test conditions. Now that we know which genes can be trusted as controls, we are one step closer to developing a reliable and simple blood test that looks at a group of key genes to help detect colorectal cancer early. Not all reference/control genes work the same for each tissue and/or cancer type. Genes that are usually used as controls are often expressed at different levels in different cancer and tissue/cell types. We had to determine which genes were the most reliable in our study population. The genes PSMC4, GUSB, EIF2B1, and PGK1 were identified as most stable and reliable in blood cells from people being screened for colorectal cancer. This will help improve future studies that look at gene activity in blood cells for general research and for studies focused on colorectal cancer.

Cancer Risk and Prevention

Monday, November 3, 2025

Physical Activity, Reproductive Factors, and Breast Cancer Risk: A Prospective Cohort Study in UK Biobank - Poster #150

Presenter: Lin Yang, Alberta Health Services

Other Authors: Andrew Harper, Hannah Harsanyi, Heinz Freisling, Sasha Lupichuk, Adetunji Toriola

ABSTRACT_ID#2550

ABSTRACT

Introduction: Reproductive factors such as early menarche, late menopause, nulliparity, and exogenous hormone use are well-established risk factors for breast cancer. Physical activity is associated with a lower risk of breast cancer, but whether this protective effect varies across reproductive risk factors is not well characterized. We investigated whether reproductive risk factors modify the association between physical activity and breast cancer risk. Methods: Females ≥ 40 years without a history of cancer from UK Biobank provided self-reported data on moderate-to-vigorous intensity physical activity and reproductive factors (2007-2010). Incident cancers were ascertained to 13 May 2022. Multivariable Cox regressions were used to examine the association between baseline physical activity and breast cancer risk. Additive and multiplicative interactions were assessed using interaction terms within Cox regression, and Relative Excess Risk due to Interaction (RERI), respectively. Results: The study followed 242 721 participants for a mean of 12.53 years (SD: 2.55); 8397 breast cancer cases were identified, including 748 pre-menopausal cases. Participants with high level of physical activity (≥ 50 metabolic equivalent hours (MET-hrs)/week) had significantly lower breast cancer risk (HR=0.87, 95%CI=0.82-0.93) compared to those with low level of physical activity (< 10 MET-hrs/week). This association was consistent across reproductive risk strata, including age at menarche ($\leq$ vs. > 12 years), parity (number of live births, age at first birth), age at menopause ($\leq$ vs. > 50 years), and use of oral contraceptive or menopausal hormone therapy (all P-value for RERI > 0.05). Conclusions: Physical activity is a useful risk reduction strategy for women, regardless of their reproductive history.

LAY SUMMARY

Being physically active reduces the risk of breast cancer. Women who engage in regular moderate to vigorous physical activity are less likely to develop breast cancer. Some reproductive factors increase breast cancer risk. For example, starting periods early, having no children, or using certain hormones can raise a woman's risk. Physical activity is beneficial no matter your reproductive history. Our study found that physical activity helps reduce breast cancer risk for all women, even those may have elevated risk due to reproductive history.

Cancer Risk and Prevention

Sunday, November 2, 2025

A Multicohort Analysis of Occupational Exposure to Endocrine-Disrupting Chemicals and Breast and Prostate Cancer in Canada - Poster #151

Presenter: Vikki Ho, Université de Montréal

Other Authors: Laura Pelland-St-Pierre, Marc-André Verner, Jérôme Lavoué, Trevor Dummer

ABSTRACT_ID#2712

ABSTRACT

Introduction: Sex hormones play a role in the etiology of breast and prostate cancer. Endocrine-disrupting chemicals (EDCs) may interfere with hormonal function and increase cancer risk, particularly in occupational settings where exposures can be higher than in the general environment. Objective: To assess associations between occupational exposure to EDCs and breast and prostate cancer risk. Methods: A case-cohort study was nested within three regional cohorts of the Canadian Partnership for Tomorrow's Health study. This study included 2,757 incident breast cancer cases in women and 2,322 incident prostate cancer cases. A subcohort of 2,606 women and 1,519 men was selected at baseline. Exposure to 17 EDCs in participants' longest-held job was estimated using CANJEM, a job exposure matrix. Participants were categorized into unexposed (probability=0), possibly exposed ($0 < \text{probability} < 25\%$) or exposed ($\text{probability} \geq 25\%$) for each EDC. EDCs were analyzed for exposure to all and grouped by mode of action: estrogenic, antiestrogenic, and antiandrogenic. Adjusted odds ratios (OR) and 95% confidence intervals (95%CI) were estimated in each regional cohort and pooled using fixed-effect models. Results: For breast cancer, compared to unexposed participants, participants possibly exposed to estrogenic EDCs (OR=1.30, 95%CI: 1.16–1.46), antiestrogenic EDCs (OR=1.23, 95%CI: 1.09–1.39), and antiandrogenic EDCs (OR=1.21, 95%CI: 1.07–1.38) had an elevated risk. For prostate cancer, possible exposure (OR=1.17, 95%CI: 1.01–1.36) and exposure to estrogenic EDCs (OR=1.15, 95%CI: 1.08–1.23) were associated with higher risk. Conclusion: Occupational exposure to EDCs, particularly estrogenic EDCs, was associated with breast and prostate cancer. However, findings should be interpreted cautiously given heterogeneity between regions.

LAY SUMMARY

How we studied workplace chemical exposure: We analyzed data from over 9,000 Canadians, including people with breast or prostate cancer and a comparison group without cancer. Using their longest-held job, we estimated exposure to 17 endocrine-disrupting chemicals, chemicals that can interfere with how our hormones work. What we found: People exposed at work to endocrine-disrupting chemicals, especially those that mimic estrogen, had a higher risk of breast and prostate cancer compared to those who were not exposed. Why it matters: Our study aims to help guide future prevention efforts. Our preliminary results suggest that workplace exposure to certain endocrine-disrupting chemicals may contribute to breast and prostate cancer risk. But, the findings should be interpreted with caution, as the analysis is still ongoing.

Cancer Risk and Prevention

Monday, November 3, 2025

The molecular function of breast cancer metastasis modulator MEMO1 - Poster #156

Presenter: Paria Nouri, University of Saskatchewan

ABSTRACT_ID#2764

ABSTRACT

MEMO1 (Mediator of ERBB2-driven cell motility 1) is a conserved, redox-sensitive protein implicated in breast cancer metastasis, though its molecular function remains incompletely defined. MEMO1 regulates cell motility and reactive oxygen species (ROS) production, and its structural similarity to iron-containing dioxygenases suggests a potential role in metal homeostasis. To investigate MEMO1 functional dependencies, we analyzed genome-wide single-gene knockout and knockdown data across 1,028 cancer cell lines and identified several copper-related genes that show genetic interactions with MEMO1. To validate these interactions, we performed shRNA-mediated knockdowns of each candidate gene in isogenic MDA-MB-231 cell lines with different levels of MEMO1 expression, high (wild type), reduced (M67-2) and complete knockout (M67-9). Knockdown of SOD3, PARK7, or ATP7A selectively impaired proliferation of high-MEMO1 cells compared to the MEMO1-knockout cells, demonstrating a gain-of-function genetic interaction and indicating that elevated MEMO1 expression increased cell dependency on copper-regulatory pathways. To assess MEMO1 effect on a copper-dependent cell function, we measured activity of lysyl oxidase (LOX), a copper-dependent enzyme involved in extracellular matrix remodeling. LOX activity was significantly reduced in MEMO1-knockout cells, further supporting a role for MEMO1 in copper-dependent biological processes. Collectively, these findings establish a role for MEMO1 in modulating copper-responsive signaling and copper-dependent enzymatic activity. Elevated MEMO1 expression creates a selective dependency on copper-related genes, revealing a context-specific vulnerability that may be exploited therapeutically in MEMO1-overexpressing breast cancers.

LAY SUMMARY

We discovered a copper-related vulnerability in certain breast cancer cells. Cancer cells with high MEMO1 levels are more affected when copper-regulating genes are disrupted, revealing a potential target for therapy. We identified key copper-related genes that interact with MEMO1. Using large-scale data and lab validation, we identified gain-of-function genetic interactions between MEMO1 and copper-related genes such as SOD3, ATP7A, and PARK7, where disruption of these genes more strongly affected MEMO1-high breast cancer cells. We showed that MEMO1 contributes to the activity of a tissue-remodeling enzyme. When MEMO1 is absent, the enzyme LOX becomes less active, which may reduce the ability of breast cancer cells to invade surrounding tissue.

Cancer Risk and Prevention

Sunday, November 2, 2025

Sunscreen is overwhelmingly promoted on TikTok, but content with misinformation exhibits proportionally high levels of audience interaction - Poster #157

Presenter: Lindsay Forsman-Phillips, Health Law Institute, University of Alberta

Other Authors: Alessandro Marcon, Marco Zenone, Vincenza Boniface, Cheryl Peters, Timothy Caulfield

ABSTRACT_ID#3143

ABSTRACT

Introduction The increasing global use of social media raises concerns about its influence on public health, particularly in cancer prevention. The rise of online health misinformation has been linked to issues like decreased vaccination rates, and the adoption of unproven and harmful health trends. This study addresses an observed surge in sunscreen misinformation on TikTok, where sunscreen is commonly portrayed as either toxic, prohibitive to benefits, or unnecessary. **Methods** To assess the portrayal of sunscreen on TikTok, we systematically analyzed the 971 most popular sunscreen-related TikToks, identified through the most popular relevant hashtags. We collected metadata, including views, likes, comments, and shares, to assess audience engagement. Statistical tests (Kruskal-Wallis and Dunn's post hoc) compared engagement metrics across content types. **Findings** Only 6.0% (58 TikToks) contained health-related critiques of sunscreen, and just 1.6% (16 TikToks) solely presented these critiques without promotion. Despite this low prevalence, misinformation garnered significantly higher audience engagement, with these 16 TikToks receiving substantially more mean likes, shares, and comments than promotional videos. While most TikToks (86.8%) promoted sunscreen, the focus was often on cosmetic benefits (e.g., anti-acne, anti-aging) rather than explicit cancer prevention. **Conclusion** Even infrequent social media misinformation can be influential due to its high engagement. This highlights the need to promptly address problematic narratives. While TikTok largely promotes sunscreen, its strong aesthetic and commercial emphasis may overshadow vital health information. Future health communication efforts should aim to balance engaging content with clear scientific information to effectively reach and inform diverse audiences.

LAY SUMMARY

Misinformation is a concern for population health, especially on popular social media sites like TikTok. **Low Prevalence of Misinformation, High Engagement:** While only a small percentage (6.0%) of popular sunscreen-related TikToks contained misinformation about health harms or preventing sun benefits, these misleading videos garnered significantly higher audience interaction (likes, shares, comments) compared to promotional content. **Misinformation's Disproportionate Impact:** The study suggests that even a small volume of misinformation can have a considerable and disproportionate impact on audience interaction, indicating that "rife" misinformation isn't necessary for it to be a concern. **Aesthetic Focus in Sunscreen Promotion:** The vast majority of sunscreen content on TikTok was promotional (86.8%), but it largely emphasized cosmetic benefits (e.g., acne, aging, texture) over explicit health benefits like cancer prevention. **Need for Targeted Public Health Response:** Public health agencies should not only monitor for emerging misinformation but also develop creative strategies to counter it effectively and engage audiences with accurate health information, especially given the "sticky" nature of sensational content.

Cancer Risk and Prevention

Monday, November 3, 2025

The Effect of Diet and Obesity on the Dopaminergic System: A Systematic Review - Poster #158

Presenter: Olivia Sharpe, University of Calgary

Other Authors: Lin Yang

ABSTRACT_ID#2842

ABSTRACT

Background Dopamine regulates motivation and behaviour, making it central to lifestyle interventions. Both diet and obesity affect dopamine signalling, but the nature of their interactions remain unclear, limiting effective intervention design. This review examines whether diet or obesity more strongly influences the dopaminergic system to inform cancer prevention strategies targeting lifestyle modification. **Methods** A comprehensive literature search was conducted across MEDLINE, EMBASE, SCOPUS, and Web of Science on July 24, 2024. Observational and interventional studies that investigated diet and/or obesity associated to dopamine-related outcomes in humans were included and organized into three categories based on their exposure and intervention: diet, obesity, diet and obesity. **Results** Out of 1,450 studies identified, 26 met the inclusion criteria. Six studies focused on diet, eight on obesity, and twelve examined both diet and obesity. Participant ages ranged from 14 to 63 years, with sample sizes of 5 to 435 individuals, across varied BMI ranges and sex distributions. Diet-only studies suggested that high-fat and high-sugar (HF/HS) diets may blunt striatal dopamine responses, while short-term dietary interventions can alter dopamine levels. Obesity-only studies found that higher BMI was associated with reduced dopamine signaling in reward regions. Studies examining both diet and obesity found that diet-induced changes in dopamine were often correlated with obesity status, with individuals of higher BMI showing attenuated dopaminergic responses to glucose or fat manipulations. **Conclusions** Current literature suggests that both diet and obesity can influence dopamine signaling. However, diet, particularly HF/HS intake, is the primary driver of changes in the dopaminergic system.

LAY SUMMARY

Both diet and obesity have been shown to influence the functioning of the dopaminergic system. This review examines whether diet or obesity independently affects dopamine-related brain function by analyzing studies focused on diet alone, obesity alone, and those that consider both. Studies on diet in isolation suggest short-term interventions involving excessive caloric intake or changes in macronutrient composition have been shown to increase dopamine release and excretion. However, habitual consumption of high-sugar/high-fat foods lead to blunted dopamine response in the brain and lower dopamine levels. Meanwhile, obesity is associated with reduced dopamine receptor availability and lowered dopamine synthesis capacity, along with structural changes in dopamine-related brain regions. Weight loss after bariatric surgery has been shown to partially restore dopamine receptor availability, however not to the level of age-matched lean controls. When obesity and diet were examined together, findings indicated that single administration dietary interventions resulted in increases in dopamine levels, but increases were smaller in individuals with obesity. Conversely, high-fat/high-sugar habitual dietary interventions altered the dopamine response to food independent of obesity measures. This suggests diet may directly modulate dopamine signalling, potentially influencing reward sensitivity and food motivation, independent of, but often interacting with obesity.

Cancer Risk and Prevention

Sunday, November 2, 2025

Unveiling Molecular Signatures of Arsenic-Induced Lung Damage: A Multi-Omics Approach Using Lung Models and Human Tissues - Poster #159

Presenter: Melis Erkan, Dalhousie University

Other Authors: Kennedy Whelan, Jayme Salsman, Alison Wallace, Graham Dellaire, Victor Martinez

ABSTRACT_ID#2633

ABSTRACT

Introduction: Arsenic, a natural carcinogen, contributes to lung cancer through chronic low-dose exposure. In Nova Scotia, arsenic in drinking water may underlie lung cancer rates, even among non-smokers. Arsenic induces gene expression changes associated with methylation alterations resulting from its biotransformation. We aim to identify biomarkers of arsenic-induced damage using lung models exposed to physiologically relevant arsenic concentrations. **Methodology:** We exposed human bronchial epithelial cells (HBEs) to varying concentrations of sodium arsenite. Cells were treated over 6 passages of 48–96-hour exposure cycles, with one group having arsenic removed after the final passage. DNA and total RNA were isolated from treated cells and sequenced using Illumina's platforms. Bioinformatics analysis will be performed via DRAGEN BioIT (DNA/RNA) and miRMaster (miRNA). **Results:** Coding and non-coding RNAs have been sequenced and processed. Methylation and DNA sequencing are in progress. Data quality assessment and normalization are ongoing for differential expression analysis with DESeq2. Our goal is to identify persistent arsenic-induced gene expression changes maintained during exposure, with a focus on regions exhibiting differential methylation. Next, we will examine irreversible molecular changes by removing arsenic post-exposure. Molecular signatures will be validated using samples from the Nova Scotia Lung Tumour Biobank and lung organoids to model chronic exposure. **Conclusion:** Our multi-omics approach will allow us to detect and correlate the relationship between methylation, transcription expression, and miRNA quantification. With this, we will create the scaffold for the clinically relevant genetic components of arsenic-specific damage and help develop prevention and early detection strategies for arsenic-induced lung cancer.

LAY SUMMARY

Chronic exposure to arsenic, a known human carcinogen, is known to cause cumulative damage to our cells and genetic material. This is thought to be contributing to the rising lung cancer incidences across Canada, especially Nova Scotia. We are hoping to use lab-made human tissues to determine the changes in human genetic material (DNA and RNA) over time, to understand how the damage accumulates. Our DNA and RNA are made of four fundamental building blocks (called "bases"), and we use "sequencing technologies" to determine the exact order of these bases in our samples. Then, we can use computational programs to detect any changes in the sequence of our bases and the genes they encompass, as genes control the creation, function, and quantity of significant molecules in our body. This is because specific changes to our genes can lead to cancer by altering or shutting down protective molecules and systems in our body. By identifying these changes and damages, we can pinpoint the "biomarkers" of arsenic-induced lung cancer. Biomarkers are the distinct signs of molecular damage, which can help us contribute to the development of effective strategies for prevention, intervention, and early detection for this devastating disease.

Cancer Risk and Prevention

Monday, November 3, 2025

Associations between classical risk factors and breast cancer molecular subtypes: luminal A, luminal B, HER2+ and Triple Negative: Findings from a large retrospective cohort study - Poster #160

Presenter: Ouerdia Haroun, CHU de Québec-Université Laval – Hôpital du Saint-Sacrement

Other Authors: Hermann Nabi, Flora Nguyen-Van-Long

ABSTRACT_ID#2870

ABSTRACT

Background: Breast cancer (BC) comprises distinct molecular subtypes—Luminal A (LA), Luminal B (LB), HER2-enriched (HER2+), and Triple Negative (TN)—which differ in incidence, prognosis, and treatment response, suggesting distinct etiologic pathways. This study investigated associations between common epidemiologic risk factors and BC molecular subtypes. **Methods:** We analyzed two cohorts of women diagnosed with primary invasive BC between 2000–2019 (n = 13,356) and 2000–2012 (n = 6,775), treated at a specialized breast disease center. Subtypes were classified using immunohistochemistry. Adjusted prevalence ratios (PRs) and 95% confidence intervals (CIs) were estimated using multivariable log-binomial regression models. **Results:** Risk factor associations varied by subtype. Women aged 60–69 had a higher prevalence of LA tumors (PR = 1.30; 95% CI: 1.22–1.37) but lower prevalence of LB, HER2+, and TN subtypes. Underweight status was associated with TN tumors (PR = 1.49; 95% CI: 1.11–2.00), while obesity was linked to LB tumors (PR = 1.45; 95% CI: 1.18–1.80). Past oral contraceptive use was associated with increased prevalence of LA and LB tumors, whereas current use was inversely associated with LA. In TN cases, postmenopausal status (PR = 1.68; 95% CI: 1.35–2.10) and prior hormone replacement therapy use (PR = 1.61; 95% CI: 1.25–2.03) were positively associated. **Conclusions:** These findings underscore the etiological heterogeneity of BC subtypes and support the development of subtype-specific prevention strategies informed by differential risk factor profiles.

LAY SUMMARY

Breast cancer is not just one disease—it includes different subtypes based on specific biological markers (like hormone and HER2 status), which affect how the cancer grows, how it's treated, and the chances of recovery. In this large study from Quebec, we found that certain risk factors—like age, menopausal status, weight, family history, and smoking—were linked to specific breast cancer subtypes. For example, older age was more often linked to the less aggressive Luminal A type, while obesity was linked to the more aggressive Luminal B type. These results can help guide prevention efforts by showing which lifestyle factors may increase the risk for more severe types of breast cancer. For instance, promoting healthy weight could help lower the risk of developing Luminal B breast cancer.

Cancer Risk and Prevention

Sunday, November 2, 2025

Canadian knowledge of cancer risk factors: Comparing survey results in 2021 and 2025 - Poster #161

Presenter: Melanie Matte, University of Calgary

Other Authors: Emily Heer, Samina Hossain, Alessandro Marcon, Marco Zenone, Cheryl Peters

ABSTRACT_ID#3256

ABSTRACT

Background: Cancer misinformation is abundant making it difficult for people to distinguish truth from myth. We compared responses from two surveys to determine whether there are differences in Canadians' ability to identify cancer risk factors in 2025 compared to 2021. Methods: In 2021, we conducted a survey of 734 Canadians to assess their agreement with cancer risk factors and, in 2025, we conducted a modified follow-up study of 2,003 Canadians. We analyzed results descriptively for both surveys, conducted correlations, and compared agreement across the two populations. Results: A greater proportion of participants in 2025 correctly identified alcohol, human papillomavirus, not getting enough fruits and vegetables, and not getting enough exercise as causes of cancer. However, the more recent cohort was worse at identifying several critical risk factors, most significantly for questions related to tobacco and sun exposure. Fewer participants identified smoking cigarettes (97.8% in 2021 vs. 89.5% in 2025) and second-hand smoke (92.6% vs. 83.4%) as causes of cancer. As for cancer prevention, fewer participants identified practicing sun safety (96.5% vs. 82.2%) as a cancer prevention activity. Conclusion: While there were some positive gains in cancer knowledge, Canadians in 2025 are generally worse at identifying basic and critical cancer prevention strategies than they were in 2021. Tobacco use and sun exposure are significant causes of cancer and this decrease in recognition should be alarming for public health organizations in Canada.

LAY SUMMARY

Cancer mis- and disinformation is making it more difficult for Canadians to know what is true and what is myth. Cancer misinformation is eroding long-fought public health campaigns about the harms of tobacco use and sun exposure. Canadians may be less knowledgeable about cancer risk factors now than they were in 2021.

Cancer Risk and Prevention

Monday, November 3, 2025

The prevalence of self-reported bowel symptoms among adults undergoing screening-related colonoscopy in Calgary, Alberta - Poster #162

Presenter: Paijani Sheth, University of Calgary

Other Authors: John Hutchinson, Dylan O'Sullivan, Robert Hilsden, Darren Brenner

ABSTRACT_ID#3230

ABSTRACT

Introduction Changes in bowel habits, including diarrhea, constipation, rectal bleeding, and abdominal pain are common symptoms of colorectal cancer (CRC). We evaluated the presence of self-reported bowel symptoms among patients undergoing screening-related colonoscopy and determined whether these varied by referral reason and other patient characteristics. **Methods** Self-reported bowel symptom data were collected from 331 adults at the Forzani & MacPhail Colon Cancer Screening Centre in Calgary, Alberta (2022–2025). Participants completed questionnaire regarding bowel symptoms experienced in the previous six weeks. We examined the proportion of study participants reporting various bowel symptoms overall, and stratified by age, sex and indication for colonoscopy. **Results** About thirty-five percent of participants (35.3%) reported no bowel symptoms, 19.3% reported having 1 symptom and 45.3% reported having 2 or more. The most commonly reported positive symptoms among participants were feeling like they have not fully emptied their bowels after a motion (37.5%), bloating (31.3%), and abdominal pain (30.5%). Women reported having approximately twice the proportion of rectum pain (19.9%), constipation (41.8%), bloating (43.3%) and abdominal pain (42.6%), compared to men (10.6%, 20.1%, 22.2%, 21.2%, respectively). Participants referred due to a positive faecal immunochemical test (FIT) alone reported a higher proportion of abdomen pain (37.6%) and rectum pain (15.8%) compared to other referral reasons (27.0% and 11.9% respectively). **Conclusions** Bowel symptoms are common prior to screening-related colonoscopy. Among participants undergoing colonoscopy after a positive FIT result, the relatively high percentage of non-bleeding symptoms is noteworthy. Future research will investigate associations between bowel symptoms and colonoscopy outcomes.

LAY SUMMARY

- Nearly two-thirds of patients undergoing screening-related colonoscopy reported experiencing at least one bowel symptom in the six weeks before their questionnaire. The most commonly reported symptoms were the feeling of not fully emptying the bowels, bloating, and abdominal pain.
- Compared to men, women in the study were twice as likely to report bowel symptoms. This suggests the existence of sex-based differences in symptom experience to be further explored.
- More patients referred for colonoscopy due to a FIT positive result reported non-bleeding symptoms like abdominal or rectal pain. This finding highlights the need to further explore how bowel symptoms relate to colonoscopy outcomes and whether they may provide additional information about patient triage.

Cancer Risk and Prevention

Sunday, November 2, 2025

Know Your Exposure: Insights from the development of a mobile phone app for cancer prevention research - Poster #163

Presenter: Melanie Matte, University of Calgary

Other Authors: Jeff Brook, Dany Doiron, Lin Yang, Kristian Larsen, Trevor Dummer, Cheryl Peters

ABSTRACT_ID#3341

ABSTRACT

Interest has increased in environmental exposures as they relate to human health. Air pollution, solar ultraviolet radiation and radon are just some of the exposures that are linked to cancer, however these exposures may be compounded for workers. In partnership with CANUE, Tactica Interactive and Alberta's Tomorrow Project, we developed an Android-based mHealth app to collect data and convey information about air pollution and occupational exposures, with the ultimate goal of reducing participants' exposure to carcinogens. A prototype app was developed with three components: Google Maps powered address search returned a nitrous dioxide (NO₂) exposure score in parts per billion GPS tracking feature calculated an NO₂ score based on aggregated commute coordinates Searchable occupation directory with occupational hazard information for four domains: hazardous substances, biological, physical & radiation and psychosocial. Generative AI chatbot ChatGPT was used to generate occupational hazard information, and Midjourney was used for the app logo. Study partners were essential to the app development phase to ensure exposure science was effectively translated into practical, user-friendly features. The use of AI to generate content was less effective than predicted and concerns about users' private personal information prevented the collection of GPS coordinates and personalized app features. This pilot study on the feasibility of using an mHealth app for cancer prevention research highlights the benefits and challenges of using mobile phone technology to engage participants. CanPath and other population health studies should be mindful of the challenges to maintaining user engagement and technological sustainability of an mHealth app intervention.

LAY SUMMARY

The use of mobile phone apps for cancer prevention research is challenging given the need to compete against so many other attention-grabbing apps. Generative AI does not perform well enough to be used preferentially in creating occupational exposure information.

Cancer Risk and Prevention

Sunday, November 2, 2025

Understanding Canadian time-use and environmental activity patterns for evaluating carcinogen exposures within the indoor and outdoor environments - Poster #165

Presenter: Monica Caicedo Roa, University of Calgary

Other Authors: Aaron Goodarzi, Cheryl Peters

ABSTRACT_ID#3253

ABSTRACT

In Canada, lung and bronchus cancer remains a significant public health concern. Multiple environmental factors contribute to lung cancer risk, including indoor radon exposure, occupational hazards (e.g., volatile organic chemicals, asbestos, arsenic), air pollutants such as combustion particulates from wildfires or vehicle fuel, and heavy metals. We analyzed time-use patterns of environmental occupancy in a cohort of over 6,600 people living and working in Canada from 2019 to 2025, spanning the pre-COVID-19 period to the 'new normal'. The analysis focused on time per year split between a person's (1) primary residence, (2) any other residential building, (3) non-residential buildings, and in (4) outdoors or in a vehicle of any type. Data were weighted as a function of employment and job type status to reflect the Canadian population using census benchmarks. Employment was a primary determinant of time-use distribution. From 2019 to 2025, the amount of time spent 'at home' per year increased by 7% (+600 h/y) for workers. Changes in environmental occupancy trends predominantly corresponded with a reduction in time spent in non-residential buildings in favour of residential housing. These changes were not equivalent amongst all Canadians, and differed by age, job, and community type (and more). The most significant increase in residential time was observed among working-age adults, who spent an additional 644.6 hours per year at home, equivalent to ~2.5 working months, thereby increasing potential exposures to indoor carcinogens such as radon. Time-use data provide valuable insights into the dosimetry of key environmental risk factors for lung cancer.

LAY SUMMARY

Understanding time-use patterns is essential for accurately assessing environmental-related cancer risk and guiding public health interventions. Time spent at home has increased substantially since the end of the COVID-19 pandemic response and has transitioned to a 'new norma' for the working population. With 1 in 5 residences in Canada containing high radon levels, these shifts in working habits mean that younger workers are, in particular, experiencing higher exposure to risk factors underlying lung cancer.

Cancer Risk and Prevention

Monday, November 3, 2025

Associations Between Glucagon-Like Peptide-1 Receptor Agonists (GLP-1 RAs) and Pancreatic Cancer Risk: A Systematic Review and Meta-Analysis - Poster #166

Presenter: Reynaldo Nambayan, University of Calgary

Other Authors: Reynaldo Nambayan, Isha Lalani, Chantelle Carbonell, Yibing Ruan, Robert Hilsden, Darren Brenner

ABSTRACT_ID#3196

ABSTRACT

Introduction: Obese or overweight individuals are at an elevated risk of developing multiple chronic illnesses, including at least 13 different types of cancer. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are a class of medications used to treat type-2 diabetes mellitus and weight-related comorbidities. GLP-1RAs may decrease risk of obesity-related cancer incidence and mortality. We conducted a systematic review and meta-analysis to synthesize the real-world evidence for the association between GLP-1RA use and cancer risk, including pancreatic cancer. **Methods:** We searched the MEDLINE and EMBASE databases from inception to April 30, 2025 for observational studies involving adults receiving GLP-1RAs. Comparators included adults receiving other antidiabetic or anti-obesity drugs. Studies reporting cancer incidence outcomes were included. Randomized controlled trials (RCTs) and case reports were excluded. **Results:** The literature search returned 1,386 studies published between 2014 and 2025. 120 studies were assessed for eligibility, of which 73 were included. 18 of the 73 studies investigated the association of GLP-1RA use on pancreatic cancer incidence. GLP-1RA use was associated with a statistically significant reduction in pancreatic cancer risk (relative risk RR, 0.67; 95% CI, 0.55 to 0.81) in comparison to other antidiabetic agents. **Conclusion:** GLP-1RA agents show potential protective effects against pancreatic cancer compared to other antidiabetic agents. Additional data are required to inform whether GLP-1RA may be useful for cancer risk reduction in a clinical setting.

LAY SUMMARY

People who are overweight or obese have a higher risk of developing certain cancers linked to obesity, including pancreatic cancer. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are medications that help treat type 2 diabetes, obesity, and other weight-related health problems. They may also help lower the risk of cancers linked to obesity. In comparison to other diabetes medications, GLP-1RAs appear to lower the risk of developing pancreatic cancer. However, more research is needed to confirm the protective effects of GLP-1RA against pancreatic cancer.

Cancer Risk and Prevention

Sunday, November 2, 2025

Why Don't They Want to Wear Sunscreen? Quantifying Anti-Sunscreen Messaging on TikTok - Poster #167

Presenter: Ari Meguerditchian, McGill University

Other Authors: Carla Herman, Nathalie Harb, Meghri Ghazarian, Eric Belzile, Nina Morena, Ari N. Meguerditchian

ABSTRACT_ID#2392

ABSTRACT

INTRODUCTION This study aims to quantify the prevalence of anti-sunscreen sentiments on TikTok and analyze their content and communication style. **METHODS** Videos tagged #nosunscreen on TikTok were collected in 07/24. Reviewers gathered video characteristics (username, date, captions, length, number of followers, likes, views, shares, and comments, sponsorship, filming location), user demographics, and communication styles. Users' opinions about not wanting to wear sunscreen were collected. Spearman's correlation coefficient, Chi-squared test, and Phi coefficient was calculated between variables. **RESULTS** 321 TikToks were selected. Average video length was 25 seconds. Mean number of followers was 90,053 (SD 32,4640). Mean number of views, likes, and comments was 73,121 (SD 408,245), 380,800 (SD 5,250), and 51 (SD 203), respectively. 12 videos included sponsorship. Female (78%) predominance was observed. 45% used music; 30% used audio memes. 32% were filmed outdoors. In 44%, a sunburn was shown. In half (49%), sunscreen sentiments were clearly described. Common reasons for not wanting to wear sunscreen include personal preferences (regarding trends), perceived benefits of natural sun exposure, sunscreen being viewed as unnecessary and/or harmful, choosing alternative forms of sun precaution, and the desire to tan. Believing it is cool/funny to be sunburnt is associated with having a reason to be anti-sunscreen, particularly the desire to tan. TikToks which convey irony and/or sarcasm (16%) are strongly associated with believing in the benefits of natural sun exposure and with choosing alternative forms of sun precaution. **CONCLUSION** YP may be influenced by humorous videos and by messaging about the perceived benefits of natural sun exposure.

Cancer Risk and Prevention

Monday, November 3, 2025

Built Environment Impact on Adherence to Cancer Prevention Guidelines, Cancer Incidence, and Mortality in a Cohort of British Columbians - Poster #168

Presenter: Sam Kenston, University of British Columbia

Other Authors: Rachel Murphy, Jerry Spiegel, Trevor Dummer

ABSTRACT_ID#2689

ABSTRACT

Background: Built environment characteristics, such as walkability and greenness, may influence health behaviours linked to cancer prevention. This study examined whether these exposures are associated with adherence to cancer prevention guidelines and subsequent cancer incidence and mortality. Methods: We analyzed data from 23,482 cancer-free participants in the BC Generations Project, linked to neighbourhood-level walkability and greenness measures from the Canadian Urban Environmental Health Research Consortium (CANUE). Adherence to five prevention guidelines (body weight, diet, physical activity, alcohol consumption, and smoking) was scored on a scale of 0 to 5 and categorized into low (0–1.5), moderate (2–3.5), or high (4–5) adherence levels. Associations between built environment and adherence were evaluated using multilevel linear regression. Cox proportional models estimated hazard ratios (HRs) for cancer incidence and mortality. Causal mediation analysis assessed indirect effects via adherence. Results: Higher walkability and greenness were associated with better adherence ($\beta = 0.18$, 95% CI: 0.14–0.23 and $\beta = 0.11$, 95% CI: 0.08–0.15, respectively). Participants in the highest walkability quartile had 15% higher odds of high adherence (OR = 1.15, 95% CI: 1.06–1.25). Walkability was associated with lower cancer incidence (HR = 0.89, 95% CI: 0.82–0.96) and mortality (HR = 0.87, 95% CI: 0.76–0.99). Greenness showed similar patterns. Mediation analysis indicated that adherence explained about 30% of the effect of built environment on cancer incidence. Conclusions: Neighbourhood walkability and greenness are positively associated with adherence to cancer prevention recommendations and reduced cancer outcomes. Urban planning strategies promoting these features may support cancer prevention at a population level.

LAY SUMMARY

Where you live matters for your health. People living in neighbourhoods with more green spaces and better walkability (e.g., access to sidewalks, parks, and shops) are more likely to adopt healthy habits, like staying active, eating well, avoiding smoking, and limiting alcohol consumption—habits that help prevent cancer. Healthy neighbourhoods are associated with lower cancer rates. Our study found that people in greener, more walkable areas had lower risks of getting cancer and were also less likely to die from it. The environment supports behavioural changes. People in healthier neighbourhoods have better cancer outcomes partly because their surroundings make it easier to live a healthy lifestyle.

Cancer Risk and Prevention

Sunday, November 2, 2025

Enabling new opportunities for exploring diet and activity across the cancer trajectory - comprehensive assessment of dietary intake and movement behaviours in the Canadian Partnership for Tomorrow's Health (CanPath) cohort - Poster #169

Presenter: Rachel A. Murphy, University of British Columbia

Other Authors: Alyssa D Milano, Michael J Widener, Michael Wallace, Travis J Hrubeniuk, Guy Faulkner, Benoît Lamarche, Charles E Matthews, Dylan Spicker, Trevor Dummer, Parveen Bhatti, Ellen Sweeney, Jennifer E Vena, Sharon I Kirkpatrick

ABSTRACT_ID#2743

ABSTRACT

Background: Healthy diets rich in vegetables and fruits, whole grains, and protein foods, reducing sedentary behaviour and increasing physical activity are cornerstones of cancer prevention. There is, however, a lack of detailed dietary intake and movement behaviour data in Canadian populations. The paucity of data limits public health surveillance and the development of evidence-based strategies to reduce the risk of cancer. The advent of online tools provides an opportunity for large-scale data collection in existing population cohort studies. Objective: The Healthy Eating and Supportive Environments (HEAL) study was launched in 2023 to address this critical data gap. Methods: HEAL is embedded in the Canadian Partnership for Tomorrow's Health (CanPath). All participants in CanPath (>330,000 adults) are being asked to complete an online demographic questionnaire and brief questionnaires about dietary intake and movement behaviour. Participants will then be invited to complete up to four online 24-hour recalls using the Automated Self-Administered Dietary Assessment Tool [ASA24-Canada] and the Activities Completed over Time in 24 Hours [ACT24]. Data will be pooled with the NutriQuébec cohort, and linked to previously collected CanPath data including behavioural, environmental, genomic, and biological samples. Results: Data collection began in 2025 with participant engagement strategies underway to address challenges, including the administrative and participant burden of using multiple tools over multiple timepoints. Conclusions: It is anticipated that dietary intake and movement behaviour data will be captured for over 100,000 participants between 2025-2027. Data will be available for researchers to answer numerous questions related to diet, movement behaviour and cancer.

LAY SUMMARY

The HEAL study is a large-scale initiative addressing a critical gap in Canada through collection of detailed dietary intake and movement behaviour data. The study leverages CanPath's extensive infrastructure to collect detailed data across multiple timeframes (past day, week, month, year) from over 100,000 participants. Although online tool administration can facilitate detailed data in large populations, strategies to increase participant engagement and retention are needed. The resulting dataset (including existing and new data) will be a powerful resource for a wide range of research questions related to cancer, as well as health equity, methodological advancements, precision health approaches, and urban design research. This effort will empower both researchers and participants in the pursuit of a healthier Canada.

Cancer Risk and Prevention

Monday, November 3, 2025

Make Healthy Changes Easy: A Qualitative Behaviour Change Investigation of Barriers and Facilitators to Reducing Sedentary Behaviour in Canada - Poster #170

Presenter: Hannah Harsanyi, Cancer Epidemiology & Prevention Research, Alberta Health Services

Other Authors: Nicole Slot, Apiramy Jeyapalan, Elizabeth Holmes, Andria Morielli, Tavis Campbell, Stuart Biddle, Christine Friedenreich, Lin Yang

ABSTRACT_ID#2557

ABSTRACT

Background: Prolonged sedentary behaviour is associated with adverse health outcomes, including cancer. Understanding determinants of sedentary behaviour across domains (transportation, occupation, and leisure time) may inform behaviour change intervention design; however limited data are available. This study identified determinants of domain-specific sedentary behaviour to inform intervention development for reducing sedentary time. Methods: One-on-one semi-structured interviews were conducted with a diverse sample of participants from across Canada. Thematic analysis of interview transcripts was used to identify barriers and facilitators to reducing domain-specific sedentary behaviour, which were characterized according to the Capability, Opportunity, Motivation- Behaviour (COM-B) model. The behaviour change wheel and behaviour change technique taxonomy were used to identify key components for intervention design. Results: Thirty participants were interviewed (63% female, 60% between aged 25-44, and 50% identified as white). While all participants endorsed potential benefits of and personal goals related to decreasing or breaking up their sedentary time, many had difficulty implementing these changes in their day-to-day life. Transportation and occupational sedentary behaviour were largely influenced by opportunity, with considerations related to weather, distance, resource availability, and social environment. In contrast, leisure time sedentary behaviour was predominantly related to motivational determinants, such as mood, habit, and goals. Conclusions: Environmental and policy-level interventions are likely required to meaningfully change transportation and occupational sedentary behaviour. Although targeted individual-level interventions focusing on capability and motivation could be useful for reducing leisure time sedentary behaviour, their design needs to consider opportunity-related barriers to create tailored reminders and recommendations suitable for each individual context.

LAY SUMMARY

Sedentary behaviour is any time spent sitting or lying down while doing activities that don't take much energy. High amounts of sedentary behaviour can increase a person's risk of cancer. There is little research on why people engage in sedentary behaviour, especially considering all different types of sedentary behaviour (at work, for transportation, or during leisure time). We interviewed Canadians from across the country to understand their reasons for having a high level of sedentary behaviour and what might help to change it. We found that people's sedentary time at work or during transportation was largely impacted by their physical and social environment. This was different than leisure time, which was more affected by their personal routine, interests, and mood. When trying to create strategies that help people reduce their sedentary time, personal motivators, like reminders or phone apps, might be useful for leisure time sedentary behaviour. This might be less useful in work or transportation settings, where changes to the environment may make a bigger difference. This could potentially involve improving public transportation, changing the way office spaces are set up, or making it easier for people to access things, like standing desks, bikes, sports equipment, or recreation centers.

Cancer Risk and Prevention

Sunday, November 2, 2025

Provision of advice to quit smoking by healthcare providers in Canada: Findings from the 2022 Canada International Tobacco Control Smoking and Vaping Survey - Poster #171

Presenter: Rafael Meza, University Of Waterloo

Other Authors: Shannon Gravely, Rafael Meza, Geoffrey T. Fong, Anne C.K. Quah, Andrew Hyland, K. Michael Cummings

ABSTRACT_ID#3218

ABSTRACT

Objectives: Quitting smoking substantially lowers cancer risk. Among Canadian adults who smoked cigarettes daily, we examined: (1) consultations with healthcare providers (HPs) in the past two years; (2) among those, the proportion who received cessation advice and the type of support recommended; (3) whether receiving cessation advice varied by social determinants of health (SDH) and health conditions. **Methods:** Data are from the 2022 Canada ITC Survey and included a national sample of 1163 Canadian adults who smoked cigarettes daily or quit smoking in the last two years but previously smoked daily. Respondents reported if they consulted with an HP, and if so, whether they were advised to quit smoking. SDH and health variables included sociodemographics, financial insecurity, depression/anxiety, lung disease, and alcohol use. **Results:** 48.7% consulted with an HP, and among those, 51.0% received cessation advice. Compared to those who did not receive advice, those who did were more likely to try to quit smoking (20.3% vs. 35.1%, $p < 0.001$). Among those who received quit advice, 46.8% were advised to use NRT, 32.0% varenicline, 14.0% bupropion, 13.3% a telephone/quitline, 14.5% a smoking cessation program, and 2.6% an e-cigarette. Adults with lung disease and greater interest in quitting were more likely to receive advice (both $p < 0.001$). There were no significant differences by SDH, mental health, or levels of alcohol use. **Conclusion:** The low rate of smoking cessation advice from healthcare providers underscores the need for systematic engagement with HPs across clinical settings to encourage quitting and to offer evidence-based methods to support cessation.

LAY SUMMARY

Healthcare providers (HPs) play an important role in helping people to quit smoking. In 2022, we found that among a nationally representative sample of Canadian adults who smoked cigarettes daily between 2020 and 2022 and reported consulting with an HP, half reported receiving any advice to quit smoking. We did not find any differences on receiving advice across social determinants of health. Mental health and heaviness of alcohol use were also not found to be statistically associated with receiving advice. As those with mental health conditions and alcohol dependence are more likely to struggle with quitting smoking, HPs should systematically screen for such conditions and offer advice on how to quit and to cope after quitting to maintain abstinence. Among Canadian adults who consulted with an HP, about half received advice to quit; of those who received advice, about 3 in 10 adults were not given a recommendation about a specific cessation aid to try. These results point to the need to increase engagement of Canadian HPs in all clinical settings to communicate with those who smoke about the importance of quitting and to provide specific information about methods that have been demonstrated to increase cessation.

Cancer Risk and Prevention

Monday, November 3, 2025

The Manitoba Tomorrow Project: Lessons learned in building a population-based cohort for cancer research - Poster #172

Presenter: Travis Hrubeniuk, CancerCare Manitoba

Other Authors: Shuchi Mistry, Allison Kolly, Donna Turner

ABSTRACT_ID#2468

ABSTRACT

Background: The Manitoba Tomorrow Project (MTP) is a prospective cohort composed of 10,000 adult Manitobans. The MTP serves as a platform for evaluating the effect of genetics, lifestyle, and other risk factors on cancer, designed to foster research improving cancer detection and prevention. Recruiting Manitobans required numerous mechanisms to be successful. The purpose of this analysis is to explore the demographics of participants who responded to each of the enlisted recruitment mechanisms. **Methods:** At enrollment participants reported how they heard about the MTP. Responses were divided into five categories: mailouts, social media, television/radio, referrals, and other. Participants subsequently completed a health and lifestyle questionnaire where they self-reported, among other things, personal demographics. **Results:** Of the 8,329 participants who reported how they were recruited, 72.3% were from the targeted mailout. Age, sex, marital status, education, and household income distribution among those recruited by the mailout aligned closely with the demographics of the cohort. Participants who responded to social media ($n = 468$) were younger (44.9 ± 10.8 years) than respondents to all other recruitment methods ($p < 0.05$) and were disproportionately female (87.0%; $p < 0.01$). Television/radio respondents ($n = 647$) were overrepresented by rural Manitobans (34.2%; $p < 0.01$) and those with a family history of cancer (67.3%; $p < 0.01$). **Conclusion:** Targeted mailout distribution was a successful mechanism for recruiting a large number of demographically diverse Manitobans. Social media was particularly useful for recruiting younger female participants, whereas television/radio advertisements and interviews aided in the recruitment of rural Manitobans.

LAY SUMMARY

Key Messages Personalized, target mailouts were the most effective method for recruiting a diverse set of participants. Social media is particularly useful for recruiting younger, female participants. Television or radio interviews/advertisements are more effective at recruiting participants who have had immediate family diagnosed with cancer.

Cancer Risk and Prevention

Sunday, November 2, 2025

Inflammatory memory and competitive advantage in human clonal hematopoiesis - Poster #173

Presenter: Stephanie Xie, University Health Network

ABSTRACT_ID#2293

ABSTRACT

A fundamental challenge in cancer biology is to understand why some people develop cancer and others do not despite similar genetic predispositions. Taking the blood system as an exemplar, we seek here to determine if the inflammatory milieu impacts the clonal fitness and competitive advantage of mutant tissue stem cells at the expense of wildtype clones. Hematopoietic stem cells (HSCs) must survive a lifetime of infection, sustaining life-long hematopoiesis. Yet, it is largely unknown how human HSCs respond and adapt to inflammatory stress, or whether inflammation-induced molecular changes in HSCs impact inflammation- and aging-associated human conditions. Here, we developed xenograft inflammation-recovery models and performed single-cell multiomics on human HSCs to empirically understand tissue stem cell adaptation to inflammatory stress. We identified two bona fide HSC subsets that are transcriptionally and epigenetically distinct. Only one subset, termed HSC inflammatory memory (HSC-iM), showed sustained molecular changes following recovery from human TNF or lipopolysaccharide treatments. Importantly, a transcriptional program reflecting HSC-iM was enriched in HSCs from recovered COVID-19 patients, in HSC from aged individuals, and individuals with clonal hematopoiesis (CH). HSC-iM were enriched for quiescence and hallmark inflammatory signatures, and showed limited hematopoietic output, while CH mutations released this differentiation block. Additionally, the pro-inflammatory HSC-iM transcriptional program was transmitted to downstream innate immune progeny. As many of the mature myeloid cells that respond to inflammation are extremely short-lived, HSC-iM may be the missing link to discover how inflammation in some people drives cancer risk and progression as we age.

LAY SUMMARY

1. Inflammation response in primary human blood stem cells is heterogeneous and makes lasting changes to blood stem cells isolated from people following severe COVID recovery, during aging and with clonal hematopoiesis.
2. We can model the effects of inflammation on human blood stem cells in human-mice hybrid experimental models and show that inflammation is sufficient to recapitulate changes we see in real people. 3) We propose we have found a subset of human HSCs that serve as a missing link to discover how inflammation in some people increases the risk for cancer but not in others.

Cancer Risk and Prevention

Monday, November 3, 2025

Phytochemicals can Ameliorate Estrogen Elevated Metastatic Mammary Line Mice Skin Tumor - Poster #174

Presenter: Tahir Abbas, HED Pakistan

Other Authors: Tehreem Fatima

ABSTRACT_ID#2305

ABSTRACT

Premenopausal estrogen elevation during hypothermy may cause cancer. During winter one to multiple tumors appear on neck, pectoral, pelvic, mammary line and lateral sides of female mice during or after lactation during 5 years observation. First tumor growth rate (length/width) was 1.30-3.66/1.08-3.13, second tumor 0.98-2.27/0.89-1.32 and third tumor was 0.82-1.12/0.88-1.14mm/day. Estrogen (8.35-23.5pg/ml), progesterone (1.086-2.41ng/ml), lymphocytes and TLC were significantly elevated while prolactin (0.5-0.43ng/ml), TSH (2.5-1.4uIU/ml), T3 (0.703-0.682pg/dl), T4 (3.416- 1.77ng/dl), Hb, neutrophils and monocytes were reduced, but FSH and LH remain unchanged comparatively controlled. Body weight was decreased by increasing size and weight of tumor. Animal survive for 65-80days after the appearance of first tumor. Histological sections (HE) of tumor specify scattered necrotic foci, neoplastic cells separated by fibrous stroma, lymphatic vessels with granular cytoplasm rosettes along with unevenly distributed mitotic figures. Splenomegaly and secondary tumor lobules in lungs indicate the metastasis. The hypothermia boost estrogen which initiate tumor activating factors to trigger melanocytic nevi cells originated neoplastic skin melanoma. Plant extract of Olive, Morus and Jambul (*Syzygium cumini*) for 30 days (0.2ml/12h) significantly reduce the appearance of second and third tumors in mice. Phytochemicals especially polyphenol of selected plants at optimum environmental temperature as epidrug can curb the methylation to provides effective protection against breast cancer. Co-treatment of ascorbic acid, anthocyanin, acetyl Co-A, carnitine, glutathione, L-Leucic acid, histidine, 2-fluoro-, L-, hydroxytyrosol and 5-methoxysalicylic acid as structural competitive molecules with arginine and lysine during epigenomic modulation, will be the targeted medicine against cancer in near future.

LAY SUMMARY

Low temperature (chilling effect) may cause elevation of estrogen in female, which trigger metastatic mammary line skin tumor with more targeted areas are lungs and spleen. The selected phytochemicals in their synchronized form have ability to block this metastasis and reduce the size of tumor. Control of temperature and proper use of fruit extracts before and during lactation of old age female is curative dot and such chemicals will be used to design epidrugs in near future.

Comparative Oncology and Model Systems

Sunday, November 2, 2025

Building Patient Avatars for Precision Medicine Using Tissue Engineering Techniques - Poster #175

Presenter: Parnaz Soori, University of Saskatchewan

Other Authors: Dean Chamberlain

ABSTRACT_ID#2451

ABSTRACT

There are not great systems to develop and optimize direct chemotherapy response testing for cancer. Canines can provide one such model. Canines have shared genetic and physiological traits with humans and live in the same environmental conditions, making them relevant for translational research. Dogs naturally develop cancers like humans, with tumours exhibiting spontaneous mutations and comparable histopathological features. These similarities, along with shorter lifespans, allow for accelerated study of tumour progression and therapeutic responses. Our project uses canine tumors to optimize personalized, precision treatment workflows. To do this we use tissue engineering techniques to create a 3D model of the canine tumors. This model aims accurately replicates the tumour microenvironment by incorporating extracellular matrix components, stromal cells, and tumour heterogeneity. We utilize 2 mm-long collagen-based microtissues embedded with the primary tumor cells. The microtissues provides an environment that simulates in vivo tumour characteristics for drug screening. We have optimized the digestion of the canine tumors and addition to the microtissues for growth. Within 5-10 days of growth, samples are exposed to various drugs, using concentrations based on the maximum safe dose for dogs, to identify effective treatments. Preliminary results show that the canine tumor microtissues give consistent results for drug treatment when repeatedly measuring the response of microtissues generated from frozen aliquots of primary canine tumor cells. We are currently optimizing the media formulation to improve the growth of the dog tumors and expanding the drug treatment to the 20 chemotherapy agents that canines are routinely treated with.

LAY SUMMARY

Dogs Get Cancers Similar to Humans: Dogs naturally develop cancers in a similar way to those in humans. This is due to similarities in how they start to develop and behave. This makes research in dogs more relevant than other animal models, and helpful for finding new cancer treatments that could also work in people. **New 3D Tumour Models Help Test Treatments:** We have created a new 3D model of dog tumours in the lab using tiny structures called Microtissues. These models can mimic real tumours within the body, allowing us to study how cancer grows and responds to different drugs in a way that's much more accurate than traditional methods. **Personalized Cancer Treatment:** By testing different chemotherapy drugs on these 3D tumour models, we can quickly find out which treatments work best for each individual dog's cancer. This approach brings us closer to personalized cancer care, improving the chances of successful treatment for both dogs and, potentially, humans.

Comparative Oncology and Model Systems

Monday, November 3, 2025

Tol mice overcome the immunogenicity of reporter proteins and enable improved in-vivo cancer-immunity and metastasis studies - Poster #176

Presenter: Areeba Qureshi, University of Toronto

Other Authors: Ping Xu, Hung-Yang Kuo, Ton Schumacher, Iacovos Michael, Robert Kerbel, Kabir Khan

ABSTRACT_ID#2734

ABSTRACT

Using reporter proteins, such as luciferase and fluorescent proteins, is critical for tracking tumour growth and dissemination in-vivo and distinguishing cancer cells from the tumour microenvironment post-implantation. However, the immunogenicity of reporter proteins in immunocompetent syngeneic mouse models often leads to an exaggerated immune response and subsequent rejection of implanted tumour cells, compromising the physiological relevance of these models for therapeutic studies. To address this, we investigated the utility of a previously described transgenic mouse, known as Tol mice, which exhibit immune tolerance to luciferase and various fluorescent proteins. Our study compared the growth of cancer cells implanted into the mammary fat pad (primary tumour) and intracranially (brain metastasis) in both wild-type (WT) and Tol mice. Bioluminescence imaging revealed complete tumour rejection in WT mice, whereas robust tumour growth was observed in Tol mice in both primary and metastatic settings. Interestingly, in WT mice where tumour growth was observed, administration of immune checkpoint inhibitors reinvigorated underlying immunity. Furthermore, in a subset of Tol mice with brain metastasis, a depletion of bioluminescence signal followed by reappearance after eight weeks, suggesting a state of tumour dormancy. These findings highlight that the Tol mouse model can effectively circumvent the issue of reporter protein-induced immune rejection. Moreover, Tol mice enable the establishment of physiologically relevant orthotopic and metastatic tumour models, which will be crucial for generating robust and reliable data on therapeutic responses. Future studies will further characterize the immune landscape in Tol mice and validate their utility across a spectrum of cancer models.

LAY SUMMARY

1. Foreign proteins used to tag cancer cells (reporter proteins) to facilitate imaging and tracking of tumor progression can trigger an immune response in wild-type (WT) mice, leading to false tumor rejection. Here, we employed a novel mouse model tolerant to reporter proteins (Tol mice) to investigate whether we can enhance the current models and observe tumor growth and therapy responses without introducing bias.
2. The Tol mice were permissive to the growth and imaging of cancer cells tagged with reporter proteins, allowing for improved modelling of primary breast cancer and breast cancer brain metastasis. In contrast, reporter-tagged cells were rejected upon implantation in WT mice.
3. The Tol mice enabled a precise examination of the impact of cancer therapies designed to reactivate the immune system to identify and attack cancer cells. In contrast, similar experiments yielded biased results in wild-type mice due to the foreign nature of the reporter proteins.

Comparative Oncology and Model Systems

Sunday, November 2, 2025

Transcriptomic landscape and clustering of soft tissue sarcomas via snRNA-seq in a spontaneous canine model - Poster #177

Presenter: Arata Matsuyama, University of Guelph

Other Authors: Ryohei Yoshitake, Kohei Saeki, Helene Philibert, Behzad Toosi

ABSTRACT_ID#2684

ABSTRACT

Introduction: Soft tissue sarcomas (STS) are common cancers with limited therapeutic options. Spontaneously occurring STS in dogs serve as an excellent comparative model for human STS, sharing their clinical behavior, biological characteristics, and genomic and transcriptomic landscapes. To uncover the transcriptomic profiles at single-cell resolution and potential therapeutic targets of STS, we conducted a pilot study of snRNA-seq on canine STS samples. **Methods:** Nuclei were isolated from eight snap-frozen canine STS samples. Libraries were prepared using Chromium kits and indexed cDNA libraries were sequenced on a NextSeq 550 with an aimed sequencing depth of 20,000 paired end reads per cell. Data was processed by Cell Ranger (v8.0) using the canine reference genome CanFam6. Seurat (v5.0.2) and VISION (v3.0.2) in R (v4.3.3), and CellBender (v0.3.0) and CellPhoneDB (v5.0.1) in Python (v3.8.20) were used for visualization. **Results:** Transcriptomic profiling revealed five distinct cell clusters, with tumor cells comprising the majority (70–90%) of the cellular population. These tumor cells displayed a fibroblastic phenotype and showed up-regulation of receptor tyrosine kinases, such as EGFR and FGFR. Immune cells represented the second largest population (10–18%) and were composed of macrophages expressing M2-associated markers. Ligand-receptor analysis highlighted tumor–endothelial interaction, suggesting the M2 macrophage and endothelial cells as candidate therapeutic targets. **Conclusion:** This pilot study demonstrates the feasibility of snRNA-seq in canine soft tissue sarcomas and reveals key tumor and immune cell populations within the tumor microenvironment. Our findings highlight tumor–immune and tumor–endothelial interactions as potential targets for future therapeutic exploration.

LAY SUMMARY

We successfully performed RNA sequence analysis at single cell level to study the cells within cancer tissue environment in a relatively rare cancer called soft tissue sarcoma in dogs. Our results showed that gene expression patterns in not only cancer cells but also various immune and blood vessel cells. They which could play a role in how the cancer grows or responds to treatment. We identified specific communication signals between different cells that may be promising new targets for developing future therapies.

Comparative Oncology and Model Systems

Monday, November 3, 2025

Chemoresistance-associated transcriptomic alterations in carboplatin treated canine osteosarcoma cell lines - Poster #178

Presenter: Jared R. Fischbach, Ontario Veterinary College, University of Guelph

Other Authors: Courtney Schott

ABSTRACT_ID#3231

ABSTRACT

Osteosarcoma is the most prevalent malignant primary bone cancer in humans and dogs. In both species, metastatic progression occurs despite aggressive treatment with chemotherapeutics. Chemoresistance represents a formidable challenge to improving clinical outcome as the underlying mechanisms driving chemoresistance in osteosarcoma remain incompletely understood. To facilitate comprehensive investigations into these mechanisms, we established a clinically-informed in vitro model by exposing five canine osteosarcoma cell lines to carboplatin under conditions translated from the standard-of-care adjuvant chemotherapy regimen and associated pharmacokinetics in canine patients. Differential responses in chemosensitivity were observed across the five carboplatin treated cell lines relative to their controls. Three cell lines (OVC-cOSA-75, OVC-cOSA-111Pa, OVC-cOSA-111Ma) displayed significant increases in carboplatin resistance of 1.24-, 1.88- and 2.89-fold, respectively, while two cell lines (OVC-cOSA-31, D-17) showed no significant change ($p < 0.05$; one-sided Z-test). RNA sequencing of treated and control cell line pairs revealed distinct transcriptional changes exclusive to the subset that developed a resistant phenotype. Gene Ontology enrichment analysis was conducted on differentially expressed genes; the pathways nuclear estrogen receptor activity and 3',5'-cyclic-GMP phosphodiesterase activity showed the highest recall among enriched terms, indicating a strong representation of these categories within the resistant phenotype. In contrast, cell lines that did not develop resistant phenotypes in vitro lack comparable transcriptional signatures, reinforcing the specificity of observed changes to the development of acquired carboplatin resistance. This study highlights the value of integrating functional drug sensitivity insights with transcriptomic profiling to elucidate the mechanisms driving carboplatin resistance in osteosarcoma.

LAY SUMMARY

Osteosarcoma is the most common bone cancer in dogs. Even with aggressive chemotherapy treatment, most dogs will rapidly develop fatal lung tumours. Chemotherapy resistance in canine osteosarcoma may be linked to specific genetic changes. An example of these changes is altered expression of genes which regulate cell signalling and communication. Identifying the unique genetic changes that contribute to the development of chemotherapy resistance may help researchers establish improved treatments for affected patients.

Comparative Oncology and Model Systems

Sunday, November 2, 2025

Dysregulated microRNAs as independent prognostic markers of survival in human and canine osteosarcoma: a cross-species review - Poster #179

Presenter: Brittany Naeckel, University of Guelph

Other Authors: Geoffrey Wood

ABSTRACT_ID#3121

ABSTRACT

Osteosarcoma is the most prevalent primary bone malignancy in both humans and dogs, characterized by aggressive metastatic behavior and poor long-term survival despite intensive treatment. Given the biological and clinical parallels between species, canine osteosarcoma serves as a valuable translational model for the human disease. As survival outcomes remain poor, there is a critical need for non-invasive biomarkers that can predict prognosis and guide clinical decision-making. Circulating microRNAs have emerged as promising biomarker candidates due to their stability in biofluids, accessibility through minimally invasive sampling, and evolutionary conservation. We aimed to identify microRNAs with consistent prognostic value, based on the hypothesis that dysregulated microRNAs associated with poor clinical outcomes in both humans and dogs may serve as reliable, independent markers of survival in osteosarcoma. A total of 78 studies (2011–2025) reporting dysregulated microRNAs in human and canine plasma, serum, tissue, and cell lines were reviewed. Across species, more than 50 dysregulated microRNAs have been reported, with over 48 associated with clinically significant features such as tumor size, metastasis, stage, and chemoresistance. Importantly, 33 microRNAs were identified as independent prognostic markers of survival. Strong cross-species candidates included miR-9, miR-34a, miR-126, miR-133b, and the miR-214 family. In human studies specifically, miR-21, miR-22, and miR-221 demonstrated particularly strong evidence. Future research will analyze plasma from human osteosarcoma patients. Pooled samples will undergo whole miRnome PCR array screening, followed by RT-qPCR measurement of individual samples. Expression patterns will be statistically correlated with clinical outcomes to identify reliable, circulating microRNA prognostic biomarkers.

LAY SUMMARY

We identified over 50 microRNAs that were differentially expressed across serum, plasma, cell lines, and tumor tissue from humans and dogs with osteosarcoma, 33 of which were confirmed to predict a patient's survival outcome regardless of other clinical factors. Over 48 microRNAs were correlated to poor clinical characteristics, such as tumor size, metastasis, stage, grade, recurrence, and chemoresistance. Several microRNAs, including miR-9, miR-34a, miR-126, miR-133b, and the miR-214 family, showed consistent associations with poor clinical survival outcomes in both species, while miR-21, miR-22, and miR-221 were specific to human studies.

Comparative Oncology and Model Systems

Monday, November 3, 2025

Determining the Cellular Localization of MicoRNAs in Canine Osteosarcoma - Poster #180

Presenter: Sophia Fernandes, University of Guelph Ontario Veterinary College

Other Authors: Geoffrey wood

ABSTRACT_ID#2826

ABSTRACT

Osteosarcoma (OSA) is the most common primary bone malignancy in both humans and dogs, with a high incidence in large-breed canines. Despite ongoing research, there have been no major advancements in treatment over the past three decades, and the disease remains highly lethal in both species due to its aggressive nature and high metastatic potential. MicroRNAs (miRNAs) are small non-coding RNAs that regulate gene expression and have emerged as key regulators of cancer progression. MiRNAs influence tumour cell proliferation, invasion and metastasis, making them potential prognostic biomarkers and therapeutic targets. This study investigates the expression patterns and cellular localization of miRNAs in canine OSA using in situ hybridization (ISH) on tissue microarrays (TMAs), containing matched primary tumors and metastatic lung lesions. Our goal was to determine the cellular source and spatial distribution of miRNAs already known to be differentially expressed between tumour tissue and healthy tissue, as well as between primary and metastatic lesions. While some miRNAs are known to be highly expressed in tumours, we do not know if the cellular source is neoplastic cells, immune cells, or stromal cells. ISH allows identification of specific cell populations within the tumour contributing to miRNA expression. Digital image analysis will quantify miRNA expression, and statistical survival analysis will evaluate correlations between expression and clinical outcomes. Due to genetic similarities between human and canine OSA and the high homology of miRNA across species, these findings could aid in developing miRNA-based diagnostics and targeted therapies, enhancing prognosis and treatment in both species.

LAY SUMMARY

MiRNAs appear to be created by tumour cells, rather than blood vessels or immune cells that also make up the bulk of the tumour. MiRNA expression levels change in primary tumours compared to corresponding metastatic tumours. Higher miRNA expression correlates with clinical outcome.

Comparative Oncology and Model Systems

Sunday, November 2, 2025

Revolutionizing Ovarian Cancer Research: Unveiling the Power of 3D Microtissues - Poster #181

Presenter: Breanne Bevelander, University of Saskatchewan

Other Authors: Reza Javan, Parnaz Soori, Taylor Dzikowski, Devrie Johnston, Nima Daneshvar Baghbadorani, Lacey Winstone, M. Dean Chamberlain

ABSTRACT_ID#2401

ABSTRACT

Ovarian cancer is among the most common and deadly cancers in those assigned female at birth. It is complex and heterogeneous, yet primarily studied using simplistic 2D cell culture methods. This oversimplification limits our understanding of chemoresistance, and the tumour microenvironment. To address this, we have developed a novel 3D model, termed “microtissues,” that incorporate spatial complexities often overlooked in 2D cell culture. Preliminary data indicate that microtissues facilitate cell migration and preserve characteristics of tissue architecture that typically are not found in traditional culturing systems. Histological analyses illustrate this in that cells within the microtissues spontaneously organize into structures representative of their histotype. Additionally, we have conducted in vivo testing of microtissues. When ovarian cancer cells are embedded in microtissues prior to injection, they develop tumours more rapidly compared to cells injected with Geltrex. Furthermore, we have focused on cryopreserving our microtissues while retaining their morphology and functionality, allowing us to share our innovative model with collaborators and enhancing the sustainability of our research. Thus far, our microtissues have proven to be readily cryopreservable, maintaining viability upon thawing. Further, within 48 hours of thaw, microtissues appear to normalize their proliferation rate to that of microtissues grown under normothermic conditions. Once confirmed across additional cell lines, cryopreserved microtissues will undergo chemotherapeutic testing to assess whether the cryopreservation process impacts experimental outcomes. With these applications and many more, microtissues have the potential to be incredibly valuable in cancer research and beyond.

LAY SUMMARY

Ovarian cancer is an extremely complex and devastating disease that affects thousands of Canadians each year. This complexity, and the chemoresistance that results from it, must be appropriately modeled in vitro to improve patient outcomes. While simplistic 2D models have been crucial in building our current understanding of cancer biology, a more complex understanding of tumour microenvironment is needed. We have worked to generate a new 3D model, termed “microtissues”, to mimic the complexities of cancers like ovarian cancer. This model has proven useful in mice studies and has shown varied gene expression and chemotherapy response from other cancer models, suggesting microtissues may provide novel insights into cancer biology that other systems fail to capture. The most exciting difference our model has shown is that when ovarian cancers of different types are placed in our microtissues, the cells spontaneously move and form the structures that are characteristic of that ovarian cancer type, making our model a good candidate in the push towards precision medicine. This cell-directed behaviour highlights our model’s potential as a pre-clinical tool towards precision medicine.

Comparative Oncology and Model Systems

Monday, November 3, 2025

Evaluating the impact of MYC amplification on dinaciclib response in canine and human osteosarcoma - Poster #182

Presenter: Emma N. Vanderboon, University of Guelph

Other Authors: Emma N. Vanderboon, Jared R. Fischbach, Amélie L. Delègue, Vanessa M. Leung, Courtney R. Schott

ABSTRACT_ID#3319

ABSTRACT

Osteosarcoma is the most common primary malignant bone tumour in humans and dogs. It is characterized by aggressive metastatic behaviour, genomic complexity, and poor outcome, with limited therapeutic advancements made in decades. Canine and human osteosarcoma share many genomic alterations and disease progression patterns, particularly pulmonary metastasis, highlighting opportunities for comparative therapeutic investigations. One shared genomic alteration is amplification of the oncogene MYC, a critical regulator of transcription, replication, and cell cycle progression. MYC copy number (CN) gains occur in ~40% of osteosarcoma in both species and are associated with poor prognosis in humans. Dinaciclib, a CDK1/2/5/9 inhibitor, has shown preclinical efficacy in MYC-amplified human osteosarcoma by reducing MYC protein expression and decreasing cell viability in vitro, as well as suppressing tumour growth and metastasis in vivo. While approaches that indirectly modulate MYC expression have shown promise in human osteosarcoma, studies in canine models remain limited. We hypothesize that dinaciclib's effect on proliferation, migration, anchorage-independent survival, invasion, and colony formation in vitro will be greater in osteosarcoma cell lines overexpressing MYC. To test this, we will (1) define MYC status in 14 canine and 10 human osteosarcoma cell lines using CN analysis (ddPCR) and protein expression (western blot), and (2) compare dinaciclib response in MYC-amplified versus non-MYC-amplified osteosarcoma cell lines using an in vitro assay pipeline. Preliminary data shows MYC protein expression aligns with CN in human lines. This study will provide insight into the therapeutic relevance of MYC amplification in osteosarcoma and inform future strategies that exploit MYC-associated vulnerabilities.

LAY SUMMARY

Bone cancer (osteosarcoma) in dogs and humans is very similar: Dogs also naturally develop the disease, and their tumours share many of the same genetic changes with human tumours, including sometimes having too much of a protein called MYC, which is known to increase tumour growth. Knowing how many copies of the MYC gene a patient's tumour has could help guide treatment: In both dogs and people, about 40% of osteosarcoma cases have extra copies of the MYC gene. This is a marker for more aggressive cancer and worse outcome in human patients. Tumours in dogs with high MYC levels may also be more sensitive to a drug called dinaciclib, which has been shown to block some of MYC's effects when tested in human osteosarcoma cell lines. Studying osteosarcoma in dogs helps advance patient care for both species: By testing how MYC levels affect response to dinaciclib in bone cancer cell lines from dogs and humans, researchers could identify a new way to treat this aggressive cancer. This comparative approach could lead to more effective treatment approaches that improve outcome and quality of life for both dogs and people with osteosarcoma.

Comparative Oncology and Model Systems

Monday, November 3, 2025

The Ontario Veterinary College Veterinary Biobank: Facilitating Translational Cancer Research - Poster #184

Presenter: J. Paul Woods, Ontario Veterinary College University of Guelph

Other Authors: Deirdre Stuart, Charly McKenna, J. Paul Woods, Brenda Coomber, Michelle Oblak, Geoffrey Wood

ABSTRACT_ID#3242

ABSTRACT

Research in veterinary medicine including comparative oncology is often limited to prospective clinical trials due to a scarcity of available retrospective samples. A lack of veterinary biobanks in Canada creates a significant gap in veterinary and translational research. Standardized veterinary biobanks facilitate access to research samples. Registration, certification, and training through the Canadian Tissue Repository Network (CTRNet) and International Society for Biological and Environmental Repositories (ISBER) maintain consistency and quality. Standardized client consent practices, data collection including diagnostic reports and treatment outcomes, and collection of biological samples within pre-determined times and processing into derivative samples strengthens research potential. Outcome and treatment data is stored in secure anonymized databases to facilitate retrospective analysis. The Ontario Veterinary College Veterinary Biobank (OVCVB) was established in 2009 as the first of its kind in Canada. Currently OVCVB has almost 2,000 cases and continues to grow, including serum, plasma, buffy coat, urine, and tissue. A wide variety of neoplasms have been collected: the most prevalent in dogs are soft tissue tumour (STT), lymphoma, and osteosarcoma (OSA); in cats, STT, mammary carcinoma, and OSA. The OVCVB is a unique resource with the mission to facilitate research to improve the lives of companion animals and people through the collection and distribution of veterinary patient samples and clinical data. Through the Ontario biobank network and regulatory bodies such as CTRNet, the increased awareness of disease similarities in humans and pets and improved quality of sampling allow for greater access to samples that contribute to more impactful translational research!

LAY SUMMARY

The Ontario Veterinary College Veterinary Biobank (OVCVB) stores samples (blood, serum, urine, tissue) collected from dogs and cats affected by spontaneous cancers presented for care to the Ontario Veterinary College Health Sciences Centre. The OVCVB contains samples from over 2,000 cases so far. Many of these companion animals were treated with standard of care therapy (surgery, radiation, chemotherapy, immunotherapy, etc.) and some were enrolled in veterinary clinical trials. The OVCVB also stores the clinical outcome of the veterinary patients' treatment. Samples and data from the OVCVB have been used in 36 internal and external projects, including biomarker analysis and test validation. The OVCVB is a unique resource that supplies banked samples with anonymized clinical data to veterinary and physician-based cancer researchers to facilitate a better understanding of cancer in both people and pets!

Comparative Oncology and Model Systems

Sunday, November 2, 2025

Clinical and immunological factors of recurrence in canine soft tissue sarcoma - Poster #185

Presenter: Kazuki Mizuta, Western College of Veterinary Medicine, University of Saskatchewan

Other Authors: Arata Matsuyama

ABSTRACT_ID#2585

ABSTRACT

Introduction: Soft tissue sarcoma (STS) is a relatively rare heterogeneous group of mesenchymal tumors. While treatment focuses on local control, how immunological factors contribute to time to post-surgical recurrence (TR) is poorly understood. Regulatory T cells (Tregs), known for their immunosuppressive roles, are emerging immunotherapy targets. As a translational human STS model, we investigated the association between STS recurrence and immune factors in spontaneously developed canine STS. Methods: Client-owned pet dogs with recurrent STS were included. Formalin-fixed paraffin embedded tissues were used for immunohistochemistry of immune related proteins and nCounter Canine Immuno-Oncology panel. Results: Tumor location was an independent risk factor [$p = 0.049$] with tumors on the neck/head having the shortest TR. Tumor-infiltrating Treg was significantly increased in neck/head STS than in trunk [$p=0.020$]. Elevated Treg infiltration was associated with shorter TR [$p=0.007$] and the presence of tertiary lymphoid structures (TLSs) in the tumor tissue [$p=0.002$]. Among 780 immuno-oncology genes analyzed, NOD2 expression independently affected TR [$p < 0.001$]. Unsupervised clustering identified two groups with significantly different TR (median 99 [Cluster 1] vs 669 days [Cluster 2; $p = 0.005$]). Cluster 1 was enriched for Treg-related genes and immunosuppressive pathways including IL10 signaling, while Cluster 2 showed reduced Treg activity via adenosine A2A pathway and enhanced pro-inflammatory responses. Conclusion: Time to recurrence was influenced by tumor locations, NOD2 expression, and the immune microenvironment including tumor-infiltrating Tregs. Further studies on TLS and its contribution to the tumor microenvironment are warranted.

LAY SUMMARY

Tumor-infiltrating regulatory T cells may contribute to earlier recurrence in canine soft tissue sarcoma. Tertiary lymphoid structures are associated with increased regulatory T cell infiltration. Tumor location, NOD2 expression, and immune contexture in the tumor microenvironment affect time to recurrence in canine soft tissue sarcoma.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

The Emerging Role of NEK10 Kinase in Triple Negative Breast Cancer - Poster #186

Presenter: Benjamin Ulmer, Lady Davis Institute for Medical Research

Other Authors: Catherine Chabot, Adriana Aguilar-Mahecha, Mark Basik

ABSTRACT_ID#2783

ABSTRACT

Targeted therapies for the most aggressive subtype of breast cancer, triple negative breast cancer (TNBC), so-called because of the lack of expression of estrogen, progesterone and HER2 receptors, are limited. To discover new targets for therapy in TNBC cells, a high-throughput shRNA screen for essential genes was performed on a unique patient-derived TNBC cell line. One of the top genes identified using this screen was NEK10, a member of the NIMA family of kinases best known for their role in cell cycle control. Previous studies have demonstrated that NEK10 is capable of phosphorylating multiple key cancer-related substrates, including p53 and beta-catenin, and NEK10 depletion is known to be associated with both mitochondrial and ciliary dysfunction, suggesting a diverse array NEK10 phosphorylation targets. Preliminary experiments using siRNA against NEK10 results in marked inhibition of proliferation and induction of apoptosis in multiple TNBC cell lines, but not in non-cancerous HEK293 cells, suggesting that there is a therapeutic window for the use of NEK10 inhibitors in TNBC. Parallel experiments using shRNA against NEK10 gave concordant results; increased NEK10 silencing was associated with reduced proliferation in breast cancer cells. Inhibition of NEK10 also ablates proliferation in TNBC cells lacking p53, demonstrating that the observed effects of NEK10 inhibition are not mediated by p53-related signaling. This work presents evidence that NEK10 inhibition should be further tested as a potentially viable treatment strategy in the context of TNBC.

LAY SUMMARY

A screen performed in patient-derived cells (produced from patient-derived xenografts) designed to identify novel therapeutic vulnerabilities in triple negative breast cancer identified a kinase called NEK10 as a potential target. NEK10 is a relatively understudied protein with putative roles in the regulation of cilia, mitochondria, and cell cycle checkpoint control. Depletion of the NEK10 kinase inhibits the proliferation of a variety of breast cancer cell lines, while NEK10 depletion has no such effect in noncancerous cell lines, suggesting that NEK10 represents a specific vulnerability of breast cancer cells. NEK10 depletion led to apoptosis in both triple negative and ER positive cells. The inhibition of proliferation which is observed upon NEK10 depletion appears not to depend on any previously described NEK10 phosphorylation target, suggesting a novel mechanism.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Better Research Through Patient Collaboration - Poster #187

Presenter: Anu Shukla-Jones, BioCanRx

Other Authors: Mackenzie Huckvale, Megan Mahoney, Stephanie Michaud

ABSTRACT_ID#2366

ABSTRACT

Patient engagement in research has demonstrated benefits for researchers, patients, and for achieving desired outputs and outcomes of health research. BioCanRx is a not-for-profit national funder of translational research for cancer immunotherapies in Canada, and since 2017, has been exploring ways to meaningfully enable patient partnerships within the research ecosystem. But what does meaningful partnership between patients, researchers, trainees, and research funders look like in practice? Do patient partnerships result in better research? How can research funders enable patient partnership in the ecosystems they support? What have investments in patient partnership contributed to research and the advancement of cancer immunotherapies? What are the broader impacts of patients, researchers and research funders better understanding one another? What are the current and evolving challenges? These questions will be explored using examples of patient co-designed initiatives from BioCanRx's translational research program and Cancer Community Partnership. Examples range from the Learning Institute, an immersive patient-trainee development program, to the inclusion of patients as required partners on translational research projects, to targeted support for research exploring best practices in patient engagement. All these and more will be used to help the audience learn about how patients have informed BioCanRx's priorities and contributed to cancer immunotherapy research, with the goal of inspiring organizations and individuals across the health sector to more creatively consider how patients can be meaningfully engaged in their own activities.

LAY SUMMARY

Meaningful partnerships between patients and patient-focused organizations and researcher funders, researchers, and trainees improves decision-making, prioritization, research practices, and higher quality translational / pre-clinical research. Health research culture is changing to more meaningfully include patients; however these behavioural changes will take time, appropriate incentive structures, and the right mindset. BioCanRx – Canada's Cancer Immunotherapy Network values patient partnerships and seeks to continuously learn alongside patients, patient organizations, researchers and research organizations.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Overcoming Venetoclax Resistance in Chronic Lymphocytic Leukemia Cells (CLL) ex vivo by Inhibiting DNA-Dependent Protein Kinase (DNA-PK) - Poster #188

Presenter: Clare Hibbert, University of Manitoba/CancerCare Manitoba

Other Authors: Ali Saleh, Sara Kost, Evgeniia Maksimova, Yongqiang Chen, Abdo-Jose Tomajian, Bozena Kuzio, Versha Banerji, Lin Yang, Spencer Gibson, James Johnston, Sachin Katyal

ABSTRACT_ID#2572

ABSTRACT

The canonical function of activated DNA-dependent protein kinase (pDNA-PK) is to induce the DNA damage response and mediate DNA repair. However, non-canonical roles include transcription, metabolism and mitochondrial processes. M3814, a DNA-PK inhibitor, is in clinical trials with radiation/chemotherapy and is not myelosuppressive. Cytotoxicity of M3814 was evaluated in 32 CLL patient samples via 7AAD/Annexin-V analysis and flow cytometry. There was 100-fold variation in cytotoxicity with the median inhibitory concentration for cell viability (IC₅₀) being 2.4 μ M, similar to plasma levels in clinical trials. Resistant cells were more likely to be IGHV unmutated and/or had p53 dysfunction. Preliminary metabolic studies using Seahorse showed M3814 decreased maximal respiration and mitochondrial membrane potential in CLL cells. Western analysis showed increased levels of mitochondrial anti-apoptotic proteins, including BCL2, in resistant samples. Venetoclax (VEN), a BCL-2 inhibitor, is a standard treatment in CLL and acute myeloid leukemia (AML); however, relapse and myelosuppression occur. Sensitivity of CLL cells to VEN varied 100-fold with an IC₅₀ of 1.7 nM, similar to plasma levels in patients. However, there was no cross-sensitivity between VEN and M3814. Synergy occurred between M3814 and VEN, particularly in VEN resistant cells and those treated with CD40L (to simulate the microenvironment, where resistance occurs). Similar results are found in AML cell lines. These unique findings demonstrate a non-canonical role of DNA-PK on the mitochondria in leukemia cells. Additionally, M3814 may enhance therapeutic efficacy of VEN and reverse resistance in CLL/AML and other cancers that have enhanced DNA-PK activity.

LAY SUMMARY

At clinically achievable doses, M3814 is cytotoxic to Chronic Lymphocytic Leukemia (CLL) cells and there is a 100-fold variation in the sensitivity to M3814, with resistant cells being IGHV unmutated, having p53 dysfunction and/or patients having had prior therapy. M3814 has been shown to have effects on the mitochondria: initial seahorse studies show M3814 inhibits mitochondrial maximal respiration and M3814 resistant cells have noteworthy increases in levels of BCL-2, MCL-1 and pBAX, all of which are anti-apoptotic and promote tumour cell survival. M3814 enhances Venetoclax (VEN) activity, shown through drug synergy, in CLL cells ex vivo likely due to VEN inhibition of BCL-2.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Nuclear N-myristoyltransferase 1: A Predictor of Favourable Treatment Response and Transcriptional Regulator in HR+ Breast Cancer - Poster #190

Presenter: Abinash Meher, University of Winnipeg/Paul Albrechtsen Research Institute, CancerCare Manitoba

Other Authors: Lambert Wilson Baiden, Danira Jaksic, David Datzkiw, Fatim Aguibou Diaby, Hailey Langford, Anuraag Shrivastav

ABSTRACT_ID#3184

ABSTRACT

Breast cancer (BC) is the most common cancer worldwide and is the most common cancer diagnosed in American women. Despite the relative success of endocrine therapies in treating hormone receptor-positive (HR+) BC, de novo and acquired resistance to these therapies (endocrine resistance) remain significant concerns. In a cohort of 448 HR+ BC patients who received adjuvant tamoxifen therapy after surgery, we found that the differential expression and localization of N-myristoyltransferase 1 (NMT1) in HR+ BC primary tumors predict treatment response. Low cytoplasmic and high nuclear NMT1 expression displayed a significant association with better clinical outcomes, whereas low cytoplasmic and low nuclear NMT1 strongly predicted a worse treatment response. The association of nuclear NMT1 (nNMT1) with a better prognosis led us to investigate the functional attributes of nNMT1 in BC cells. We identified a specific serine residue (S47) that, upon phosphorylation, renders NMT1 nuclear by a site-directed mutagenesis study in BC cells (MCF7). Constitutive phospho-mimic at S47 by mutating serine to glutamic acid (S47àE) translocated NMT1 in the nucleus and induced cell death in MCF7 cells. Furthermore, we identified a hitherto unknown function of NMT1 in gene regulation by chromatin immunoprecipitation (ChIP-RT-qPCR and ChIP-Seq) analyses. We identified insulin-like growth factor 1 receptor as a potential target of NMT1 by ChIP-RT-qPCR. ChIP-seq experiments using monoclonal and polyclonal anti-NMT1 antibodies revealed many genes and intergenic regions where NMT1 protein potentially binds and regulates the expression of target genes. These results may aid in developing more effective treatment regimens for managing breast cancer.

LAY SUMMARY

Breast cancer (BC) has become the most prevalent cancer worldwide, exceeding lung cancer incidence, and remains the most frequently diagnosed cancer in American women. Despite the efficacy of endocrine therapies in treating hormone receptor-positive (HR+) BC, acquired resistance to these treatments presents significant clinical challenges. In a cohort of 448 HR+ BC patients who received endocrine therapy, we observed that the expression patterns of N-myristoyltransferase 1 (NMT1) protein in breast cancer cells correlated with treatment response. We observed that low cytoplasmic and high nuclear NMT1 expression was significantly associated with better clinical outcomes. We investigated the potential role of NMT1 protein in the nucleus of breast cancer cells. We found that the NMT1 protein may play a role in regulating the expression of genes involved in cell proliferation.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Anti-Minor Histocompatibility Antigen Expanded T Cells to Treat Blood Cancers Relapsing After Allogeneic Stem Cell Transplantation - Poster #192

Presenter: Denis-Claude Roy, Hôpital Maisonneuve-Rosemont

Other Authors: Sylvie Lachance, Jean-Sebastien Delisle, Imran Ahmad, Felix Couture, Annabelle Minguy, Jean-Philippe Bastien, Natasha Kekre, Lea Bernard, Brian Leber, Jessica Trottier, Jaime Lionel Sanchez-Dardon, Cedric Carli, Sandra Cohen, Thomas Kiss, Jean Roy, Guy Sauvageau, Olivier Veilleux, Lambert Busque, Claude Perreault, Vibhuti Dave

ABSTRACT_ID#3065

ABSTRACT

Background & Aim: To increase graft-versus-leukemia effect (GVL) observed after allogeneic hematopoietic stem cell transplantation (HSCT) and prevent donor T cells from causing graft-vs-host disease (GVHD), we have isolated and expanded T cells targeting host minor histocompatibility antigens (MiHAs) preferentially expressed on hematologic cancer (HC) cells (Perreault, Leukemia 2016). We have used this strategy in a Phase I clinical trial and have developed a second-generation tumor immunisation process with increased anti-HC activity. **Methods & Results:** In a first in human open-label, multi-center Phase 1 clinical trial (NCT 03091933), patients with HC who relapsed after an allogeneic HLA-matched HSCT were treated with GLIDE: ex vivo- expanded sibling donor T cells selected and expanded for reactivity toward recipient MiHAs. Ten patients, median age of 58 (33-69), 4F/6M with AML(n=7), NHL(1), CLL(1), and myelofibrosis(1) relapsing after HSCT received GLIDE products consisting mainly of T-cells (CD8>CD4>NKT>NK cells) with an effector memory and central memory phenotype. TCR sequencing showed oligoclonal expansion and T-cell survival for up to a year post-transplant. Cells were well tolerated with only one patient developing thrombocytopenia and chronic GVHD. There were no other potentially treatment-related adverse events. Survival at one year was observed in 4 patients with 3 patients in sustained remission. Further, we have identified a hierarchy of immunogenicity within MiHAs, and developed an improved dendritic cell and T-cell immunization process that is being incorporated in a successor clinical trial. **Conclusion** Our Phase-I clinical trial and process development work have positioned us for a successor clinical study with greater anti-leukemia potential.

LAY SUMMARY

1. We have shown in a first-in-human Phase I clinical study the feasibility, safety and anti-leukemia potential of anti-minor histocompatibility antigen (MiHA) T cells (GLIDE) in patients with very high-risk blood cancers (patients relapsing after an allogeneic stem cell transplantation).
2. This study has led to the identification of a hierarchy of immunogenicity within Minor histocompatibility antigens (MiHA) that will allow improved selection as well as more precise and efficient targeting of hematologic cancer cells in our GLIDE-2 successor clinical trial.
3. In our second-generation immunotherapeutic approach (GLIDE-2), we have also improved cell processing. These anti-MiHA T cells now demonstrate improved biologic characteristics, including greater anti-hematologic cancer reactivity through multiple MiHA exposure, CD8 cell selection, and enhanced dendritic cell antigen-mediated activation. This novel strategy is now being evaluated in a clinical trial to prevent cancer relapse in patients with high-risk hematologic cancers.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Title: Investigating the Metabolic Underpinnings of Venetoclax Resistance in DLBCL - Poster #193

Presenter: Daisy Tran, University Health Network

Other Authors: Evelyn Teh, Susanne Penny, Devanand Pinto, Samuel Saibil, John Kuruvilla, Rob Laister

ABSTRACT_ID#3305

ABSTRACT

Large B cell lymphoma is a heterogeneous lymphoid malignancy where the expression of anti-apoptotic protein BCL-2 is often dysregulated by chromosomal translocation and/or overexpression. Patients with BCL-2 dysregulation have worse prognosis under standard chemoimmunotherapy treatment regimens and represent a clinical unmet need. BCL-2 inhibitors such as venetoclax provide an opportunity to target BCL-2, however, resistance to BCL-2 inhibition has been observed. The goal of our study is to delineate the metabolic basis of venetoclax resistance, to better understand BCL-2 biology and to aid in identifying potential routes for the development of combination therapies. We observed that venetoclax resistant (VR) cell lines have significantly altered bioenergetics as compared to venetoclax sensitive (VS) cells. The VR cells are more metabolically active at baseline and more flexible following stress. We also observed greater mitochondrial ATP production rates and, consequently, greater reactive oxygen species production. Using electron microscopy, we find that VR cells contain both fissured and fused mitochondria, whereas mitochondria in VS cells take on a more uniform morphology. In line with the observed mitochondrial phenotype, VR cells trend towards greater mitochondrial mass and mass spectrometry proteomics revealed upregulated pathways related to mitochondrial protein degradation, aerobic respiration, and mitochondrial translation. We interpret these results to suggest that the mitochondria in VR cells, while operating at elevated levels of oxidative phosphorylation and producing elevated levels of ROS, activate additional pathways for the surveillance and removal of damaged or unfolded proteins.

LAY SUMMARY

Venetoclax resistant (VR) cell lines have altered expression of BCL-2 family proteins, and enhanced mitochondrial metabolism and dynamics. To support increased mitochondrial activity, VR cells appear to upregulate several pathways related to mitochondrial protein quality control and homeostasis. Operating at elevated levels of oxidative phosphorylation requires VR cells to rely on the activation of pathways that survey, remove and replace damaged mitochondrial proteins.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Targeting N-Myristoyltransferases as a Therapeutic Strategy in Chronic Lymphocytic Leukemia - Poster #194

Presenter: Elizabeth McMurtry, University of Alberta

ABSTRACT_ID#2638

ABSTRACT

Chronic Lymphocytic Leukemia (CLL) is a malignancy marked by the accumulation of dysfunctional CD5+ CD19+ B cells, driven in part by continuous activation of pro-survival B cell receptor (BCR) signalling. Targeted therapies such as ibrutinib (BTK inhibitor) and venetoclax (Bcl-2 inhibitor) have improved outcomes, yet drug resistance remains a barrier to durable remission. Myristoylation, a lipid modification catalyzed by N-myristoyltransferases (NMTI/2), is required for membrane localization and function of key BCR signalling proteins including Lyn and HGAL. Zelenirstat, a pan NMTI/2 inhibitor, disrupts myristoylation-dependent pathways, such as BCR signalling. It is currently in clinical trial for B cell lymphoma. We hypothesized that zelenirstat induces apoptosis in CLL cells by blocking BCR-mediated survival signalling. Treatment of primary CLL cells and B cell malignancy-derived BJAB and MEC-1 lines with zelenirstat led to increased apoptosis, as determined by Annexin V/7AAD assay. This was accompanied by reduced phosphorylation levels of signalling proteins Lyn AKT, ERK, BTK, and Src-family kinases. Total phosphotyrosine was also reduced. Combination treatment of zelenirstat with venetoclax led to increased cell death, suggesting potential synergy. Expression of anti-apoptotic Bcl-2 family proteins (Bcl-2, Mcl-I) remained unchanged following treatment. Zelenirstat also induced mitochondrial dysfunction, including loss of membrane potential and increased reactive oxygen species (ROS) production in these cells. These findings support NMT inhibition as a novel approach to bypass therapeutic resistance in CLL by targeting BCR signalling upstream of resistance pathways and impairing mitochondrial function.

LAY SUMMARY

1. Zelenirstat, a new experimental drug, can block a cellular process called myristoylation, a type of protein modification that CLL cells rely on to send survival signals. By interfering with this process, zelenirstat cuts off critical communication that helps CLL cells stay alive.
2. Zelenirstat blocks B cell receptor (BCR) survival signaling, a pathway CLL cells use to resist treatment and avoid cell death. We saw reduced activation of proteins like AKT, ERK, and BTK, which are normally switched "on" by the BCR to promote cancer cell survival. This targeted inhibition helps drive CLL cells into cell death, even without changing levels of typical survival proteins like Bcl-2 and Mcl-I.
3. Zelenirstat also causes mitochondrial dysfunction-damaging the part of the cell that produces energy. We observed increased oxidative stress and the loss of mitochondrial membrane potential. This mitochondrial damage further weakens the cancer cells, making them easier to eliminate.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

ER+ signaling in breast cancer: KO by SUMOs? - Poster #197

Presenter: Anaïs Vivet, Immunology and Cancerology Research Institute

Other Authors: Antoine Bouchard, Laurent Cappadocia, Sylvie Mader

ABSTRACT_ID#2493

ABSTRACT

Estrogen receptor alpha (ERα) is a key oncogenic transcription factor in breast cancer, expressed in approximately 70% of tumors. Its hormone-driven activation promotes cell proliferation and survival, making ERα a major therapeutic target. Fulvestrant, a clinically relevant pure anti-estrogen, inhibits ERα by inducing its ubiquitination and multi-SUMOylation. While ubiquitination promotes receptor degradation, SUMOylation represses ERα transcriptional activity through destabilization of DNA binding and induction of chromatin closure. Our recent findings further suggest that the E3 SUMO ligases PIAS1 and PIAS2 contribute to ERα SUMOylation in the presence of fulvestrant2, and that the StUBLs RNF4 and RNF111 can induce ubiquitination of ERα in a SUMO-dependent manner. We now hypothesize that the recruitment of a SUMO E3 ligase would be sufficient to induce ERα multi-SUMOylation and downstream transcriptional inactivation. To test this hypothesis, fusion proteins were then generated by linking ERα to either PIAS1 or the minimal catalytic domain of ZNF451 (30 amino acids). SUMOylation of the fusion proteins irrespective of the presence of antiestrogens was confirmed by western analysis in HEK293 and MCF-7 cell lines, and their nuclear localization was validated by immunofluorescence. Luciferase reporter assays showed that both ERα-PIAS1 and ERα-ZNF451 fusion proteins were transcriptionally repressed and exerted dominant-negative effects on estrogen signalling. This inhibition was partially reversed by expression of the deSUMOylase SENP1 or by addition of SUMOylation inhibitors. Together, these results support the suggestion that targeted SUMOylation of transcription factors represents a potential therapeutic approach to suppress the activity of oncogenic transcription factors, including ERα.

LAY SUMMARY

This study shows that fusing ERα to SUMO E3 ligases (PIAS1 or ZNF451) induces its SUMOylation and suppresses its transcriptional activity, even without anti-estrogens. The resulting repression was partially reversible, suggesting SUMOylation directly contributes to ERα inactivation. These findings support targeted SUMOylation as a potential therapeutic strategy to inhibit oncogenic transcription factors like ERα.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

AI-guided Drug Development of DNA Topoisomerase II Inhibitors for Anticancer Treatment - Poster #198

Presenter: Graciella Rosellinny, University of British Columbia

Other Authors: Victor Matias-Barrios, Mariia Radaeva, Artem Cherkasov, Xuesen Dong

ABSTRACT_ID#2376

ABSTRACT

Topoisomerase II (TOP2) is an essential enzyme that regulates DNA topology during replication and cell division. In many aggressive cancers, TOP2 is overexpressed, contributing to rapid tumor growth and poor patient prognosis. While current TOP2 inhibitors such as etoposide are widely used in chemotherapy, they act as poisons by inducing DNA strand breaks. This genotoxic mechanism, though effective, is associated with severe dose-limiting toxicity and therapy-related secondary malignancies. Catalytic TOP2 inhibitors, which suppress TOP2 activity without causing DNA damage, offer a safer therapeutic alternative. However, no catalytic TOP2 inhibitor has been approved for cancer treatment, highlighting an urgent need for novel candidates. Using computer-aided drug design in combination with artificial intelligence, we developed a series of small molecule inhibitors. Validated by a series of quantitative biochemical and biological analyses, T724 was confirmed to be a lead compound. Fluorescence polarization and electrophoretic mobility shift assays demonstrated that T724 binds TOP2 with high affinity and disrupts TOP2–DNA interactions. Thermal shift assays further showed that T724 stabilizes TOP2 protein, confirming direct binding. Moreover, in vitro studies revealed that T724 strongly suppressed cancer cell proliferation with IC₅₀ at ~2μM, without inducing noticeable DNA damage index reflected by γH2A.X levels in great contrast to etoposide. These findings support the continued development of T724 as a promising novel candidate and underscore the potential of catalytic inhibitors as a safer, targeted anticancer therapy with reduced long-term risks for patients.

LAY SUMMARY

Many cancer treatments work by targeting a protein called Topoisomerase II (TOP2), which helps cells divide. However, most of these drugs act by breaking DNA strands to block cell division. While effective at killing cancer cells, this approach often causes serious side effects like heart damage and therapy-related secondary cancers. There is an urgent need for safer drugs with minimal side effects. We designed a new drug, called T724, that targets TOP2 by a new mechanism of action. Through multiple biochemical tests, this new drug was observed to bind strongly to TOP2, block its ability to interact with DNA, and help keep the protein in an inactive state. When tested in various cancer cell models, including aggressive prostate and leukemia models, T724 significantly suppressed cancer growth, but showed minimal cytotoxicity. These findings suggest that T724 could lead to safer treatment options for cancer patients, especially those facing long-term treatment or at higher risk of serious side effects, thus bringing us closer to next-generation therapies that improve quality of life without compromising effectiveness.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Inhibition of gamma-glutamyl transferase with novel drug design attenuates cisplatin-induced hearing loss in paediatric cancer patients - Poster #199

Presenter: Asna Latif, University of Alberta

Other Authors: Rafael Dias Do Espirito Santo, Jun Jie Fong, Frederick West, Amit Bhavsar

ABSTRACT_ID#3337

ABSTRACT

Cisplatin is a highly effective and commonly used chemotherapeutic that presents unique challenges in treatment brought upon by its associated toxicities. Most pressing of these toxicities is permanent hearing loss, or ototoxicity, that occurs in over 50% of paediatric cancer patients treated with cisplatin and leads to impaired socio-emotional development long-term, necessitating mitigations for its side effects. Currently, the mechanism of cisplatin-induced hearing loss is unclear. However, cisplatin is known to interact with glutathione, an anti-oxidative tri-peptide that is typically involved in removing toxins from the cell but can be hijacked by cisplatin to cause exacerbated toxicity in non-tumorigenic cells. This pathway is catalyzed by a few key enzymes that serve as promising targets for oto-protective therapy. One such enzyme, gamma-glutamyl transferase (GGT), presents as a particularly favourable drug target. Using informed drug design, we developed analogs of GGT inhibitors that were screened for innate toxicity and protection against cisplatin-induced toxicity in a murine inner ear cell line. Upon testing, several inhibitors showed marked protection against cell death in-vitro without interfering with cisplatin's anti-cancer efficacy. Cisplatin-glutathione metabolism presents as a rarely-studied pathway in cisplatin-induced ototoxicity that is shown in this work to contribute to toxicity and justifies the design of novel GGT inhibitors for oto-protective therapy. Through this project, we have identified the role of the glutathione pathway and GGT in cisplatin-induced ototoxicity and demonstrate the potential for development of small molecule inhibitors to improve long-term health outcomes of childhood cancer patients.

LAY SUMMARY

Cisplatin is an indispensable chemotherapeutic in paediatric cancer treatment that leads to permanent, bilateral hearing loss in >50% of treated patients. Glutathione is an anti-oxidant tripeptide that cisplatin hijacks to worsen toxicity in healthy cells, and we found that this pathway could be contributing to cisplatin-induced hearing loss by causing increased death of cells from the inner ear. We designed analogs of inhibitors that target an enzyme called gamma-glutamyl transferase (GGT) in the glutathione-cisplatin pathway that could be leading to hearing loss and tested them in an inner ear cell line derived from a mouse. Several of these inhibitors decreased cisplatin-induced cell death and inflammation in this cell line without interfering with its anti-cancer activity. This work identified the role of the glutathione anti-oxidation pathway in cisplatin-induced toxicity, as well as demonstrated the potential of inhibiting GGT as a therapeutic target.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Prolactin and Cholesterol Signaling Crosstalk: a therapeutic target in breast cancer associated bone metastasis - Poster #200

Presenter: Isabel Lopetegui Gonzalez, University of Calgary

Other Authors: Odul Karayazi-Atici, Juan Arteaga, Arvind Singla, Destiny Francis, Frank R Jirik, Sarah Manske, Carrie S Shemanko

ABSTRACT_ID#2674

ABSTRACT

Breast cancer is the most diagnosed cancer among women worldwide and is often associated with osteolytic bone metastasis, characterized by bone destruction. This research explores the connection between prolactin (PRL) signaling and cholesterol metabolism in breast cancer associated bone metastasis. We found that breast cancer cells engineered to secrete human PRL are more susceptible in vitro to the cholesterol-lowering drug, simvastatin (ST), than wild-type or empty vector controls, through a mechanism independent of hydroxy methyl glutaryl Coenzyme A reductase (HMGCR) levels. This sensitivity appears unrelated to breast cancer subtype, as it was observed in both estrogen receptor-positive MCF7 and Her2-positive SKBR3 cells. For in vivo analysis, MCF7 cells secreting PRL (MCF7_LGB_hPRL) and control cells (MCF7_LGB_EV) were injected into the tibias of immunodeficient mice pre-treated with ST for nine days. The treatment continued until day 42. ST significantly increased tissue mineral density (TMD) in mice bearing PRL-secreting tumors, but not in control mice, with little effect on the tumor size. This effect on TMD was also independent of the ability of ST to reduce cholesterol levels in mice. These findings support a functional interaction between PRL signaling and cholesterol metabolism, leading to heightened sensitivity of PRL-secreting tumors to ST, with potential effects not only on the tumor cells but also in their interaction with the bone microenvironment. This may provide a path for personalized therapy.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Exploring the Unique Mechanisms of Aclarubicin Cytotoxicity in Leukemia Cells Using CRISPR Screens - Poster #201

Presenter: Belal Osman, University of Montreal /IRIC

Other Authors: Khady Thiombane, Kayhan Zarnani, Thierry Bertomeu, Andrew Chatr-Aryamontri, Mike Tyers, Coery Nislow, Sylvie Mader

ABSTRACT_ID#2704

ABSTRACT

Introduction Topoisomerases are enzymes that help manage topological constraints on DNA during replication, transcription, and chromatin remodeling. Because of their central role in DNA processes, they are common targets in cancer treatment. Anthracyclines like Doxorubicin (Dox) and Daunorubicin (Dauno) poison topoisomerase II. Aclarubicin (Acla), used for leukemia treatment in some Asian countries, inhibits both topoisomerase I and II. Acla shows less cardiotoxicity and works even in some Dox-resistant cases, but its exact mechanisms of action remains unclear. Methods and Results Genome-wide CRISPR-Cas9 screens in NALM-6 pre-B acute lymphoblastic leukemia cells revealed clear differences between Acla and Dox, with minimal overlap in the genes affecting sensitivity to each drug. While knocking down topoisomerase II or DNA repair genes reduced or increased Dox toxicity, respectively, these changes did not affect sensitivity to Acla. Instead, loss of genes involved in the nucleolar stress response helped cells survive Acla treatment, while disruption of chromatin structure regulators like CTCF and the Cohesin complex made cells more vulnerable. This points to mechanisms leading to cell death due to altered chromatin architecture, gene transcription and proteotoxic stress rather than direct DNA damage. Conclusion Our findings reveal that Acla acts through unique pathways compared to other anthracyclines and support use of these drugs in a sequential manner. Understanding the mechanisms by which Acla works could lead to better treatment strategies and safer drug designs. Future work will explore these mechanisms in other leukemia models and patient-derived cells.

LAY SUMMARY

Aclarubicin (Acla) kills cancer cells differently than common chemotherapy drugs like Doxorubicin (Dox): While Dox damages DNA by targeting topoisomerase II, our research shows that Acla works through unique pathways involving changes in how DNA is organized and expressed, rather than causing direct DNA damage. Different genes matter for Acla vs. Dox sensitivity: Using a genome-wide CRISPR screen in leukemia cells, we found that the genes that make cells resistant or sensitive to Dox do not affect Acla in the same way. Instead, cells survive Acla treatment by disabling genes involved in nucleolar stress, and they become more sensitive when chromatin structure is disrupted. These results could help personalize cancer treatment: Because Acla is effective in some drug-resistant cases and causes less heart damage, understanding its distinct action may lead to safer, more effective therapies. Our findings support using different anthracyclines in sequence and call for deeper studies in other leukemia models and patient cells.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Extrahepatic Delivery of siRNA Targeting MLL/AF4 for Inhibiting t(4;11) Leukemia - Poster #202

Presenter: Remant KC, University of Alberta

Other Authors: Mohammad Nasrullah, Hasan Uludag

ABSTRACT_ID#2917

ABSTRACT

The potential for short-interfering RNAs (siRNAs) in gene-targeted therapies for human health conditions is significant, however, delivery challenges limit their clinical use. A major challenge in using siRNAs as a therapy is their difficulty in crossing cell membranes, primarily due to their negative charge and large size. This issue is even more pronounced in lymphocytes, which have a smaller size and limited cytoplasm. To address this, we developed a lipopolymer delivery system, PEI-C. This system utilizes lipid-conjugated polyethyleneimine (PEI) specifically designed to enhance the efficient delivery of siRNA into lymphocytes. We tailored low molecular weight PEI polymers by grafting lipids, yielding lipopolymers that self-assembled into nanocomplexes with siRNA. This approach leverages the positive charge of PEI to form stable complexes with negatively charged siRNAs, while the lipid components enhance cellular uptake of siRNA. We evaluated the performance of this delivery system in both in vitro and in vivo models of t(4;11) leukemia, concentrating on its ability to downregulate key oncogenes MLL/AF4. Our results showed significant cellular delivery of FAM-labelled siRNA in ALL cell lines RS4;11 and SEM. Using the lipopolymers complexed with MLL/AF4 siRNA, we observed enhanced gene silencing in leukemia cells, which resulted in increased apoptosis, reduced colony formation in vitro, and significantly decreased leukemia burden in vivo and enhanced survival rates. Additional experiments using PEI-C formulated siRNA nanoparticles showed enhanced extrahepatic biodistribution to leukemia-relevant organs such as spleen, bone marrow, and lungs. Our study presents the lipopolymer-based siRNA delivery system as an effective approach for treating t(4;11) leukemia.

LAY SUMMARY

A novel lipid-modified polymer, PEI-C, successfully overcomes delivery barriers by enabling efficient siRNA uptake into small, hard-to-transfect lymphocytes, such as leukemia cells. PEI-C nanoparticles carrying MLL/AF4-targeting siRNA induced strong gene silencing, leading to increased apoptosis, reduced cancer cell growth, and significantly lowered leukemia burden in preclinical models. The system showed effective delivery to leukemia-relevant organs (spleen, bone marrow, lungs), positioning PEI-C as a promising non-viral platform for siRNA-based leukemia therapy.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Epigenetic regulator MMSET in multiple myeloma chromatin states and drug resistance - Poster #203

Presenter: Dalia Barsyte Lovejoy, University of Toronto

Other Authors: Sehyun Cheon, Raquel Machado, Matthew Maitland

ABSTRACT_ID#3066

ABSTRACT

MMSET (multiple myeloma SET domain) methylates H3K36me2 and through the PWWP1 domain binding to H3K36me2 propagates transcriptionally activated state and counteracts transcriptional repressive chromatin marks. In 15-19% of Multiple Myeloma (MM) patients with t(4;14) translocation, IgH enhancer drives high levels of MMSET. Most t(4;14) MM patients exhibit a disruption or deletion of the PWWP1 domain, leading to shorter overall survival. To gain insight into how PWWP1-disruption influences MMSET function, we identified MMSET protein interactors using the BioID. PWWP1 deleted MMSET (Δ PWWP1 MMSET), and wild type full length MMSET were both tagged with a miniTurbo enzyme that biotinylates proximal protein interactors and provides the interaction network in the native cellular environment. Additionally, we performed chromatin profiling of H3K36me2 and the opposing repressive mark H3K27me3 using Cut&Run. The BioID results showed that MMSET interacts with transcriptional regulators and additionally Δ PWWP1 MMSET associates with the ribogenesis factors which is consistent with the nucleolar localization and our previous observation of the nucleolar retention of MMSET upon PWWP1 antagonist chemical probe UNC6934 treatment. Interestingly, the global levels of H3K36me2 were not affected by the deletion of PWWP1, but the distribution of H3K36me2 and H3K27me3, the transcriptome and cell adhesion behavior that accompanied the dexamethasone sensitivity were altered. Our study highlights the role of MMSET and its PWWP1 domain in regulating the epigenetic and transcriptional landscape in t(4;14) MM and dexamethasone resistance. Understanding how MMSET-mediated chromatin modifications contribute to the gene expression balance in t(4;14) MM will uncover the epigenetic mechanisms underlying MM disease outcomes.

LAY SUMMARY

MMSET (multiple myeloma SET domain) methylates H3K36me2 and through the PWWP1 domain binding to H3K36me2 propagates transcriptionally activated state and counteracts transcriptional repressive chromatin marks. In 15-19% of Multiple Myeloma (MM) patients with t(4;14) translocation results in high levels of MMSET and most t(4;14) MM patients exhibit a disruption or deletion of the PWWP1 domain, leading to shorter overall survival. To gain insight into how PWWP1-disruption influences MMSET function, we identified MMSET protein interactors using the BioID and showed that Δ PWWP1 MMSET associates with the ribogenesis factors which is consistent with the nucleolar localization and nucleolar retention of MMSET upon chemical probe UNC6934 treatment. Chromatin profiling of H3K36me2 and the opposing repressive mark H3K27me3 using Cut&Run indicated changes in the distribution of H3K36me2 and H3K27me3. The transcriptome and cell adhesion behavior that accompanied the dexamethasone sensitivity were distinct in Δ PWWP1 MMSET. Our study highlights the role of MMSET and its PWWP1 domain in regulating the epigenetic and transcriptional landscape in t(4;14) MM and dexamethasone resistance. Understanding how MMSET-mediated chromatin modifications contribute to the gene expression balance in t(4;14) MM will uncover the epigenetic mechanisms underlying MM disease outcomes.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Cationic Lipopolymers for siRNA Delivery in Lung Cancer Therapy - Poster #204

Presenter: Remant KC, University of Alberta

Other Authors: Mohammad Nasrullah, Hasan Uludag

ABSTRACT_ID#3183

ABSTRACT

Significant advancements have been made in the treatment of lung cancer, such as the non-small cell lung cancer (NSCLC), accounting about 85% of total lung cancers, and still the overall cure and survival rates for NSCLC remain low. Short interfering RNAs (siRNAs) have emerged as a promising approach. Effective biomaterials for packaging siRNA into nanoparticles will be key to clinical translation. In this study, we evaluated the potential of a series of cationic lipopolymers for delivering therapeutic siRNAs (Survivin, CDC20, and STAT5) in lung cancer models. Transfection reagents ALL-Fect, Leu-Fect, and Prime-Fect were obtained from RJH Biosciences (Edmonton, CANADA). The siRNA binding and dissociation efficiency of the polymers and physicochemical properties of the freshly prepared complexes were investigated first. Cellular uptake (flowcytometry, confocal), target gene silencing (qPCR), and cell killing efficacy (MTT) of the complexes were studied in A549 and Calu-3 cells. These polymers exhibited strong siRNA binding and dissociation properties, forming cationic nanoparticles with a diameter: 150 nm to 400 nm suitable for efficient cellular uptake, achieving over 75% FAM-positive cell populations. Remarkably, these complexes demonstrated significant cell killing effects even at a low siRNA dose (20 nM), with maximal effects observed at 40 nM siRNA concentration, resulting in over 75% cell killing. CDC20-targeting siRNA achieved over 50% tumor inhibition in xenografted A549 lung cancer models, with minimal toxicity. In conclusion, these findings underscore the potential of these cationic lipopolymers as safe and effective non-viral vectors for siRNA-based lung cancer therapeutics.

LAY SUMMARY

Cationic lipopolymers (ALL-Fect, Leu-Fect, Prime-Fect) safely and efficiently delivered siRNAs targeting key lung cancer genes (CDC20, SURVIVIN, STAT5), leading to high cellular uptake and potent gene silencing. These siRNA-loaded nanoparticles induced over 75% cancer cell death in vitro, and CDC20-targeting siRNA caused more than 50% tumor shrinkage in vivo with minimal side effects. The study highlights these polymers as promising non-viral carriers for siRNA therapeutics in lung cancer treatment.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Optimizing manufacturing for a solid tumour CAR-T clinical trial increases potency with shorter expansion time - Poster #205

Presenter: Danyel Evseev, University of Calgary

Other Authors: Hongrui Liu, Yeon Suh, Chimdindu Ohaegbu, Hayley Todesco, Kennedy Millar, Rebecca Malott, Nicole Prokopishyn, Franz Zemp, Kyle Potts, Douglas Mahoney

ABSTRACT_ID#3245

ABSTRACT

GCAR1, a first-in-human solid tumour CAR-T cell for rare sarcomas, renal cell carcinomas, and breast cancer, is being manufactured for a phase I clinical trial using Miltenyi CliniMACS Prodigy instruments. Growing evidence suggest that shorter ex vivo expansion times preserve less differentiated, more potent T-cell phenotypes, potentially enhancing in vivo persistence and antitumour activity. To explore whether reducing the post-transduction expansion time of GCAR1 CAR-T cells in a conventional Prodigy 12-day manufacturing process would enhance their potency, we sampled GCAR1 CAR-T cells at days 7, 9, and 12 during manufacturing and evaluated their antitumor efficacy in a murine xenograft model of alveolar soft part sarcoma. In parallel, in vitro experiments assessed functional resilience to repeat stimulation, cytokine production, and immunophenotypic markers of differentiation and exhaustion. Earlier harvests produced cells that were less differentiated and had greater antitumour activity in vivo at lower doses. These findings highlight a potential trade-off between maximizing cell yields and preserving in vivo functional fitness, with implications for improving manufacturing efficiency, product consistency, and patient outcomes. This work supports a data-driven refinement of the clinical manufacturing process ahead of GCAR1 Phase I trial initiation.

LAY SUMMARY

CAR-T cell therapy shorter ex vivo expansion time can lead to a more potent product. Biomanufacturing optimization contributes to improving patient outcomes.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

mRNA Delivery to Rejuvenate TRAIL Therapy for Lung Cancer Treatment - Poster #206

Presenter: Remant KC, University of Alberta

Other Authors: Mohammad Nasrullah, Daniel Nisakar Meenakshi Sundaram, Gurkirat Sandhu, Cezary Kucharski, Hasan Uludag

ABSTRACT_ID#3200

ABSTRACT

Tumor Necrosis Factor-Related Apoptosis-Inducing Ligand (TRAIL) is a promising cancer therapy known for its ability to selectively induce apoptosis in cancer cells. This study explores the therapeutic potential of a commercial lipopolymer, mRNA-Fect, and its formulation to express TRAIL from TRAIL-encoding mRNA (m-TRAIL). Transfection reagents mRNA-Fect and trans booster were obtained from RJH Biosciences (Edmonton, CANADA). This study evaluated two types of mRNA complexes: i) mRNA-Fect/mRNA, which involves a simple mixture of mRNA with mRNA-Fect, and ii) mRNA-Fect Kit/mRNA, which includes a pre-blending step with the mRNA-Fect Kit B component. Their efficiency in terms of cellular uptake, transgene expression, and cell killing efficacy was assessed across various cancer cell lines, including lung, pancreatic, colorectal, breast, and hepatic cancers. Additionally, the therapeutic efficacy of m-TRAIL loaded complexes was tested in a lung cancer xenograft model. Expression of the reporter gene GFP (m-GFP) in lung cancer cells was observed as early as 4 hours post-mRNA delivery, peaking at 24 hours. m-TRAIL delivery significantly inhibited cell growth by 40 to 60%, with up to 90% inhibition using the mRNA-Fect Kit. HCT116 and A549 were most responsive. We assessed m-TRAIL delivery in the A549 xenograft model through local (SC) or IV administration. No significant tumor growth differences were observed until the second treatment. However, after the fifth treatment, notable differences emerged, with lower tumor weights in the m-TRAIL group. In conclusion, mRNA-Fect could be a promising non-viral vector for mRNA-based lung cancer therapies, with potential applications in targeted cancer tumor growth inhibition.

LAY SUMMARY

This study demonstrates that mRNA-Fect polymers can deliver TRAIL-encoding mRNA selectively into lung cancer cells, leading to potent, targeted cell death without harming healthy cells. A modified mRNA-Fect Kit formulation showed improved release and stronger anti-tumor effects—up to 90% cancer cell killing in vitro and significant tumor shrinkage in mice—highlighting its therapeutic advantage. This non-viral nanoparticle system safely and effectively delivered therapeutic mRNA in multiple cancer models, offering a durable and scalable approach for future lung cancer treatments.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Lipopolymer Mediated siRNA Delivery for Targeting Leukemia-Associated Oncogenes in Preclinical Models - Poster #208

Presenter: Remant KC, University of Alberta

Other Authors: Mohammad Nasrullah, Aysha S. Ansari, Daniel Nisakar Meenakshi Sundaram, Cezary Kucharski, Hasan Uludag

ABSTRACT_ID#3201

ABSTRACT

The IRIS trial established imatinib as a first-line treatment for newly diagnosed chronic myeloid leukemia (CML), but around 24% of patients developed resistance within 18 months and 30–50% had therapy failure by 5 years. In acute myeloid leukemia (AML), the RATIFY trial reported a 40% non-remission rate for Midostaurin, while the ADMIRAL trial for gilteritinib showed a 30% relapse rate. These issues highlight the need for alternative therapies. Transfection reagents Prime-Fect and Leu-Fect were sourced from RJH Biosciences. Cellular uptake and transgene expression were measured by flow cytometry, and cytotoxicity was assessed using the MTT assay across various cancer cell lines. Finally, the therapeutic efficacy of the siRNA-loaded complexes was assessed in a leukemia xenograft model. The therapeutic efficacy of Prime-Fect/siFLT3 complexes in AML cells and Leu-Fect/siBCR-ABL complexes in CML cells was assessed in vitro, revealing a significant decline in cell proliferation and an increase in apoptosis by days 3 and 4 post-treatment. Colony-forming ability was also impaired, showing a ~42% reduction in colony numbers compared to controls. For in vivo assessment, MV4-11 and K562-xenograft models demonstrated tumor growth inhibition after the third treatment, leading to about a 50% reduction in tumor volume. In a follow-up study with drug-resistant K562 cells, treatment with Leu-Fect/siBCR-ABL initiated on day 4 post-inoculation significantly extended median survival to 63 days, compared to 56 days in the control group, and reduced leukemic burden in the bone marrow. These findings highlight the potential of these polymers as a promising non-viral vector for siRNA-based leukemia therapies.

LAY SUMMARY

siRNA-loaded lipopolymers (Prime-Fect and Leu-Fect) effectively silenced key leukemia genes (FLT3 in AML and BCR-ABL in CML), reducing cell growth and triggering apoptosis, even in drug-resistant cells. In leukemia xenograft models, these siRNA formulations significantly reduced tumor volume (~50%) and extended survival, with noticeable suppression of leukemic burden in the bone marrow. The study supports Prime-Fect and Leu-Fect as safe and effective non-viral siRNA carriers, offering a novel therapeutic strategy for patients with treatment-resistant AML and CML.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Structure-based discovery of mevalonate kinase inhibitors as potential anticancer agents - Poster #209

Presenter: Jaeok Park, Memorial University of Newfoundland

Other Authors: Arefeh Sadeghi, Saman Salari, Youla Tsantrizos, Sherri Christian

ABSTRACT_ID#2891

ABSTRACT

The mevalonate pathway is a biochemical network that produces isoprenoid building blocks such as farnesyl pyrophosphate (FPP) and geranylgeranyl pyrophosphate (GGPP). These intermediates are essential for the synthesis of downstream metabolites, including cholesterol and ubiquinone, and for the prenylation of small GTPases like Ras and Rho—a post-translational modification critical for their membrane localization and function. Owing to its roles in metabolic regulation and oncogenic signalling, the mevalonate pathway has emerged as a promising target for cancer therapy. In this study, we tested the hypothesis that synthetic compounds mimicking FPP could inhibit mevalonate kinase (MK), a key regulatory enzyme subject to feedback inhibition by FPP. Screening a panel of FPP analogs, we identified several MK inhibitors with nanomolar potency, including a lead compound with an IC₅₀ of 8 nM. In breast cancer cell models, this inhibitor exhibited markedly greater cytotoxicity than risidronate, a clinical drug that targets the mevalonate pathway. For example, in MDA-MB-231 cells, its EC₅₀ was ~20-fold lower than that of risidronate. These findings highlight MK as a potential anticancer target and introduce a novel class of small-molecule probes to investigate its therapeutic relevance. Ongoing work aims to elucidate the cellular mechanism of action of these inhibitors. In parallel, crystallographic studies are underway to characterize their binding interactions with MK and guide lead optimization toward preclinical development.

LAY SUMMARY

The mevalonate pathway is an essential metabolic system in cells that helps produce cholesterol and other molecules needed for cell growth and function. It also modifies certain proteins involved in cancer growth, making this pathway a promising target for cancer treatment. We discovered a new class of small molecules that can block an enzyme called mevalonate kinase (MK)—a key regulator in this pathway. These compounds were designed to mimic a natural pathway intermediate and showed strong ability to shut down MK activity in lab tests. One of our lead compounds was much more effective than risidronate, a current drug that acts on the same pathway. In breast cancer cells, it was about 20 times more potent at killing cancer cells. MK inhibitors may offer a new strategy for targeting cancers that rely on the mevalonate pathway.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Phospholipid-Coated Sodium Chloride Nanocrystals Disrupt Ionic Homeostasis to Induce Immunogenic Cell Death and T Cell Activation in Breast Cancer - Poster #210

Presenter: Navid Ahmadasab, University of Alberta

Other Authors: Zahra Elahi, Afsaneh Lavasanifar, Shokrollah Elahi

ABSTRACT_ID#2940

ABSTRACT

Despite advances in treatment, breast cancer remains a major global health challenge, largely due to immune evasion and resistance to conventional therapies. Sodium chloride (NaCl), a ubiquitous extracellular ion, has recently emerged as a noncanonical inducer of immunogenic cell death (ICD) by disrupting ionic homeostasis and activating stress-associated signaling pathways. To optimize and expand this therapeutic potential, we engineered two nanotechnology-based delivery systems: (1) concentrated NaCl solution loaded liposomes (NaCl-Lip) and (2) phospholipid-coated NaCl nanocrystals (NaClNCs), both designed to improve intracellular delivery and enable controlled ion release. Each formulation was characterized for its physicochemical properties such as average diameter and polydispersity, morphology, and ion release kinetics. Then the formulations were systematically compared to free NaCl solution (80 mM) in terms of cytotoxicity and mechanisms of cell death in EO771 and 4T1 murine breast cancer cell lines and human peripheral blood mononuclear cells (PBMCs) using MTS and Annexin V/Live-Dead staining, respectively. To assess immune activation, PBMCs were exposed to either nanoparticle or free NaCl. The expression of CD25 and CD69 was then measured in CD4⁺ and CD8⁺ T cells exposed to PBMCs. Compared to both free NaCl and NaCl-Lip, NaCl-NCs significantly reduced tumor cell viability (IC₅₀ 3.08 mM) and induced higher levels of apoptosis and necrosis and robustly enhanced CD4⁺ T cell activation. These results highlight NaClNCs as a low-cost, biocompatible, and dual-functional nanoplatform capable of inducing cancer cell death and stimulating adaptive anti-tumor immunity in breast cancer.

LAY SUMMARY

1. Sodium chloride-based nanoparticles may help fight breast cancer: We explored how sodium chloride can be used in innovative ways to kill breast cancer cells and enhance the immune system's response to tumors.
2. Engineered sodium chloride nanocrystals outperform free NaCl: We developed two nanotechnology-based delivery systems one using liposomes and another with phospholipid-coated sodium chloride nanocrystals. These systems made it easier for NaCl to enter cancer cells and allowed the ions to be released gradually.
3. Improved immune activation and cancer cell death: In laboratory tests using both mouse and human cells, the sodium chloride nanocrystals were more effective at killing breast cancer cells than either free NaCl or NaCl-loaded liposomes. They also activated CD4⁺ T cells key players in the immune system suggesting a strong stimulatory effect on immune function.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Sensitization of lung cancer cells to cisplatin by targeting ERCC1-XPF heterodimerization - Poster #211

Presenter: Masoumeh Keshavarz, Department of Oncology, Cross Cancer Institute, University of Alberta

Other Authors: Ahmed Abdelfattah, Mesfin Fanta, David Jay, Afsaneh Lavasanifar, Michael Weinfeld

ABSTRACT_ID#3110

ABSTRACT

Heterodimerization of ERCC1-XPF protein complex, as a DNA repair endonuclease, plays a critical role in nucleotide excision repair, and inter-strand crosslink and double-strand break repair. As such, its catalytic activity can counteract the cytotoxic effects of DNA lesions induced by platinum-based chemotherapeutic agents. Thus, targeting ERCC1-XPF heterodimers using small-molecule inhibitors may represent a beneficial therapeutic approach. In addition, drug repurposing remains an attractive strategy owing to its reduced risk profile and the potential for significant savings in both time and costs. In this study, we employed an antimalarial drug, pyronaridine (PYD) — previously identified as an inhibitor of the ERCC1-XPF complex's activity — and its liposomal formulation (LPYD) as a sensitizing agent to enhance the responsiveness of non-small cell lung cancer cells to cisplatin. LPYD was prepared using an ethanol injection method, and the average particle size using dynamic light scattering analysis and encapsulation efficiency were obtained (74.9 ± 1.22 nm and 98 ± 3.5 %, respectively). PYD's ability to disrupt ERCC1-XPF interaction was confirmed using the proximity ligation assay. The results of MTT and clonogenic assays demonstrated a significant decrease in the proliferation of both A549 and H1299 cells in response to the combination treatment with PYD or LPYD compared to cisplatin alone. Taken together, our findings confirm that the inhibiting ERCC1-XPF heterodimerization using PYD represents a promising strategy to enhance the efficacy of platinum-based chemotherapeutic agents.

LAY SUMMARY

1. Inhibition of ERCC1-XPF heterodimerization, a key endonuclease complex in the DNA repair pathway, enhances the efficacy of cisplatin in lung cancer cells.
2. The antimalarial drug, pyronaridine, demonstrates promising potential as an inhibitor of the ERCC1-XPF complex interaction.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Bioengineered probiotic E. coli to secrete RNA-loaded outer membrane vesicles for cancer treatment - Poster #213

Presenter: Kana Oshima, University of Alberta

Other Authors: David Stuart

ABSTRACT_ID#2644

ABSTRACT

Background: RNA-based therapeutics, including siRNA and shRNA, offer high target specificity and reduced side effects as they silence target genes in a sequence-specific manner. Despite their promise for cancer treatment, efficient delivery remains a challenge due to their instability and poor cell permeability. Current RNA delivery systems often require complex, environmentally sensitive preparation procedures and are costly. Therefore, it is essential to develop a simpler and more efficient RNA delivery system. In this study, we propose an RNA delivery system using the bioengineered probiotic E. coli that continuously secretes RNA-loaded outer membrane vesicles (OMVs) for cancer therapy. Methods: Synthetic DNA fragments encoding siRNAs were designed and incorporated into expression vectors, and transformed into the siRNA-feeding E. coli strain. A deletion of *nlpI* gene—known to increase OMV production—was introduced into the E. coli strain by P1 phage transduction. OMVs from the E. coli culture were isolated by ultracentrifugation, and siRNA expression and OMV packaging were investigated via RT-PCR on RNAs extracted from OMVs. To assess cancer-therapeutic potential, we plan to test the E. coli system producing OMVs carrying siRNA or shRNA targeting a mutant KRAS gene on mouse cells. Results: Protein quantifications revealed that *nlpI* deletion significantly increased OMV production, yielding over three times more OMVs than the wild-type strain. RT-PCR confirmed siRNA expression and successful packaging into OMVs, with higher secretion levels of smaller siRNA fragments. Conclusion: Our proposed probiotic E. coli system allows siRNA expression and packaging into OMVs, presenting a promising, low-cost delivery platform for RNA-based cancer therapy.

LAY SUMMARY

Developing a new way to deliver RNA-based cancer-drugs that can turn off specific genes involved in cancer development and progression. Engineering a “good bacteria”, which naturally lives in your intestines and helps keep your digestive system healthy, to secrete small membrane-bound sacs (called OMVs) carrying RNA drugs. These membrane-bound sacs can carry the RNA drug safely and deliver it to cancer cells. Our results show that the engineered bacteria makes more of these membrane-bound sacs and successfully package the RNA-drug inside, suggesting this bacterial system could become a low-cost and efficient way to deliver RNA-based cancer treatments in the future.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Testing NMDA Receptor Inhibitors in 3D Cell Models to Enhance Glioblastoma Treatment - Poster #214

Presenter: Nicolas Paradis-Isler, Centre de recherche de l'Hôpital Maisonneuve-Rosemont

Other Authors: Yoshiaki Tanaka

ABSTRACT_ID#2775

ABSTRACT

Glioblastoma (GBM), the most aggressive primary brain cancer, has limited treatment options and high recurrence rates. Glutamate, the brain's principal excitatory neurotransmitter, also promotes GBM cell survival, growth, and invasion. N-methyl-D-aspartate (NMDA) receptors—glutamate-gated ion channels expressed by both healthy neural and GBM cells—have emerged as promising targets for adjuvant therapy. However, whether NMDA receptor inhibitors can effectively combat GBM or which ones are most effective remains unclear. We investigated the potential of NMDA receptor blockade to inhibit GBM progression using 3D in vitro models that recapitulate key features of the brain tumour microenvironment. A panel of NMDA receptor antagonists and channel blockers was tested on glioblastoma spheroids (tumour-like 3D cell clusters) derived from three cell lines (LN-229, T98G, U-87 MG). All cell lines exhibited sensitivity to at least one form of NMDA receptor inhibition. To further assess effects in a neural context, we generated cortical organoids from human stem cells and introduced fluorescently labelled GBM cells. NMDA receptor inhibition reduced GBM cell invasion and growth within these organoids, with effectiveness varying by cell line and compound. Our findings demonstrate that NMDA receptor inhibition slows GBM cell growth and decreases invasiveness in both tumour spheroids and neural organoids. Notably, most inhibitors tested are already approved for other conditions, highlighting their potential for rapid repurposing as GBM adjuvant therapies. This work reinforces the therapeutic promise of NMDA receptor targeting and provides a translatable preclinical platform for evaluating brain tumour treatments.

LAY SUMMARY

Background: Glioblastoma is the deadliest brain cancer with very few treatment options and almost inevitable recurrence. Our research tested whether existing drugs that block a specific type of receptors found on brain cells could help fight this cancer.

Key Insights from Our Research:

- **Blocking a Brain Receptor May Slow Glioblastoma Growth:** We found that drugs blocking NMDA receptors, normally involved in brain cell communication, can slow the growth and spread of glioblastoma cells.
- **Promising Results in Advanced Lab Models:** Using 3D cell models that recreate features of tumours and brain tissue, we showed that, while the degree of response varied between drugs and across different tumour cell types, drugs that inhibit NMDA receptors can reduce glioblastoma cell growth and invasiveness.
- **Potential for Faster Treatment Options:** Many of the NMDA receptor blockers we tested are already approved for other diseases, mostly neurological disorders. This means they could potentially be repurposed quickly as new adjuvant treatment for glioblastoma.

In Summary: Targeting NMDA receptors may offer a new way to help treat glioblastoma, and our research provides a practical platform for preclinically testing such potential therapies.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Tumoroid Model Reveals Synergistic Impairment of Metabolism by Iron Chelators and Temozolomide in Chemo-Resistant Patient-derived Glioblastoma Cells - Poster #215

Presenter: Mohsen Akbari, University of Victoria

Other Authors: Meitham Amereh, Patrick Walter

ABSTRACT_ID#2848

ABSTRACT

Chemoresistance poses a significant clinical challenge in managing glioblastoma (GBM), limiting the long-term success of traditional treatments. Here, a 3D tumoroid model is used to investigate the metabolic sensitivity of temozolomide (TMZ)-resistant GBM cells to iron chelation by deferoxamine (DFO) and deferiprone (DFP). This work shows that TMZ-resistant GBM cells acquire stem-like characteristics, higher intracellular iron levels, higher expression of aconitase, and elevated reliance on oxidative phosphorylation and proteins associated with iron metabolism. Using a microphysiological model of GBM-on-a-chip consisting of extracellular matrix (ECM)-incorporated tumoroids, this work demonstrates that the combination of iron chelators with TMZ induces a synergistic effect on an in vitro tumoroid model of newly diagnosed and recurrent chemo-resistant patient-derived GBM and reduced their size and invasion. Investigating downstream metabolic variations reveal reduced intracellular iron, increased reactive oxygen species (ROS), upregulated hypoxia-inducible factor-1 α , reduced viability, increased autophagy, upregulated ribonucleotide reductase (RRM2), arrested proliferation, and induced cell death in normoxic TMZ-resistant cells. Hypoxic cells, while showing similar results, display reduced responses to iron deficiency, less blebbing, and an induced autophagic flux, suggesting an adaptive mechanism associated with hypoxia. These findings show that co-treatment with iron chelators and TMZ induces a synergistic effect, making this combination a promising GBM therapy.

LAY SUMMARY

TMZ-Resistant GBM Cells Depend on Iron Metabolism: Temozolomide (TMZ)-resistant GBM cells exhibit stem-like traits, elevated iron levels, and increased reliance on iron metabolism and oxidative phosphorylation. Higher aconitase expression makes these cells susceptible to iron chelators like deferoxamine (DFO) and deferiprone (DFP), suggesting a new target to overcome chemoresistance. **Iron Chelators Boost TMZ Effectiveness:** In a 3D GBM-on-a-chip tumoroid model, combining iron chelators with TMZ significantly reduced tumor size and invasion in both newly diagnosed and recurrent, TMZ-resistant patient-derived GBM cells. This synergistic effect highlights iron chelators as a promising addition to TMZ therapy. **Metabolic Shifts Drive Cell Death:** The combination therapy lowered iron levels, increased reactive oxygen species, and induced autophagy and cell death in resistant GBM cells. Hypoxic cells showed reduced responses, indicating that tumor oxygen levels may influence treatment efficacy, emphasizing the role of the tumor microenvironment.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Time-dependent chromatin maturation during 3D spheroid culture improves preclinical modeling of non-small cell lung cancer - Poster #216

Presenter: Noël Raynal, Université de Montréal/CHU Sainte-Justine

ABSTRACT_ID#2340

ABSTRACT

Non-small cell lung cancer (NSCLC) is the deadliest cancer worldwide. Therapeutic progress stagnate, highlighting the complexity to replicate NSCLC in preclinical models. Drug discovery studies rely mostly on cancer cells in two-dimension (2D), which poorly predict drug efficacy in patients. There is a growing interest in three-dimensional (3D) preclinical models, such as 3D spheroids, to better model tumor phenotype and improve therapeutic prediction. However, a comprehensive view of 3D culture methods impact on transcriptomes, epigenomes and pharmacological responses and their correlations to NSCLC tumors is still missing. Here, we demonstrate that NSCLC spheroids undergo time-dependent transcriptomic and epigenomic changes, which peak after 3 weeks of culture. While DNA methylome remained stable, chromatin methylation and acetylation marks gained features of advanced NSCLC in a time-dependent manner. Single-cell transcriptomic profiling of spheroids demonstrated that time of 3D culture improved the correlation to NSCLC tumors. Moreover, long-term culture of 3D spheroids increased drug screening predictability, by showing resistance to drugs that failed in NSCLC patients (such as HDAC inhibitors) while demonstrating novel pharmacological vulnerabilities and synergistic interactions (such as combination of PRMT and HDAC inhibitors). Strikingly, reverting 3D spheroids back to 2D culture rapidly reversed transcriptomic, epigenetic and pharmacological signatures acquired after 3 weeks of 3D culture, highlighting the critical impact of cell culture conditions on NSCLC phenotype. Collectively, our findings demonstrate that implementing a time-dependent maturation process into 3D spheroid culture induces chromatin and transcriptomic changes that enhance NSCLC preclinical modeling.

LAY SUMMARY

Collectively, our findings demonstrate that implementing a time-dependent maturation process into 3D spheroid culture induces chromatin and transcriptomic changes that enhances preclinical drug evaluation. Thus, using well-characterized 3D spheroids that undergo a time-dependent transcriptomic and epigenomic maturation process may represent robust models that faithfully recapitulate the human tumors, allowing target identification and improved prediction of therapeutic efficacy in patients.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Identifying Novel Therapeutic Strategies for Neuroendocrine-Transformed Lung Cancer following TKI Resistance - Poster #217

Presenter: Matthew Djan, University of Toronto

ABSTRACT_ID#2807

ABSTRACT

Background: Lung cancer remains the leading cause of cancer-related mortality worldwide, with non-small cell lung cancer (NSCLC) comprising 85% of cases and small cell lung cancer (SCLC) 15%. Tyrosine kinase inhibitors (TKIs) are frontline treatment for NSCLC patients with oncogenic driver mutations (e.g., EGFR, ALK, KRAS), yet resistance inevitably develops. In 5–10 % of TKI-resistant cases, adenocarcinoma-type NSCLC undergo lineage switching to a more aggressive neuroendocrine (NE) including SCLC phenotype, a process putatively driven by transcriptional reprogramming that alters key survival, proliferation, and differentiation pathways. However, its molecular underpinnings remain poorly understood, and no effective therapies exist for NE-transformed patients. Methods: We established 4 long-term NE-transformed patient-derived organoids (PDOs) with detailed clinical-pathological and molecular annotations. These models were screened with a 68-compound epigenetic probe library (Structural Genomics Consortium), targeting diverse chromatin regulators (i.e. bromodomains, methyltransferases, demethylases, acetyltransferases) at 1,000 nM in 1536-well plates. Cell viability was quantified on day 6 using Alamar Blue and normalized to DMSO control. Top hits were further evaluated using 21-point dose-response assays to determine IC₅₀. Results: KDM4 inhibition emerged as the top hit, reducing cell viability by $\geq 50\%$ across all four PDOs. Dose-response assays determined IC₅₀ values of ~ 200 nM, over five-fold more potent than EGFR-TKI (Osimertinib) or Chemotherapy (Etoposide), which exceeded 1,000 nM in the same models. Conclusion: PDOs provide a platform to identify potential therapeutic vulnerabilities in NE-transformed, TKI-resistant NSCLC. KDM4 inhibition demonstrated potent activity, supporting further mechanistic studies and in vivo validation of KDM4 as potential therapeutic target for this refractory population.

LAY SUMMARY

Lung cancer can change its identity and become more aggressive (commonly termed as “neuroendocrine transformation”) Lung cancer is broadly divided into non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC), each with distinct treatment strategies. Many NSCLC patients benefit from targeted therapies called tyrosine kinase inhibitors (TKIs), designed for tumours with specific oncogenic mutations such as EGFR, ALK, etc. However, around 5–10% of NSCLC tumors stop responding to TKIs and transform into a more aggressive neuroendocrine SCLC. These transformed tumors become resistant to TKIs, limiting patient treatment option to primarily chemotherapy, which has modest effectiveness in SCLC. We developed patient-derived models to study this rare lung neuroendocrine transformation Using patient tumour biopsies, we created long-term organoid models to recapitulate neuroendocrine-transformed SCLC tumours in the lab. These models enable us to study the biology of transformation and search for novel therapeutic approaches. Our drug screen revealed promising new therapeutic lead We tested 68 epigenetic compounds and identified a compound hit that consistently reduced organoid growth across these organoids. This finding offers a potential path towards more effective therapies for neuroendocrine-transformed patients which are refractory to TKIs.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Examining drug delivery in tumours using ultrasound stimulated docetaxel-loaded lipid-stabilized nanobubbles - Poster #218

Presenter: Osama Khan, University of Toronto

Other Authors: Patrick Chang, Sharshi Bulner, Kevin Kiezun, Alex Wright, Sebastien Zou, Yun Xiang, David Goertz, Naomi Matsuura

ABSTRACT_ID#3317

ABSTRACT

Introduction. Ultrasound stimulation of docetaxel-loaded phospholipid-stabilized nanobubbles (DTX-NBs) may offer an alternative to chemotherapy for the treatment of tumours.^{1, 2} However, the sonication parameters for maximizing drug quantity delivered to the tumour has not been optimized. The study elucidates varying focused ultrasound (FUS) pressure levels on DTX-NB-mediated tumour drug accumulation in vivo. **Methods.** Lipid-stabilized perfluorobutane-filled DTX-NBs (dmode=265±16nm; 99.7±2.0%<1µm; ~2×10¹⁰#/mL) were produced in-house³ and characterized using a Coulter Counter. Treatment using DTX-NBs were administered by tail-vein bolus injections on subcutaneous EMT-6 tumour-bearing 6-to-7-week-old female BALB/c mice in comparison to Taxotere® (clinical DTX formulation). DTX-NBs (5mg/kg) were also exposed to FUS pressures from 0.25, 0.5, 0.75, and 1.5MPa compared to co-delivered NBs+Taxotere®. Organs and plasma were harvested at 1hr post-administration. DTX was quantified by liquid chromatography/mass spectrometry. **Results.** DTX-NBs subject to 0.5MPa FUS demonstrated greatest significant (p<0.0001) drug uptake in tumours compared to all groups: ~3.5x to DTX-NBs only, ~2.7x to Taxotere® only, ~1.7x to DTX-NBs at 0.25MPa, ~1.9x at 0.75MPa, ~2x at 1.5MPa, ~1.8x than NBs+Taxotere® at 0.5MPa, and ~2x to NBs+Taxotere® at 1.5MPa. No significance was observed across all other organs for experimental groups. **Conclusion.** Exposing DTX-NBs to 0.5MPa of FUS provided the greatest tumour drug uptake, demonstrating an optimum targeted delivery pressure. The in vivo therapeutic effect of DTX-NBs following this sonication scheme will be evaluated longitudinally preclinically in comparison to the appropriate controls. **References.** ¹Helfield et al., *Frontiers in Physics*, 2021; ²Batchelor et al., *Curr. Opin. Col. & Inter. Sci.*, 2021; ³Matsuura et al., *IEEE Int. Ultra. Sym. Prod.*, 2015

LAY SUMMARY

Chemotherapy is a clinical treatment where patients undergo anticancer drug infusions to treat solid tumours. Systemically administered drugs target cancerous and healthy tissues non-specifically leading to undesired side effects. One promising approach to improve targeted drug delivery to tumours is using non-invasive ultrasound to remotely cavitate ('pop') gas-filled drug-loaded nanobubbles. This method can potentially improve drug uptake to the tumour and mitigate drug uptake to healthy tissues. However, the specific ultrasound pressure to cavitate these drug-loaded nanobubbles has not been optimized as the elicited pressure can vary the degree of tumour blood vessel permeabilization and drug release from the bubbles. Therefore, this study investigates the correlation between the measured drug quantity in preclinical tumour-bearing animal models following exposure of drug-loaded nanobubbles to various ultrasound pressures (0.25, 0.5, 0.75, 1.5MPa) in comparison to the appropriate controls. Our results demonstrate that exposing the drug-loaded nanobubbles to an ultrasound pressure of 0.5MPa provides the greatest tumour drug uptake compared to drug-loaded nanobubbles (~3.5x) and the clinical drug formulation (Taxotere®) (~2.7x) with no ultrasound exposure, and those exposed to 0.25MPa (~1.7x), 0.75MPa (~1.9x), and 1.5MPa (~2x), as well as unloaded nanobubbles co-delivered with Taxotere® at 0.5MPa (~1.8x) and 1.5MPa (~2x).

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Antiproliferative Activity of the Aminosteroid RM-581 on Leukemia Cell Lines - Poster #219

Presenter: Maude Fleury, CHU de Québec-Université Laval Research Center

Other Authors: Jenny Roy, René Maltais, Donald Poirier

ABSTRACT_ID#3057

ABSTRACT

Leukemia is a major cancer affecting a significant portion of the population, particularly children. To date, few steroid compounds have been studied for their potential activity against leukemia. The aminosteroid RM-581, an anticancer agent synthesized by our research group, exhibits strong antiproliferative activity against cell lines from more than ten solid tumor cancers, including some with poor prognosis. However, RM-581's impact has never been assessed on leukemia. The biological activity of RM-581 was evaluated on four leukemia cell lines (HL-60, JURKAT, K562, and THP-1), showing antiproliferative effects with half maximal inhibitory concentration (IC50) values of 1.71 μ M, 1.18 μ M, 4.47 μ M and 5.61 μ M, respectively. The proportion of late apoptotic cells increased significantly from 3.1% to 69.1% according to RM-581 concentrations, while the necrotic cells dropped from 3.4% to 0.01%. Although no selectivity was observed toward healthy peripheral blood lymphocytes (PBLs) in our preliminary tests, flow cytometry revealed a significant impact on T lymphocytes (CD3+), particularly cytotoxic T cells (CD8+). Notably, at 50 μ M, RM-581 significantly reduced CD8+ T cells, increasing the CD4/CD8 ratio from 2.05 to 4.35. This contrasts with many chemotherapy agents, which broadly suppress immune cells but often cause a more prolonged reduction in CD4+ helper T cells, contributing to immune deficiency. These findings indicate that RM-581 exhibits antiproliferative activity against both solid tumors and leukemia. Further analyses are underway to assess its effects on the cell cycle.

LAY SUMMARY

RM-581, an aminosteroid anticancer agent, showed strong antiproliferative effects against four leukemia cell lines, with IC50 values between 1.18 μ M and 5.61 μ M. RM-581 induced late apoptosis in leukemia cells without increasing necrosis, suggesting a controlled cell death mechanism. RM-581 notably reduced CD8+ cytotoxic T cells and increased the CD4/CD8 ratio, an immunomodulatory effect that differs from typical chemotherapy and warrants further investigation.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Nanophytoglycogen-mediated delivery of Poly I:C enhances cellular uptake, immune activation and cytotoxicity in ovarian cancer cells - Poster #220

Presenter: Abigail Peralta, Wilfrid Laurier University

ABSTRACT_ID#2742

ABSTRACT

Ovarian cancer is the most lethal gynecological malignancy and the fifth leading cause of cancer-related deaths in women. Prognosis is poor as 80% of patients are diagnosed at advanced stages, where the five-year survival rate drops to 31%. Current standard treatments rely on invasive surgery and chemotherapy, yet more than 70% of patients experience relapse with chemoresistant disease. This highlights an urgent need for safer, more effective therapeutic options. Immunostimulatory nucleic acids, such as long double-stranded RNA (dsRNA), have emerged as promising treatments by activating innate immune pathways within cancer cells and cells of the tumour microenvironment, inducing an anti-tumor response. However, their clinical use is limited by poor stability and low uptake. We investigated nanophytoglycogen (NPG), a biodegradable, non-toxic, glycogen-based nanoparticle derived from sweetcorn, as a novel delivery system for polyinosinic:polycytidylic acid (poly I:C) in high-grade serous ovarian cancer (HGSOC), the most common and lethal subtype. Treatment of HGSOC cell lines (OV866(2), TOV1946, TOV2223G) with NPG–poly I:C significantly upregulated interferon-stimulated genes and pro-inflammatory cytokines compared to poly I:C alone, with greater induction of interferon-related proteins than inflammatory cytokines. Viability assays revealed cell line-specific cytotoxicity, with up to a 50% reduction in viability after 48 hours. Fluorescent microscopy confirmed enhanced intracellular delivery with NPG. These findings support NPG–poly I:C as a promising immunotherapeutic strategy that improves immune activation, cellular uptake, and cytotoxicity in ovarian cancer. Future work will use receptor knockout lines to identify key immune pathways and expand this platform for broader cancer and immunologic applications.

LAY SUMMARY

Ovarian cancer is the fifth leading cause of cancer-related death in women, and current treatments often fail—about 70% of patients experience a relapse. My research investigates how we can turn our body's natural immune response against viral infection into a response that fights ovarian cancer. We're studying a molecule called poly I:C, which mimics viral infection and can activate the immune system to attack cancer cells. However, on its own, poly I:C is unstable and doesn't enter cells efficiently. To solve this, I use a plant-based nanoparticle called nanophytoglycogen (NPG). It's a non-toxic, sugar-based particle derived from sweetcorn that helps protect poly I:C and improve its delivery into cancer cells. When we treated ovarian cancer cells with the NPG–poly I:C combination, we saw increased uptake, a stronger immune response, and greater cancer cell death compared to using poly I:C alone. These results suggest that our delivery system enhances the therapeutic potential of poly I:C and could support the development of safer and more effective immune-based cancer treatments.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Bone marrow competition affects the long-term dynamics of imatinib resistance in chronic myeloid leukemia - Poster #221

Presenter: Mia Brunetti, Université de Montréal

Other Authors: Morgan Craig

ABSTRACT_ID#2987

ABSTRACT

Over the past decades, the adoption of molecularly targeted therapies has significantly improved the survival of patients with chronic myeloid leukemia (CML). However, sustained remission is not always achieved, and resistance to the standard first-line tyrosine kinase inhibitor (TKI), imatinib, continues to result in a range of unfavourable treatment responses. To better understand the long-term dynamics of leukemic cell populations that contribute to imatinib resistance, we developed a mathematical model of the hematopoietic hierarchy under CML that incorporates spatial competition within the bone marrow. The model was calibrated using clinical BCR-ABL1 transcript data from patients undergoing imatinib treatment with either short- or long-term responses. By performing parameter sensitivity analysis on our mechanistic model and applying the European LeukemiaNet 2020 guidelines, we identified key disease features associated with treatment failure. Furthermore, our simulations revealed that differences in the long-term kinetics of the various cell populations, influenced by bone marrow competition, have minimal impact on BCR-ABL1 transcript levels. These findings suggest that while BCR-ABL1 transcript monitoring is effective for assessing treatment response, it may not fully capture the underlying in vivo cell dynamics driving imatinib resistance in CML. Hence, our model offers new insights into the mechanisms of imatinib resistance and contributes to a deeper understanding of CML treatment dynamics over time.

LAY SUMMARY

Our mechanistic model of CML, which incorporates spatial competition within the bone marrow, successfully identified features of the disease associated with imatinib treatment failure. Thus, mathematical modelling can serve as a valuable tool for studying the long-term behaviour of both healthy and leukemic cell populations under standard therapy. We found that variations in the long-term kinetics of leukemic and healthy hematopoietic cells have minimal impact on BCR-ABL1 transcript levels. This suggests that imatinib resistance in CML is driven by complex in vivo cellular dynamics, which are not fully captured by transcript monitoring alone.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Targeting Collagen Deposition in Radiation Fibrosis: Proof-of-Concept Compound Discovery - Poster #222

Presenter: Pierre-Antoine Bissey, Princess Margaret Cancer Centre / UHN

Other Authors: Ross Mancini, Justin Williams, Wei Shi, Leonardo Massignan, Mikolaj Zabinski, Mark Reed, Kenneth Yip, Liu Fei-Fei

ABSTRACT_ID#2439

ABSTRACT

Introduction: Radiation therapy is essential in cancer treatment but often causes radiation-induced fibrosis (RF), marked by excessive collagen deposition. RF impairs tissue function, increases surgical risk, and lacks effective therapies. Building on prior work identifying caffeic acid phenethyl ester (CAPE) as a modulator of fibrotic pathways, we conducted a structure-activity relationship (SAR) study to develop more potent analogs. Methods: A library of 150 CAPE analogs was screened for anti-fibrotic activity. Collagen secretion was first evaluated via ELISA, followed by RT-qPCR and Western blot in vitro. In vivo efficacy was assessed in a limb irradiation model (40Gy) in mice. Weekly X-ray imaging measured tibiofemoral joint angles, with 7g foot weights ensuring standardized limb extension. At endpoint, collagen was quantified by Picrosirius red, Masson's trichrome staining, and ELISA. Compounds were administered intraperitoneally (100 mg/kg for 6 weeks or 50 mg/kg for 12 weeks). Results: About 75% of the compounds reduced collagen secretion in vitro, and a similar proportion downregulated collagen gene expression. Western blot confirmed consistent suppression in 25% of the original library. After metabolic stability profiling, compound 3w emerged as the top candidate. In vivo, 3w significantly increased tibiofemoral joint angles, indicating improved limb mobility and reduced fibrosis. Histological analysis showed decreased collagen deposition in irradiated skin. ELISA confirmed lower pro-collagen 1a1 levels, supporting the compound's efficacy. Conclusion: We identified compound 3w, a potent CAPE analog that reduces fibrosis by suppressing collagen production and deposition. These findings support further development of 3w as a therapeutic candidate for RF.

LAY SUMMARY

Radiation therapy can cause long-term tissue damage: While radiation helps treat cancer, it can also lead to fibrosis—scarring that stiffens tissues, reduces mobility, and makes surgeries more difficult. We developed a new compound that shows promise in preventing this damage: By modifying a natural substance called CAPE, we created a new version (called compound 3w) that effectively reduced collagen buildup—the main cause of fibrosis—in both lab tests and animal models. The compound improved movement and reduced scarring in treated mice: Mice given compound 3w after limb irradiation had better joint movement and significantly less tissue scarring, suggesting it could become a future treatment for radiation-induced fibrosis.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Bioresponsible Stable Lipid Nanoparticles for Lung-Selective RNA Transfection to Treat Lung Metastasis and Respiratory Viral Infections - Poster #223

Presenter: Mo Chen, McGill University

Other Authors: Yueyang Deng, Zhenlong Liu, Rongtuan Lin, Guojun Chen

ABSTRACT_ID#2837

ABSTRACT

Lipid nanoparticles (LNPs) are key non-viral carriers for RNA-based therapeutics, yet their broader utility is limited by intrinsic instability, uncontrolled cytosolic RNA release, and predominant hepatic accumulation. Here, we report a coated LNP (cLNP) formulation that integrates a phenylboronic acid (PBA)-modified anchoring lipid to enable covalent polyvinyl alcohol (PVA) coating, conferring enhanced structural stability and a PEG-free stealth surface. Additionally, incorporation of glutathione (GSH)-cleavable ionizable lipids facilitates intracellular, bioresponsive LNP disassembly and RNA release in the cytosol. The cLNPs efficiently deliver diverse RNA cargos, including mRNA, siRNA, and antiviral 5'pppRNA, with preserved transfection potency post-lyophilization and favorable biocompatibility. Notably, systemically administered cLNPs exhibit unique lung-selective RNA transfection, despite predominant liver accumulation. Therapeutically, cLNP-mediated delivery of IL-12 mRNA inhibited lung metastases in a murine melanoma model, and cLNP delivery of 5'pppRNA conferred broad-spectrum antiviral efficacy, markedly improving survival in influenza and respiratory syncytial virus infection models. Collectively, this cLNP platform advances RNA delivery technology, offering a promising strategy for pulmonary-targeted RNA therapeutics.

LAY SUMMARY

1. Traditional lipid nanoparticles (LNPs) are fragile and often unstable during storage or in the body. This study introduces a new LNP coating using polyvinyl alcohol (PVA) attached through strong chemical bonds, which enhances structural stability and avoids immune reactions caused by PEG-based stabilized lipids.
2. The new LNPs include special lipids that break down only inside cells where glutathione levels are high. This targeted breakdown allows for controlled and efficient release of RNA into the cell, improving the effectiveness of RNA-based treatments.
3. Unlike conventional LNPs that mainly go to the liver, these modified nanoparticles selectively deliver RNA to the lungs. This targeting makes them especially promising for treating lung-related diseases like cancer and respiratory infections with fewer side effects on other organs.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Identification of Gemcitabine-Resistant Populations Using scRNAseq in TNBC Patient-Derived Xenografts - Poster #224

Presenter: Sandrine Busque, McGill

Other Authors: Hellen Kuasne, Hamed Najafabadi, Constanza Martinez, Paul Savage, Anne-Marie Fortier, Anie Monast, Sandrine Omeroglu, Jamil Asselah, Nathaniel Bouganim, Sarkis Meterissian, Ariel Madrigal, Morag Park

ABSTRACT_ID#2452

ABSTRACT

The standard of care for Triple-negative breast cancer (TNBC) remains neoadjuvant chemotherapy in combination with surgery, radiation, and immunotherapy. While chemotherapy is effective in some patients, up to 50% of patients are intrinsically unresponsive or acquire resistance. These patients exhibit the highest rates of recurrence and distant metastasis. Hence, addressing chemotherapy resistance in metastatic TNBC, is a pressing clinical challenge. We hypothesized that establishing and analyzing models of disease progression of mTNBC would allow us to gain mechanistic insight into secondary resistance and identify potential markers and new therapeutic targets. We generated treatment progression models from a primary TNBC patient-derived xenograft (PDX) and its patient-matched lung metastasis PDX. We treated the metastasis PDX with Gemcitabine, mirroring the patient's treatment, and obtained residual disease that relapsed and, through treatment cycles, developed resistance. We performed single-cell RNA sequencing of these models and demonstrated that resistant cells clustered distinctly from untreated and rebound models, with residual disease split between these populations. We identified markers of a subpopulation in the untreated metastasis, which is maintained at the residual disease stage and is enriched in the resistant tumour, including WFDC2, a small secreted protein used as a biomarker in ovarian cancer. We validated these findings in vivo in tumour lysates and tumour sections at each step of treatment progression. Our current work focuses on characterizing the role of WFDC2 in resistance to gemcitabine in PDX-derived cell lines and in vivo.

LAY SUMMARY

Patient-derived xenograft treatment progression models allows us to model residual disease, relapse and resistance in vivo
Single-cell RNA sequencing of these models identifies markers of gemcitabine resistant populations

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Utilizing phytoglycogen nanoparticles for the anti-cancer therapeutic delivery of long double-stranded RNA - Poster #228

Presenter: Sarah Poynter, Wilfrid Laurier University

Other Authors: Natalie Aldor, Abigail Peralta, Nicholas Jadaa, Erin Ernst

ABSTRACT_ID#3014

ABSTRACT

Long double-stranded (ds)RNA molecules are known to be immunostimulatory across a variety of cell types and species. To maximize the therapeutic potential of immunostimulatory nucleic acids, an effective carrier is essential—both to protect the RNA in the extracellular environment and to enhance cellular uptake. Immune evasion is a hallmark of cancer, posing a significant challenge to the immune system's ability to eliminate tumor cells. dsRNA has the potential to activate immune signaling in cancer cells, leading to the production of type I interferons and proinflammatory cytokines, which in turn alert and engage the immune system. The objective of this study is to utilize nanoparticles to deliver immunostimulatory nucleic acids as a strategy to activate antitumor immune responses. Nanophytoglycogen nanoparticles were employed to deliver long dsRNA, either in the form of in vitro transcribed RNA or polyinosinic:polycytidylic acid (poly I:C), to a panel of human cancer cell lines (SKOV-3, OVCAR-3, SKBR-3, and MCF7). Cells treated with dsRNA-loaded nanoparticles exhibited a significantly enhanced immune response compared to treatment with dsRNA or nanoparticles alone. This response was quantified using qRT-PCR, revealing increased transcript levels of interferon-stimulated genes and proinflammatory cytokines, alongside reduced cell viability as assessed with fluorescent indicator dyes. Fluorescent microscopy using rhodamine-labeled dsRNA showed robust uptake in all tested cell lines, with nanoparticle-mediated delivery achieving greater internalization than free dsRNA, primarily via class A scavenger receptors. These findings support the potential for future in vivo investigations and offer a promising direction for the development of nanoparticle-based anticancer immunotherapies.

LAY SUMMARY

Our immune system can be trained to recognize and fight cancer, but tumors often find ways to hide from it. One way to “wake up” the immune system is by using special molecules called double-stranded RNA (dsRNA), which can trigger immune responses in many types of cells. dsRNA needs protection and help getting into cells to work effectively as a therapy. In this study, we explored the use of tiny delivery vehicles called nanoparticles to carry dsRNA into cancer cells. We used nanoparticles made from a type of starch that comes from sweet corn to deliver dsRNA into several human cancer cells. We found that cells treated with dsRNA-loaded nanoparticles showed strong immune responses. These immune responses included increased levels of molecules that help signal the immune system and a reduction in cancer cell survival. We also used fluorescent tags to track the dsRNA and saw that the nanoparticles helped it enter the cells more efficiently. Overall, this approach shows promise for developing new cancer treatments that help the immune system recognize and attack tumour cells. Future research in animals will be needed to test how well this strategy works in the body.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Improving glioblastoma therapy via multivariant-receptor targeted photodynamic porphyrin nanoparticles - Poster #230

Presenter: Hanyi Weng, University of Toronto

Other Authors: Maneesha Rajora, Juan Chen, Gang Zheng

ABSTRACT_ID#2810

ABSTRACT

Porphyrin-mediated photodynamic therapy (PDT) uses light to activate porphyrins to produce reactive oxygen species, causing localized cell death restricted to cells that internalize porphyrin and are exposed to light. PDT holds strong potential for eliminating infiltrative glioblastoma (GBM) cells beyond while sparing healthy brain tissue. To realize its full potential, porphyrin nanoparticles capable of crossing the blood-brain barrier (BBB) and achieving GBM-specific uptake are urgently needed. Lipoprotein receptors are expressed on the BBB and are often upregulated in GBM cells, making them attractive targets for delivery. Inspired by the natural lipoproteins, we developed a porphyrin lipoprotein (PLP) nanoparticle incorporating a synthetic targeting peptide with the following advantages: • Structural advantage: The PLP forms stable, spherical nanoparticles (~20 nm), favouring brain diffusion. It carries ~30% porphyrin with ~97% fluorescence quenching, minimizing premature porphyrin activation before cellular internalization. • Targeting advantage: The targeting peptide enables uptake via multiple lipoprotein receptors (LDLR, LRP-1, SRB1, HSPG), supporting ubiquitous delivery despite inter-patient receptor variability. • Enhanced and selective PDT efficacy: In GBM cells, the PLP delivers 3× more porphyrin and achieves 2× greater PDT efficacy than existing porphyrin nanoparticles, while showing selectivity over healthy brain cells. • Clinical translatability: The peptide was safe-to-use in human as it was evaluated in a Phase I clinical trial. As a synthetic molecule, it is also scalable and cost-effective. This novel PLP combines structural, targeting and clinical design advantages for potential BBB penetration and GBM-specific uptake. Clinically relevant animal models will be used to validate its performance in vivo.

LAY SUMMARY

A novel nanoparticle was developed with a clinical translation perspective to address drug delivery challenges in treating invasive glioblastoma (GBM) cells, which often resist standard therapies and infiltrate healthy brain regions beyond surgical reach. By targeting multiple receptors expressed on both the blood-brain barrier and GBM cells—at higher levels than in healthy brain tissue—the nanoparticle enables specific delivery to GBM cells in brain and accommodates patient-to-patient variability in receptor expression. The targeting component has already been tested in Phase I clinical trials to prove safety in human and it is scalable and cost-effective to manufacture. This nanoparticle carries a light-sensitive therapeutic agent (porphyrin) and keeps it inactive until it reaches GBM cells. It stably loads ~30% porphyrin and quenches 97% of its activity, preventing premature activation during circulation. Once internalized by GBM cells, porphyrin can be activated by light, enabling localized destruction of cancer cells while sparing healthy brain tissue. Compared to existing platforms, the nanoparticle delivers 3-fold more porphyrin to GBM cells and achieves 2-fold the treatment effect in laboratory models. It also demonstrates selective toxicity to cancer cells over normal brain cells, supporting both its high therapeutic efficacy and safety.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Drug tolerance and persistence to EGFR inhibitor treatment are mediated by an ILK-SFK-YAP signaling axis in lung adenocarcinoma - Poster #231

Presenter: Rocky Shi, BC Cancer Research Institute

Other Authors: Dylan Farnsworth, Christopher Febres-Aldana, Justine Chow, Ravinder Sheena, Tejveer Atwal, Juan Lui Gomez Marti, Samantha Li, Kiersten Thomas, Che-Min Lee, Shannon Awrey, Paul McDonald, Romel Somwar, Shoukat Dedhar, Marc Ladanyi, Kevin Bennewith, William Lockwood

ABSTRACT_ID#2904

ABSTRACT

Combating resistance to targeted therapy remains a major challenge to improving lung cancer care. Epithelial-mesenchymal transition (EMT) in tumour cells is an established non-genetic resistance mechanism to EGFR tyrosine kinase inhibitors (TKI) that is also associated with worse outcome in patients. Here we demonstrate that integrin-linked kinase (ILK) is an important driver of EMT-mediated TKI resistance in lung adenocarcinoma (LUAD) by promoting a drug-tolerant persister (DTP) cell phenotype. Our results indicate that high ILK expression is associated with EMT in LUAD patients and that genetic suppression of ILK can limit EMT progression and reduce the viability of DTP cells by impairing YAP activation, ultimately improving osimertinib (Osi) sensitivity in LUAD cells. In addition, we found that LUAD cells in extracellular-matrix rich microenvironments have increased dependences on ILK-mediated cytoprotective effects during Osi treatment. Importantly, LUAD cells with high ILK expression are able to persist during EGFR-TKI treatment, progressing through EMT to develop EGFR-TKI resistance. To improve clinical translatability of our findings, we showed that pharmacological inhibition of ILK can suppress EMT and improve Osi response in LUAD cells. Lastly, we found that strong immunohistochemistry staining of ILK in patient biopsies was significantly associated with, and may be used to predict, receptor tyrosine kinase-independent mechanisms of EGFR-TKI resistance. Overall, our results suggest that ILK is an important regulator of EGFR-TKI response and may be exploited as a predictor for acquired resistance, providing evidence for co-targeting ILK with EGFR to better control minimal residual disease and EGFR-TKI resistance in lung cancer.

LAY SUMMARY

Integrin-linked kinase (ILK) is associated with tumour progression and therapy resistance in lung cancer. ILK promotes lung cancer cell survival during initial stages of treatment by activating SFKs and YAP, providing a window of opportunity for lung cancer cells to evolve and become fully drug resistant. Suppression of ILK improves response to therapy and delays the onset of drug resistance in lung cancer.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

Advances in High Parameter Spatial Proteomics for Cancer Research - Poster #232

Presenter: Toby Astill, Standard BioTools

Other Authors: Jim Mansfield

ABSTRACT_ID#2839

ABSTRACT

Target identification and validation is a crucial process in any cancer drug development pipeline. Successful detection of target proteins in diseased samples can improve drug discovery by streamlining the process and thereby reducing failure rate and cost. Particularly in cancer, accurately evaluating clinically relevant targets provides key biological mechanisms. Its potential for disease modulation may also allow for more personalized and effective therapies. IHC is the goldstandard technique for target detection by spatial proteomics. Limitations related to plexity, quantitation and false signal detection are frequently observed using IHC. Furthermore, day-to-day variability due to multiple steps of signal amplification and pigment mistaken for true signal can misinform pathologists about target expression. Imaging Mass Cytometry (IMC) technology is a multiplexed imaging technique that incorporates quantitative assessment of 40-plus biomarkers simultaneously on the same slide. The Hyperion XTi Imaging System permits 24/7 data acquisition and provides biological insights critical for assessment of the tumor microenvironment (TME). Our work in this study demonstrates the added value that imaging with IMC technology brings to target validation in drug discovery. Like IHC, IMC technology provides measurements of proteins, but with an improved linear dynamic range that can also be put into context with 40-plus different proteins on the same tissue section. IMC technology achieves this level of detection through multiplex primary antibody detection. The ability to multiplex enables multiple drug targets of interest to be analyzed simultaneously, providing insights into the potential for combination therapies.

LAY SUMMARY

Cancer target assessment in tissues using imaging mass cytometry technology offers an advantage over traditional IHC methods by providing accurate measure of protein expression with biological context. The ability of imaging mass cytometry technology to provide high-dimensional spatially resolved data makes it a powerful tool for target validation applications and shows that it is poised to significantly contribute to cancer drug discovery.

Drug Discovery, Design, Delivery and Resistance

Sunday, November 2, 2025

Combining novel Ran GTPase inhibitors with PARP inhibitors as a therapeutic strategy in ovarian cancer -

Poster #233

Presenter: Sabrina Ritch, University of Montreal Hospital Research Centre (CRCHUM)

Other Authors: Euridice Carmona, Ryhem Gam, Wenying Piao, Isaline Bontemps, Benjamin Péant, Xiaohong Tian, Jian Hui Wu, Diane Provencher, Anne-Marie Mes-Masson

ABSTRACT_ID#2818

ABSTRACT

High grade serous ovarian cancer (HGSC) is characterized by high degrees of aneuploidy. We have shown that downregulation of the small GTPase Ran decreases cell proliferation and promotes apoptosis of aneuploid cells, with little effect on normal diploid cells, making RAN a promising therapeutic target for HGSC. We have developed a series of novel small-molecule inhibitors of Ran (RANi) that disrupt aneuploid HGSC cell line proliferation without affecting normal diploid cells, while also inhibiting the homologous repair (HR) pathway. Resistance to PARP inhibitors (PARPi) is thought to involve the reactivation of the HR pathway in cancer cells. Thus, combining PARPi with drugs that inhibit the HR pathway offers a potential strategy to overcome this resistance. This study aims to test various RANi alone or in combination with Olaparib (PARPi) on ex vivo live human ovarian cancer (OC) samples using microfluidic models. Specifically, micro-dissected tumors (MDTs) of OC xenograft and patient samples were loaded into microfluidic devices and treated with selected RANi inhibitors and/or Olaparib. MDTs were then formalin-fixed, paraffin embedded and analyzed via immunofluorescence for proliferation (KI67) and apoptosis (cleaved-caspase 3 (CC3)). Preliminary results demonstrated a dose-dependent decrease in KI67+ cells, followed by an increase in CC3+ cells within the tumour epithelium of OC MDTs treated with RANi alone compared to controls. MDTs treated with a combination of Olaparib and RANi demonstrated higher levels of apoptosis than with each inhibitor alone. While a larger sample cohort needs to be studied, our data suggest a potential therapeutic efficiency of this combination.

LAY SUMMARY

A distinct feature of ovarian cancer cells is their abnormal number of chromosomes. We have shown that blocking the protein Ran causes death in cells with an abnormal number of chromosomes but not in normal cells. Therefore, we are developing new drugs to target the Ran protein in ovarian cancer cells, to kill tumours without affecting normal tissue. Resistance to PARP inhibitors, a common treatment used against ovarian cancer, involves the reactivation of a DNA repair pathway that is blocked by our Ran inhibitors. Therefore, we aim to test whether combining these two inhibitors can make the treatment more effective. Inhibitors were selected for this project based on their ability to slow the growth of ovarian cancer cells with little effect on normal cells. Our goal is to test the effects of these Ran inhibitors alone or in combination with PARP inhibitors, using tumour samples grown in mice or generously donated by ovarian cancer patients. Using miniature lab models, we have observed that RANi alone caused cancer cells to stop growing and begin dying. Even better, combining RANi with Olaparib led to even more cancer cell death, suggesting this combination could be a powerful new treatment option, pending further testing.

Drug Discovery, Design, Delivery and Resistance

Monday, November 3, 2025

THNB-3: Discovery, development and in vitro validation of a first-in-class LIN28 Inhibitor for the treatment of refractory ovarian cancer - Poster #234

Presenter: Amy Pham, University of Calgary

Other Authors: Yusheng Zhao, Patrick Sipila, Gregg Nelson, Anu Narendran

ABSTRACT_ID#2954

ABSTRACT

Background: Ovarian cancer remains the most lethal gynecological neoplasm, with high relapse rates and limited treatment options. The RNA-binding protein LIN28 regulates stem cell pluripotency and is frequently reactivated in germ cell and ovarian tumors. By blocking the tumor suppressor miRNA let-7, LIN28 promotes oncogene activation, stemness, and disease progression. Overexpression of LIN28 is associated with tumor aggressiveness and therapy resistance, making it an important target for novel therapeutics discovery for ovarian cancer. Methods: THNB-3 (MW 639.73 g/mol) is a novel small molecule inhibitor identified via structure-based virtual screening for high-affinity binding to LIN28's zinc knuckle domain. It was synthesized and evaluated for cytotoxicity in LIN28-positive (PA-1) and LIN28-negative (SKOV-3) ovarian cancer cell lines. Cell viability following treatment was assessed using Alamar Blue assays. let-7 miRNA levels were measured by RT-qPCR. Synergy assays with a panel FDA-approved drugs (n =225) were performed using the BLISS/ZIP method and validated in primary ovarian tumor cultures. Nonspecific toxicity was assessed in normal lymphocytes and fibroblasts. Results: THNB-3 selectively inhibited growth in LIN28-positive cells ($IC_{50} = 58 \mu M$), with minimal toxicity in LIN28-negative and normal cells. It increased let-7 expression and induced apoptosis, marked by PARP cleavage. Synergistic interactions were observed with agents including paclitaxel and etoposide. Conclusion: THNB-3 demonstrates selective activity in LIN28-expressing ovarian cancer cells let-7 upregulation. Its synergy with established chemotherapeutics highlights its potential clinically feasible combination therapies. These results support continued preclinical development, including in vivo validation and pharmacokinetics, to advance safe and effective LIN28-targeted treatment strategies for ovarian cancer.

LAY SUMMARY

We discovered a new drug (THNB-3) that targets a protein called LIN28, which is linked to aggressive and treatment-resistant ovarian cancer. LIN28 blocks a natural tumor suppressor called let-7, helping cancer cells grow and survive. By targeting LIN28, our goal is to shut down this pathway and stop tumor progression.

THNB-3 worked specifically in ovarian cancer cells that have LIN28, without harming normal cells. In lab tests, the drug reduced cancer cell growth and triggered cell death, while having little effect on healthy cells—showing promise for a more targeted and safer treatment option.

When combined with existing chemotherapy drugs like paclitaxel, THNB-3 worked even better. These combinations killed more cancer cells than either drug alone, suggesting that THNB-3 could strengthen the effects of current treatments and help overcome resistance.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Preventing Cancer Through Métis Cultural Revitalization: A Framework for Saskatchewan - Poster #236

Presenter: Maria Diaz, University of Saskatchewan

Other Authors: Gary Groot, Calvin Racette

ABSTRACT_ID#2448

ABSTRACT

The Canadian Métis population experiences disproportionately higher cancer rates, leading to reduced life expectancy. Culture is vital to Indigenous health, offering belonging and improving well-being. This community-based study aimed to create a Métis Cultural Continuity Framework for cancer prevention that can be adapted for use in other Métis communities. Participants were recruited from seven Métis Nation–Saskatchewan (MN-S) cultural events held between June 2022 and March 2023, where researchers were engaged in Métis cultural activities (e.g., beading, hide tanning). Semi-structured telephone interviews with 24 MN-S citizens (12 men, 12 women) were analyzed using the Collective Consensual Data Analytic Procedure (CCDAP), enabling the diverse Research Advisory Committee members to identify key themes. These themes included colonization and its impacts, community, cultural revitalization, strength, and pride. Participants highlighted the profound effects of colonization on their lives and communities while celebrating the resilience and solidarity that define the Métis community. Six key themes were used to develop a Métis Cultural Revitalization Framework for Cancer Prevention, rooted in the Red River Cart's symbolism, a significant cultural emblem for the Métis people. Each theme was conceptualized as a spoke of the cartwheel, playing a vital role in ensuring both stability and progress for the cart and the Métis community. For the Métis community, strong cultural connections are essential for fostering a sense of belonging, reducing risk behaviors, and promoting both mental and physical well-being. These factors are vital for a healthy lifestyle and can help prevent diseases such as cancer

LAY SUMMARY

Métis communities experience higher cancer rates and lower life expectancy compared to the general Canadian population. This highlights the urgent need for culturally relevant cancer prevention strategies. Strong cultural connections may reduce risk behaviors that lead to cancer. Cultural continuity appears to enhance holistic well-being, contributing to a healthy lifestyle, promoting health, and potentially preventing cancer. The Métis Cultural Revitalization Framework for Cancer Prevention, symbolized by the Red River Cart, offers a culturally grounded tool that supports health promotion and can be adapted across other Métis communities in Canada.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Storytelling: Cancer Control through Traditional Healing: An Indigenous-Driven Implementation Science Project - Poster #236.1

Presenter: Lana Ray

Other Authors: Gabrielle Smith, Mulugeta Wondim

ABSTRACT_ID#3371

ABSTRACT

The Truth and Reconciliation Commission of Canada's 18th call to action recognizes that the current status of Indigenous health is a direct result of Canadian government policies and the 22nd call to action urges for more recognition of the value of traditional healing practices and involvement of Traditional knowledge holders in health care. Despite these calls to action there remains a lack of cancer control interventions and cancer research focused on traditional healing. This poster presentation provides an overview of The Waasegiizhig Nanaandawe'iyewigamig Mino-Bimaadiziwin: Cancer Prevention through Traditional Healing (WNMB) project. The project utilizes an Indigenous-directed implementation science approach to examine traditional healing as a multi-faceted cancer control strategy as well as the enablers and barriers to the effective implementation of traditional healing as a cancer control strategy in an Indigenous governed health care setting. With the project currently underway, the poster presentation will focus on a description of the role of traditional healing in cancer control and the study design.

LAY SUMMARY

- Traditional healing is an underexplored multi-faced cancer control strategy.
- It is possible and necessary to engage in Indigenous-driven cancer research.
- Traditional healing practices are relevant today, yet there is a research gap regarding how to most effectively implement traditional healing in health care settings.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Breast Cancer System Performance: Wait Times, Stage at Diagnosis, and Survival Rates for First Nations and All Other Women in BC - Poster #237

Presenter: Kevin Linn, University of British Columbia

Other Authors: Nadine Caron

ABSTRACT_ID#3190

ABSTRACT

Objectives: This presentation will focus on breast cancer wait times for resolution after abnormal screening mammography, stage at diagnosis, and breast cancer survival, and will discuss potential policy options. **Methods:** A population-based retrospective cohort methodology was utilized using linked administrative health data. Wait time was defined as the percentage of women with an abnormal program screening mammogram result who received their diagnosis (benign or cancer) within the recommended time interval from the screening date (≤ 5 weeks without tissue biopsy, OR ≤ 7 weeks with tissue biopsy). The percentage of incident stage I-IV and stage unknown breast cancers was calculated as a proportion of the total incidence count of all five stages. For survival, age-stratified hazard ratios of the cancer-specific survival rate in First Nations women relative to the survival rate in all other women in the province were calculated. **Results:** A higher proportion of breast cancers among First Nations women were found to be diagnosed at Stage II, while most breast cancers are diagnosed at Stage I among all other women in BC. Delays are seen in the time to resolution of abnormal screens, with First Nations women waiting longer for resolution. Lastly, breast cancer survival rates among First Nations were lower than those of All Other BC residents in the period of 2008-2017, with an age-stratified hazard ratio of 1.36 (95% CI: 1.14-1.62). **Conclusion:** Our findings show disparities in breast cancer care in the province. The cumulative impact of delays and disparities in care may be contributing to lower survival.

LAY SUMMARY

Disparities in breast cancer care exist in British Columbia Targeted interventions and system improvements are needed.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Understanding the Cancer Screening Experiences of Métis People in Alberta - Poster #238

Presenter: Amanda Andrew, Otipemisiwak Métis Government

Other Authors: June Kima, Ashton James, Reagan Bartel, Karen Kopciuk, Elsie Zhang, Meghan Macphail

ABSTRACT_ID#2422

ABSTRACT

Background Métis people experience unique barriers to cancer screening programs in Alberta. The Otipemisiwak Métis Government sought to explore Métis Albertans' cancer screening experiences to understand the barriers and facilitators to accessing screening programs. **Methods** We used qualitative approaches informed by Métis Ways of Knowing to engage 31 individuals across Alberta. Data collection was completed through two in-person Métis gatherings and six semi-structured telephone interviews. Gatherings included talking circles and cultural activities, with discussions lasting approximately three hours. Topics discussed included experiences with accessing screening programs, the quality of care received during appointments, and the supports needed to improve access to screening programs. Discussions were audio-recorded, transcribed, de-identified, and thematically analyzed using NVivo 14 Software. **Results** Four themes emerged: 1) the impact of patient-provider communication on screening experiences, 2) a broken healthcare system and lack of access to care, 3) a need for support and safety, and 4) health prevention behaviours. Participants overwhelmingly described long wait times and unclear next steps after a cancer screening appointment, emphasizing the need for navigation and mental health support. **Conclusion** This study provides a comprehensive understanding of Métis cancer screening experiences and offers direction for improving them. Providing accurate and timely information, enhancing mental health support, and opportunities for community connection are concrete actions that can be implemented. Additionally, the healthcare system should adopt interventions to enhance cultural safety and implement alternative delivery models of screening to reduce inequities, improve access, and ultimately increase cancer screening participation among Métis people in Alberta.

LAY SUMMARY

There is a need for clear communication from healthcare professionals. Unclear next steps, a lack of follow-up, and long wait times contribute to increased anxiety about cancer screening and future avoidance of the healthcare system. Clear and effective communication from healthcare professionals is crucial in helping people access cancer screening programs within the recommended guidelines, including referrals and follow-up after cancer screening appointments. Mental health and navigation support are needed throughout the continuum of cancer care, including cancer screening, to help address the anxiety associated with long wait times and overall lack of communication from healthcare professionals. Various types of support, such as self-advocacy and empowerment, a navigator or support person, and peer support connections, are necessary to help Métis Albertans feel supported while navigating the healthcare system. Healthcare systems should create respectful and inclusive environments where Métis Albertans feel safe and supported while accessing cancer screening. This can be achieved by implementing practices that support healthcare professionals' understanding of Métis people's experiences and realities, thereby reducing power imbalances and building trust with Métis Albertans.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Nishtohtamihk li kaansyr (Understanding Cancer): Cancer Screening, Outcomes and Experiences among Métis People in Alberta - Poster #239

Presenter: Karen Kopciuk, Cancer Care Alberta

Other Authors: June Kima

ABSTRACT_ID#2794

ABSTRACT

Cancer prevention and screening are topics of great importance to the Métis community. Métis-specific data on inequities in cancer screening participation, experiences, follow-up care, and outcomes are lacking, which are essential to develop targeted strategies and interventions that will reduce the cancer burden for Métis people. The objectives of this research project were to: (1) develop a Métis – specific cancer screening questionnaire and use it to assess knowledge, attitudes, beliefs, behaviours, barriers, and facilitators in the Métis population within Alberta, and, (2), to quantify screening uptake and follow-up care between Métis people compared to their non-Métis AB counterparts using a system-level approach of screening program data. The Otipemisiwak Métis Government, in partnership with the Alberta Health Services Screening Programs, carried out this research project. An overview of how the Métis and Western principles and perspectives were employed to benefit from the wisdom of both forms of knowledge in this research project will be shared. Some of the key research results will also be shared as well as the benefits to the research team who learned about Métis Ways of Knowing and culture. Practical approaches for conducting collaborative, respectful research projects will also be given. Improved cancer prevention, screening support and resources based on Métis-specific data and information will now be possible.

LAY SUMMARY

Comprehensive and objective data about cancer screening participation, follow-up, experiences, and outcomes for Métis people are lacking. Co-designed and co-lead research project between the Otipemisiwak Métis Government and Alberta Health Services Screening Programs developed and utilized a Métis – specific cancer screening questionnaire and compared screening program indicators between Métis and non-Métis populations following data linkage with Métis citizen identifiers and provincial screening databases. Results from a Western perspective will be shared, including the benefits to the research team from learning about Métis culture, history and Ways of Knowing.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Métis end-of-life care: Weaving Métis experiences, knowledge and wisdom into the Indigenous End of Life Doula Guide. - Poster #240

Presenter: Katina Pollard, MNBC

Other Authors:

ABSTRACT_ID#2929

ABSTRACT

Métis health is rooted in family ties, kinship, and community strength that wraps around their physical, mental, emotional, social, and spiritual well-being. Métis people's understanding of wellness shines through their experiences, resiliency, and cultural identity. No Métis person is the same, nor is their experience of being Métis the same. In partnership with Douglas College, the re-design of the Indigenous End of Life Doula Guide (IEOLDG) to be Métis – specific will provide Métis people the opportunity to weave their knowledge, values, and wisdom into a new curriculum that will increase awareness and offer a new resource on Métis end-of-life-care. MNBC's Ministry of Health is guided by seven foundational principles to our work. These seven principles will guide the Métis -Specific Indigenous End of Life Doula curriculum with a strong inclusion of principle #1, community-based. This principle ensures the work is grounded in community voice, is done in collaboration and consultation with the community, and allows Métis people to tell their own stories. Métis voice is critical to improving health equity and health and wellness outcomes for Métis. The re-design will include voices from Community Leaders, Elders, Youth, Women, and 2SLGBTQIA+ representatives. Métis people have experience, knowledge, and traditions to share. Traditional cultural practices, ceremonies, protocols, and medicines will be embedded within the materials and available for those who wish to learn about Métis end-of-life care.

LAY SUMMARY

Métis End-of-Life Doula plays a deeply compassionate role, ensuring that individuals experience their final transition with dignity, cultural connection, and support. Here are three key insights that guide this work: Honoring Métis Traditions & Teachings – End-of-life care isn't just about physical comfort; it's about spiritual and cultural continuity. Métis doulas incorporate storytelling, land-based healing, music, and ceremony to ensure individuals feel connected to their ancestors and heritage in their final moments. Building Community-Led Support – Métis end-of-life care is often collective, emphasizing the importance of family, Elders, and knowledge keepers. A doula serves as a bridge, helping to navigate grief, facilitate conversations, and ensure families have access to culturally safe wellness resources. Respecting Individual Journey & Choices – Every transition is unique, and Métis doulas empower individuals to shape their final days in ways that feel meaningful. Whether through personal traditions, comfort rituals, or advocacy for care decisions, the goal is to create a peaceful and supported passage that reflects their identity.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Distinctions-Based Cancer Prevention and Screening: Integrated Learnings from the Indigenous Alberta Healthy Communities Approach (IAHCA) and Alberta First Nations Cancer Prevention and Screening Practices (FNPP) Projects - Poster #241

Presenter: Jessica Smith, Alberta Health Services/ Primary Health Alberta

Other Authors: Trevor Baxter, Brenda Roland, Angeline Letendre

ABSTRACT_ID#2957

ABSTRACT

Métis Settlement and First Nations communities in Alberta experience distinct cancer prevention and screening challenges shaped by systemic, historical, and cultural factors. The Indigenous Alberta Healthy Communities Approach (IAHCA) and Alberta First Nations Cancer Prevention and Screening Practices (FNPP) Project have resulted in distinctions-based, Indigenous-led frameworks that support community-driven cancer prevention and screening grounded in cultural knowledge, ceremony, and holistic wellness. Guided by Indigenous methodologies, communities applied multiple assessment tools, including readiness assessments, the Community Supportive Settings Survey (CSSS), SOAR (Strengths, Opportunities, Aspirations, Results) analysis, and asset mapping, to identify priorities and community strengths. Engagement with Elders, Knowledge Holders, healthcare providers, and leadership ensured that action planning reflected community voices, cultural protocols, and self-determination. Findings demonstrate increased knowledge, strengthened leadership engagement, and improved supportive environments that foster positive healthy lifestyle choices. Community-led activities, such as gardens, physical activity programs, culturally appropriate screening promotion, UV protection initiatives, and inter-community partnerships, have contributed to sustainable, community-owned cancer prevention efforts. The integration of both IAHCA and FNPP pathways highlights how distinctions-based frameworks can support long-term improvements in health outcomes while honoring the unique governance, knowledge systems, and cultural identities of Métis and First Nations communities. This poster presents visual infographics illustrating readiness levels, priority actions, and sustained community-driven prevention activities, reflecting the strengths-based, culturally appropriate pathways emerging from these distinctions-based collaborations.

LAY SUMMARY

The IAHCA and FNPP projects provided distinctions-based, Indigenous-led frameworks for cancer prevention and screening in Métis Settlement and First Nations communities. Communities used readiness assessments, asset mapping, and cultural protocols to identify priorities, strengthen leadership, and guide prevention efforts. Sustainable, community-owned prevention actions, such as community gardens, screening programs, physical activity, and UV protection, reflect culturally appropriate pathways that promote wellness and support long-term health improvements.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Nâcinîhikêwin: To make an offering for help and healing - Poster #242

Presenter: Katina Pollard, MNBC

ABSTRACT_ID#2930

ABSTRACT

The Hope and Healing Gathering took place from February 19th to 21st, 2025, on the traditional, ancestral, and unceded territories of the Musqueam, Squamish, and Tsleil-Waututh First Nations, as well as the chosen home of the Sea to Sky Métis Association. This gathering was a central engagement initiative within our program Nâcinîhikêwin, fostering connection, learning, and culturally rooted wellness. The gathering featured a range of presentations, ceremonies, and workshops, beginning with an opening prayer, intention setting, and a Smudging Ceremony. Métis leaders and health professionals provided keynotes and discussions, including Madeleine McCallum's address on her breast cancer journey, as well as BC Cancer's presentation on Métis data and screening updates. Other notable sessions included Slow Medicine by Métis Registered Herbal Therapist Vanessa Prescott and Support for the Caregiver by Heather McCullough. A unique aspect of the gathering was its flexible, participant-centered approach on Day 2, allowing attendees to choose their own schedule. Workshops included Advanced Care Planning, Métis End-of-Life Guidance, Cancer Resources, and Sharing Your Story. Attendees could also visit vendors, booths, and self-care services, such as hair & makeup sessions and massage therapy. The final day focused on Métis Cultural Teachings and Networking, closing with a sharing circle on learnings and takeaways. This gathering served as an important engagement effort, bringing together Métis individuals, health professionals, and community members to exchange knowledge, strengthen connections, and foster healing. The insights gained will continue to shape future programming, ensuring that culturally safe, Métis-specific health supports remain accessible and impactful.

LAY SUMMARY

Culturally Safe Healing & Wellness – The gathering centered Métis traditions, ensuring spiritual, emotional, and physical well-being were honored. Smudging ceremonies, storytelling, and community connection reinforced holistic healing. **Flexible & Participant-Led Engagement** – Attendees were given agency over their experience, allowing them to choose workshops and wellness activities. This structure recognized the importance of rest, personal reflection, and self-care in the healing process. **Community & Knowledge Sharing** – From cancer screening updates to Métis end-of-life care, the gathering created space for education, collaboration, and storytelling. The voices of Elders, Knowledge Keepers, and health professionals strengthened learning and collective growth.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Enhancing Alberta Métis Settlements Participation in Cancer Screening: A Distinctions-Based, Community-Grounded Approach - Poster #243

Presenter: S. Trevor Baxter, Alberta Health Services/ Primary Health Alberta

Other Authors: Jessica Smith, Angeline Letendre, Brenda Roland

ABSTRACT_ID#2735

ABSTRACT

Métis Settlement communities in Alberta experience systemic barriers in accessing culturally appropriate cancer screening services. The Enhancing Alberta Métis Settlements Participation in Cancer Screening (EAMSS) project applies a distinctions-based, Indigenous-led approach to address these disparities that recognizes the unique histories, rights, and needs of Métis Settlement communities. The project aims to co-develop a Métis Settlements Cancer Prevention and Screening Strategy by: (1) gathering evidence to inform the strategy; (2) piloting culturally appropriate screening innovations; and (3) identifying Indigenous-specific indicators to enhance screening participation. This presentation will share how distinctions-based, community-led approaches have addressed inequities in cancer screening and advanced sustainable, community-driven solutions for Métis Settlements in Alberta. Key project components, including the application of Indigenous methodologies, community-based research principles, and Indigenous data governance will be described. Partnerships with Elders, healthcare providers, and community leadership have guided the integration of cultural protocols such as tobacco offering, smudging, ceremony, and gift-giving throughout the research process. Multiple data collection activities—environmental scans, literature reviews, sharing circles, community gatherings, and healthcare provider surveys—have supported meaningful community engagement that informed the development of new innovations. The pilot of these innovations are expected to demonstrate promising practices for improved screening participation, strengthened capacity, and an informed comprehensive, distinctions-based Cancer Prevention and Screening Strategy. EAMSS focus on positive healthy lifestyle choices will further reinforce these community-led solutions in promoting health and preventing cancer.

LAY SUMMARY

Métis Settlement communities in Alberta experience unique barriers to cancer screening, shaped by historical, systemic, and cultural factors. The EAMSS project partnered directly with Métis Settlements to co-develop a distinctions-based Cancer Prevention and Screening Strategy that integrates Indigenous knowledge, cultural protocols, and community leadership. Through Indigenous-led research methods, pilot interventions improved screening participation, strengthened community capacity, and promoted positive healthy lifestyle choices to support long-term cancer prevention.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

MNBC's Regional Health Coordinators - Poster #244

Presenter: Katina Pollard, MNBC

ABSTRACT_ID#2936

ABSTRACT

Métis Nation British Columbia (MNBC) plays a crucial role in advancing culturally safe and community-driven healthcare for Métis individuals across the province. At the heart of this initiative are Regional Health Coordinators (RHCs)—dedicated professionals who work closely with Métis communities to improve access to equitable, responsive, and culturally informed health services. RHCs serve as liaisons between Métis citizens, healthcare providers, and government agencies, ensuring that Métis perspectives, traditions, and priorities are embedded in health programming and policy development. Their work includes advocating for culturally safe care, supporting holistic wellness initiatives, and addressing systemic barriers that affect Métis health outcomes. Through community engagement, collaborative partnerships, and the promotion of self-determination in healthcare decisions, MNBC's Regional Health Coordinators strengthen relationships with Indigenous and non-Indigenous health systems while advancing key health priorities such as mental wellness, chronic disease prevention, elder support, and access to traditional healing practices. This model reinforces Métis-led health solutions, ensuring that care is rooted in Métis ways of knowing, being, and healing—ultimately shaping a more inclusive and responsive healthcare landscape in British Columbia.

LAY SUMMARY

Culturally Safe & Community-Driven Care – MNBC prioritizes Métis-specific healthcare by embedding traditions, perspectives, and priorities into health programs, ensuring services are equitable, responsive, and culturally informed. **Strengthening Advocacy & Partnerships** – Regional Health Coordinators (RHCs) serve as key connectors between Métis citizens, healthcare providers, and government agencies, advocating for holistic wellness while addressing systemic barriers in healthcare access. **Advancing Métis-Led Health Solutions** – MNBC

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Territorial collaboration for CRC screening: Strengthening programs through shared learning - Poster #245

Presenter: Josh Tobias, Canadian Partnership Against Cancer

Other Authors: Krisha Quiambao, Ashley Beggs, Gregory Doyle, Talia Pfefferle

ABSTRACT_ID#3282

ABSTRACT

Colorectal cancer (CRC) is a leading cause of cancer-related morbidity and mortality across Canada, with notable disparities in screening access and outcomes in the Territories. This session will explore how the Community of Practice (COP) strengthens CRC screening efforts by bringing together professionals and implementers from across the Territories. While each jurisdiction faces distinct challenges and develops its own programs, the COP creates space for sharing strategies, learning from one another, and co-developing approaches that are both innovative and culturally safe. Established with support from national partners in a convening role, including the Canadian Partnership Against Cancer, the COP has helped to facilitate cross-jurisdictional collaboration and ongoing knowledge exchange. Presenters will highlight outcomes that have emerged through this collaboration, such as increased access to screening, more culturally safe practices, and improved community engagement. Co-presenters working directly within territorial programs will share real-world examples of how participating in the COP has supported local success while also contributing to broader system improvements. By fostering interjurisdictional dialogue and amplifying community-driven insights, the COP enables sharing of lessons learned, avoids duplication of effort, and accelerates innovation. Ultimately, this collaborative model contributes to a shared goal – reducing CRC disparities and improving screening programs across the Territories.

LAY SUMMARY

1. Screening isn't just a one-time procedure – it's about building long-term, trusting relationships with communities. In many First Nations, Inuit, and Métis communities, long-standing systemic mistrust in healthcare requires programs to prioritize cultural safety, respectful engagement, and community-driven approaches. Successful programs integrate Western screening practices into culturally grounded understandings of health and wellness.
2. Many communities in the Territories face unique challenges, including operating across vast geographies with small, dispersed populations and limited healthcare infrastructure. The COP creates a collaborative space where implementers can share contextual challenges and co-create tailored, flexible solutions that respond to the lived realities of delivering care in remote settings.
3. Each Territory designs and delivers its own colorectal cancer screening program. This COP helps reduce administrative silos by connecting jurisdictions with similar challenges. This pan-Canadian collaboration enhances program quality, resource-sharing, and collective problem-solving, fostering lasting partnerships that support coordinated, innovative action.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Essential Medical Travel Assistance Program (EMTAP) - Poster #246

Presenter: Katina Pollard, MNBC

ABSTRACT_ID#3048

ABSTRACT

The Essential Medical Travel Assistance Program (EMTAP), led by Métis Nation British Columbia (MNBC), provides pre-approved funding and reimbursement to eligible Métis Citizens for travel to essential medical appointments, procedures, specialists, and other necessary health services not available in their local area. This pilot program is designed to assist low-income Métis Citizens facing financial barriers in accessing critical medical care, including cancer-related travel. EMTAP covers travel costs, accommodations, and meals, ensuring Métis individuals receive the care they need without undue financial hardship. Eligibility requires MNBC Citizenship, proof of medical necessity, and meeting income thresholds. The program prioritizes culturally safe healthcare access, supporting Métis individuals in navigating systemic barriers to medical services. By reducing financial obstacles and improving access to essential healthcare, EMTAP strengthens Métis health equity and ensures that all Citizens can receive timely, necessary medical care, including cancer treatment and screenings.

LAY SUMMARY

Reducing Financial Barriers to Care – EMTAP ensures low-income Métis Citizens can access essential medical services, including cancer treatment and screenings, without financial hardship. Coverage includes travel, accommodations, and meals, easing the burden of out-of-town medical appointments. **Improving Access to Specialized Healthcare** – Many Métis individuals live in rural or remote areas, where specialized care—such as oncology, diagnostics, and surgical procedures—is unavailable locally. EMTAP helps bridge this gap by supporting travel to necessary medical services. **Prioritizing Culturally Safe Healthcare** – The program recognizes the unique health challenges Métis Citizens face, ensuring they can access culturally informed care while navigating systemic barriers in the healthcare system.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Reaching front-line care teams and enhancing their capacity to support culturally safer care for First Nations, Métis and Inuit in cancer care. - Poster #247

Presenter: Krista Marsden, Cancer Care Alberta

Other Authors: June Kima, Amanda Andrew, Debora Allatt

ABSTRACT_ID#3003

ABSTRACT

Since beginning development of Walking Together to Strengthen Indigenous Cancer Care in Alberta: Our North Star to Action, published in 2023, Cancer Care Alberta has recognized cultural transformation is key to making a difference for Indigenous people. The goals of the Action Plan are: That organizational change is supported in cancer care and collaborative action with Indigenous communities and organizational partners is taken such that Indigenous people in Alberta: Are diagnosed with cancer at the earliest stage. Feel safer and welcome at cancer centres. Experience high satisfaction with their cancer care. Complete cancer treatment and have survival rates consistent with those of non-Indigenous people. Staff education, opportunities for learning and reflection are key change agents. Through a staff survey in spring 2024, it was confirmed that efforts to reach Cancer Care staff were not working. More than 50% of respondents reported they did not use pertinent web pages, did not share the Indigenous Cancer Health resources, were not comfortable with arranging supports for patients and clearly indicated their interest in receiving education. In a system with constrained resources, providing support to staff is challenging. Previous rounds, lunch and learns, and bulletins hadn't served the front-line needs. The Indigenous Cancer Care team, working with site managers, conceived Indigenous Cancer Care micro-learnings, specially designed for team huddles and meetings to impart critical information in 10 – 15 minutes. This presentation will describe the concept of micro-learnings, the subjects, the process, and the early response.

LAY SUMMARY

Improving cancer care outcomes of Indigenous people requires deep organizational change and a commitment to culturally safe practices. Micro-learnings can meet staff where they're at, making education more digestible, practical, and integrated into routine workflows, while addressing the expressed desire for more learning opportunities.

First Nations, Inuit and Métis Cancer Research

Monday, November 3, 2025

Building Trust and Nurturing Relationships with Indigenous Community Partners Integral to the Arthur J.E. Child Comprehensive Cancer Centre's Care Environment - Poster #248

Presenter: Krista Marsden, Cancer Care Alberta

Other Authors: Warren Michael, Amy Melnick

ABSTRACT_ID#3028

ABSTRACT

“healing starts by aligning cultural protocols and traditions both in the environments we create ... and meaningful engagement we have with cancer care providers” (community partner). The Arthur J.E. Child Comprehensive Cancer Centre in Calgary, Alberta which opened in October 2024, provides care to First Nations, Métis, Inuit, and all peoples in the southern half of the province. Colonial history and systemic racism have created a trust gap that impacts how Indigenous peoples in Canada and Alberta access and benefit from cancer care. Site planners were late recognizing the importance of engaging Indigenous partners, but meaningful progress was made cultivating relationships and working on the critical system change needed to make a difference in access, cultural safety, inclusion, and care. Working in humility and relationship, ensuring Indigenous partners set the pace and direction, Cancer Care Alberta, including site leaders, and First Nations of Treaty 7, Otipemisiwak Métis Nation within Alberta and Aboriginal Friendship Centres, community partners developed and implemented a shared understanding of Indigenous vision and priorities for the new centre. This includes an Indigenous Family Room, Family Garden, access to Traditional Ceremony, signage in Indigenous languages, access to information specifically for Indigenous patients and families, and staff orientation. Since opening, communities have honored the staff and patients with ceremony and ongoing partnership. In the spring of 2025, Cancer Care Alberta conducted a small-scale qualitative evaluation of the relationships. The lessons learned, which will help move work forward in a good way, will be shared.

LAY SUMMARY

Reconciliation requires culturally safer spaces and relationships. Working in meaningful partnership is an intentional act and proceeds at the pace of trust. Indigenous partner led priorities enhance cultural safety.

First Nations, Inuit and Métis Cancer Research

Sunday, November 2, 2025

Knowledge Mobilization: A Mixed Methods Indigenous & Western Science Community-Based Research Approach - Poster #249

Presenter: S. Trevor Baxter, AHS

Other Authors: Jessica Smith, Angeline Letendre, Brenda Roland

ABSTRACT_ID#2955

ABSTRACT

Title: Knowledge Mobilization: A Mixed Methods Indigenous & Western Science Community-Based Research Approach

Abstract: Métis Settlement communities in Alberta experience higher cancer rates and face challenges in prevention and screening. The IAHCA and EAMSS projects provided an Indigenous-led framework to support these communities, respecting Métis cultural contexts and promoting self-determination. The “Enhancing Alberta Métis Settlements Participation in Cancer Screening” (EAMSS) project builds on prior initiatives involving First Nations and Métis Settlement communities. While it introduces innovative strategies tailored specifically for Métis Settlements, this project represents a significant advancement as its processes, theoretical framework, and outcomes have collectively evolved into a form of innovation. The four key concepts of the EAMSS are as follows: 1. Engage Métis Settlements to identify their specific needs related to cancer screening participation. 2. Examine health inequities associated with screening services and their delivery. 3. Pilot new or adapted screening innovations and services that are grounded in culturally appropriate and safe practice environments, such as providing suitable peer support and qualified service providers. 4. Evaluate the project’s processes and outcomes, including recommendations that contribute to an Alberta Métis Settlement Knowledge Mobilization cancer prevention and screening strategy. The project created an Indigenous model for Métis communities, emphasizing a distinction-based approach. It involved collaboration with Elders and Knowledge Holders to address specific challenges in cancer screening and prevention. The outcome enhanced processes by integrating Indigenous Ways of Knowing with Western scientific methods.

LAY SUMMARY

1. Métis Settlement communities in Alberta experience higher cancer rates and face challenges in prevention and screening.
2. The “Enhancing Alberta Métis Settlements Participation in Cancer Screening” (EAMSS) project's theoretical framework, processes, and outcomes have collectively evolved into a form of innovation.
3. The project co-created an Indigenous model for Métis communities, emphasizing a distinction-based approach. It involved collaboration with Elders and Knowledge Holders to address specific challenges in cancer screening and prevention. The outcome enhanced processes by integrating Indigenous Ways of Knowing with Western scientific methods.

Genetics and Epigenetics

Monday, November 3, 2025

Multi-Omics Approach to Colorectal Cancer Non-Coding Driver mutations in an Isolated Population - Poster #250

Presenter: Hayley Alloway, Memorial University of Newfoundland

Other Authors: Khadija Rebbani, Guillaume Tricknot, Akinola Alafiatayo, Modeline Longjohn, Lousia Wiede, Daniel Loos, John Thoms, Touati Benoukraf

ABSTRACT_ID#3260

ABSTRACT

Newfoundland and Labrador (NL) provides a unique opportunity to study the biology of colorectal cancer (CRC) due to its genetically isolated population, unique environmental exposures, and one of the highest CRC incidence rates in the world. Along with the Marathon of Hope Cancer Centres Network, led by the Terry Fox Research Institute via the Atlantic Cancer Consortium, we have generated preliminary genomics and transcriptomics data on a local cohort of CRC patients. With this project, we aim to characterize non-coding pathogenic variants and mutations that disrupt gene regulation. Specifically, we focus on non-coding germline variants and somatic mutations located in regulatory elements such as promoters and enhancers. We performed in-house Chromatin Immunoprecipitation followed by sequencing (ChIP-Seq) with CRC tissue samples collected from local patients to target H3K27ac, a histone mark associated with active regulatory elements, including super-enhancers. By combining ChIP-Seq data with whole genome sequencing (WGS) and RNA sequencing (RNA-Seq), we provide a comprehensive view of the regulatory landscape in CRC, highlighting potential non-coding drivers of disease. To date, twenty-four patients from the cohort have undergone WGS (Illumina), RNA-Seq (Illumina) and H3K27ac ChIP-seq (Complete Genomics/Illumina). These preliminary data indicate that genomic mutations are linked to epigenetic variations, which are associated with changes in gene expression. Emerging evidence suggests that mutations in regulatory elements can either disrupt existing transcription factor binding sites or create novel binding sites, contributing to tumorigenesis. Our study integrates these findings with multi-omics data to identify population-specific genetic mutations and regulatory disruptions in NL's colorectal cancer patients.

LAY SUMMARY

Newfoundland and Labrador provides a unique opportunity to study Colorectal Cancer (CRC) Our in-house ChIP-seq of tissue allows for cost effective sequencing of local patients Multi-Omics allows for depth of analyses and facilitates identification of super enhancers of CRC.

Beyond Viral Mimicry: Transposable Element Expression Defines Prognostic Stress-Response Phenotypes in Bladder Cancer - Poster #251

Presenter: Andrew Garven, Queens University

Other Authors: Hamid Ghaedi, Amber Simpson, David Berman

ABSTRACT_ID#3154

ABSTRACT

Introduction: Transposable elements (TEs) are mobile genetic sequences derived from endogenous retroviruses, comprising ~45% of the human genome. In bladder cancer (BC), epigenetic instability leads to aberrant TE transcription. While prior studies suggest TE expression may enhance anti-tumor immunity via viral mimicry, its immunologic consequences in BC remain unclear. Given the clinical relevance of immunotherapy, we investigated the relationship between TE expression and immune response using transcriptional and immunohistochemical profiling. **Methods:** We analyzed TE and gene expression across muscle-invasive (TCGA-BLCA, n=412) and non-muscle invasive (UROMOL, n=535) BC cohorts. Unsupervised clustering of TE expression defined molecular subgroups evaluated for prognostic significance, differential expression, and pathway enrichment. Genes correlated with TE levels were assessed for pathway activation. Findings were validated by immunohistochemical staining of LINE-1 ORF1p in an independent cohort (n=371). **Results:** TE defined molecular subgroups was the strongest predictor of overall survival in MIBC, outperforming stage, AUA risk score, and molecular subtype. The TE-Resilient phenotype (high TE expression) was associated with favorable outcomes and activation of stress adaptation pathways. The TE-Exhausted phenotype (low TE expression) showed poor prognosis, inflammation, and T cell exhaustion. Despite elevated TE activity, no evidence of antiviral immune activation was found, indicating a disconnect from the viral mimicry hypothesis. **Conclusion:** This study establishes a novel TE-based classification in BC, identifying low TE expression as a marker of immune exhaustion and poor outcomes. TE expression may serve as a prognostic biomarker and inform future therapeutic strategies.

LAY SUMMARY

1. Transposable elements help predict bladder cancer outcomes The activity of transposable elements (TEs), genetic sequences derived from ancient viruses, can stratify bladder cancer into two distinct phenotypes with different clinical outcomes. Measuring TE expression identifies a high-risk group with significantly shorter survival, outperforming traditional clinical markers such as stage, grade, and molecular subtype.
2. TE expression does not trigger antiviral immune responses Despite previous assumptions, elevated TE expression in bladder cancer does not activate typical antiviral immune pathways. No meaningful increase in interferon signaling, viral recognition, or chemokine production was observed in tumors with high TE expression. This finding challenges the idea that TEs promote anti-tumor immunity through viral mimicry.
3. Low TE expression signals immune dysfunction and aggressive disease Bladder tumors with low TE expression show evidence of immune exhaustion, including T cell dysfunction and increased inflammatory signaling. This TE-Exhausted phenotype is associated with poorer outcomes, a more suppressive tumor environment, and reduced effectiveness of immune surveillance. TE expression levels may therefore reflect the functional state of the immune system in bladder cancer and serve as a biomarker for patient risk stratification.

Metabolomic Signatures of Glycosyltransferase Deficiency Reveal Sex-Specific Associations with Cancer Risk: Findings of the Canadian Longitudinal Study on Aging - Poster #252

Presenter: Ana Lucia Rivera Herrera, Université Laval

Other Authors: Michèle Rouleau, Mahukpe Narcisse Ulrich Singbo, Tania Cuppens, Julien Prunier, Arnaud Droit, David Simonyan, Chantal Guillemette

ABSTRACT_ID#2626

ABSTRACT

The glycosyltransferases UGT2B17 and UGT2B28 play a key role in the regulation of steroid hormone metabolism and activity, as well as xenobiotic inactivation. However, the genes encoding these enzymes are among the most frequently deleted of the human genome. The complete deficiency of UGT2B17 affects 9% of Caucasians to 70% of Asians, whereas for UGT2B28, 3% Caucasians and 10% of Africans are affected. A recent metabolomic profiling of UGT-deficient individuals has uncovered unexpectedly broad molecular alterations, extending beyond their canonical substrates. Understanding how these metabolic shifts influence cancer susceptibility may reveal shared mechanisms across malignancies. In this study, we investigated the association between 359 significantly altered metabolites and cancer incidence in 696 participants from the Canadian Longitudinal Study on Aging (CLSA), including 67 UGT-deficient and 627 UGT-proficient individuals. Our analysis revealed that UGT2B28-deficient males exhibited a significantly increased risk of cancer and showed an inverse association with an acylcholine metabolite known for its antioxidant activity. Notably, a bile acid glucuronide conjugate and a pregnenolone steroid sulfate were associated with cancer in UGT2B17-deficient men. By contrast, UGT2B28-deficient females showed a protective metabolomic profile, with several fatty acid metabolites inversely associated with cancer risk. These findings highlight sex-specific metabolomic consequences of common UGT gene deletions and suggest that these copy number variations may serve as biomarkers for cancer susceptibility. Further investigation into the functional roles of these metabolites may uncover novel pathways involved in cancer pathogenesis and prevention.

LAY SUMMARY

The genes encoding the UDP-glycosyltransferase enzymes (UGTs) UGT2B17 and UGT2B28 are often naturally absent from the human genome, meaning that some people are born without these genes and have a complete deficiency of UGT2B17 or UGT2B28 proteins. These UGTs are known to be important for the regulation of sex steroid activity in the body. Our recent investigation in 4731 Canadian individuals has revealed that UGT-deficient men and women display unique blood profiles of small molecules called metabolites, affecting many types of molecules beyond sex steroids. It prompted us to investigate how these metabolic shifts may influence cancer susceptibility in men and women. By comparing UGT-deficient and proficient individuals, we found that UGT2B28-deficient men exhibited an increased risk of cancer; some bile acids and steroids were associated with cancer in UGT2B17-deficient men; several fatty acids were associated with a reduced risk of cancer in UGT2B28-deficient females. These findings support that a complete deficiency of these genes affect cancer risk in a sex-specific manner and may serve as biomarkers for cancer susceptibility.

Genetics and Epigenetics

Sunday, November 2, 2025

Identification of novel functional non-coding variants in glioblastoma - Poster #253

Presenter: Amy Banks, University of Calgaru

Other Authors: Sorana Morrissy, Jennifer Chan, Ana Nikolic

ABSTRACT_ID#2603

ABSTRACT

Glioblastoma (GBM) is the most common malignant brain tumour identified in adults, with an estimated survival of approximately 15 months, despite aggressive treatment. Our understanding of this disease has expanded in the past decade, with an increased focus on the molecular biology of these tumours, highlighting the presence of inter- and intra-tumoural heterogeneity contributing to disease recurrence and treatment evasion. Single-nucleotide variants (SNVs) in regulatory elements have been shown to influence epigenetic mechanisms through transcription factor binding and enhancer activation, and play a role in certain cancers, like prostate and breast. The aim of this project is to expand our knowledge of GBM and to identify functional non-coding variants influencing disease progression. A tool known as Assay for Transposase-Accessible Chromatin with sequencing (ATAC-seq) is leveraged to highlight regions with regulatory function and variant calling software is used to identify SNVs in these regions. These variants are annotated by location in the genome, genes in closest proximity, presence of regulatory elements and transcription factor binding affinities to generate a predicted function and significance. We identify epigenetic loci enriched for mutations in GBM genomes, highlighting a possible interplay between genetic and epigenetic regulation in GBM progression.

LAY SUMMARY

Identification of mutations in non-coding DNA playing a role in glioblastoma tumour growth and recurrence. Generation of potential new drug targets for glioblastoma treatments and biomarkers for determining patient prognosis. Expanding our understanding of biological mechanisms hijacked in glioblastoma contributing to treatment evasion and tumour growth.

Genetics and Epigenetics

Monday, November 3, 2025

Epigenetic Combination Therapy in Xenografted Sporadic Microsatellite Instability-high Colorectal Cancer - Poster #254

Presenter: Anneke Korver, University of Calgary

Other Authors: Julian Chua, Oliver Bathe, Parham Minoo, Arshad Ayyaz

ABSTRACT_ID#2834

ABSTRACT

Human cancers frequently display drastic changes in the epigenetic landscape. Although select epigenetic-modulating agents demonstrated therapeutic potential in certain cancer types, the mechanistic basis by which epigenetic regulation influences tumor behavior and therapeutic response remains incompletely understood. I am addressing these questions by testing microsatellite instability-high (MSI-H) colorectal cancer (CRC) as a model system, wherein resistance to conventional therapies has been associated with epigenetic silencing of the mismatch repair gene MLH1, a key tumor suppressor. Patient-derived organoids (PDOs) from human CRC patient samples have been generated for in vivo and in vitro testing of novel treatment combinations that specifically target the epigenetic pathway through which 15-20% of CRC cases develop due to DNA methylation-induced gene silencing of key tumor-suppressor genes. Treatments combining traditional chemotherapy drugs and epigenetic modulating drugs have been tested on organoids in vitro with preliminary results showing that combined epigenetic and traditional treatments decrease the viability of PDOs. Further testing is being done to validate these results. PDOs have also been xenografted into mice, with the aim of testing these treatment options in vivo. This project thus aims to expand treatment options for CRC, potentially paving the way for personalized medicine approaches. Testing treatments that target epigenetic changes using patient-derived models of this specific subset of CRC will serve as a model in which to study epigenetic changes in cancer and add to the growing understanding of the role of the epigenetic landscape in cancer initiation and progression, as well as cancer response to current treatments.

LAY SUMMARY

Studying a subset of CRC with a development different from the traditional somatic gene mutation pathway adds to a growing understanding of the unique and important role that epigenetics play in cancer development and treatment. Use of organoids allows for the best possible recapitulation of the human tumor molecular makeup unique to patients with this subtype of CRC.

Genetics and Epigenetics

Sunday, November 2, 2025

Investigating the role of CHD4 in prostate cancer - Poster #255

Presenter: Lingwei (Stephanie) Han, Goodman Cancer Institute, McGill University

Other Authors: Catherine R. Dufour, Anthony Alfonso, Megan Katz, Annabelle Beaumier, Anna Dolgova, Vincent Giguère

ABSTRACT_ID#2511

ABSTRACT

Prostate cancer (PCa) progression is largely regulated through androgen acting on the androgen receptor (AR), leading to a global change in gene transcription. Mammalian target of rapamycin (mTOR) is another key factor in PCa progression and emerging studies revealed that mTOR directly regulates gene expression by associating with chromatin in the nucleus (nmTOR). However, the detailed mechanism on how nmTOR regulates gene expression with AR in PCa remains unclear. Recently our group uncovered a nmTOR-AR functional crosstalk in PCa cells where upon androgen stimulation, nmTOR associates with chromatin and activates an oncogenic metabolic gene program in an AR-dependent manner. Using a proteomic approach referred to as rapid immunoprecipitation mass spectrometry of endogenous protein (RIME), we previously identified the nucleosome remodeling and deacetylase (NuRD) complex as a partner of nmTOR-AR bound on chromatin in PCa cells. Through ChIP-seq, RNA-seq, and ATAC-seq approaches, our investigation reveals a dynamic transcriptional complex formed by CHD4, the catalytic subunit of NuRD, nmTOR and AR on the chromatin in a time-dependent manner. While CHD4's role in PCa's role remains to be investigated, our results revealed depletion of CHD4 triggers a widespread transcriptional rewiring in PCa cells, impacting a significant portion of androgen-response genes and neuroendocrine PCa markers. This underscores the novel function of CHD4 in coordinating with AR and mTOR to regulate gene expression and potentially drive PCa progression and therapeutic resistance.

LAY SUMMARY

- Prostate cancer (PCa) often grows because of signals from a hormone called androgen and its receptor androgen receptor (AR). Treatment often target AR but PCa progresses eventually develop resistance though AR gene mutation or deletion. Currently we don't fully understand mechanisms underlying PCa progression and how PCa can survive without AR.
- Our lab discovered another important factor in cell growth, mTOR, functions along AR in PCa. It's understudied role in the nucleus of cells potentially explains PCa's treatment resistance and mechanism of progression.
- We also discovered CHD4, a protein that controls DNA wrapping and expression, as a player within the AR-mTOR crosstalk. Our results suggest potential crucial role of CHD4 in regulating AR activity and PCa progression toward very aggressive PCa subtypes.

Genetics and Epigenetics

Sunday, November 2, 2025

Metastatic cells adapt their migration strategy in response to changes in nuclear stiffness - Poster #257

Presenter: Anastasia Roemer, University of Alberta

Other Authors: Raina Stewart, Noah Wood, Michael Hendzel

ABSTRACT_ID#3346

ABSTRACT

To metastasize, cancer cells must navigate confined spaces in the extracellular matrix (ECM) and undergo intravasation and extravasation. To facilitate this, they can either remodel the ECM or adjust their stiffness by altering the stiffness of the nucleus through changing the proportions of heterochromatin and euchromatin, as heterochromatin is stiffer than euchromatin. However, it is unclear whether increasing or decreasing nuclear stiffness promotes metastasis. Some studies show that metastatic cancer cells are softer than their non-metastatic counterparts. Conversely, other research indicates heterochromatin levels increase after confined cell migration. To investigate this discrepancy, we conducted a 2D scratch assay with a library of small molecules targeting epigenetic readers and writers. We used metastatic B16F10 cells, with non-metastatic B16F0 cells as our control. The top five drugs that increased migration were all histone deacetylase inhibitors. Of the top five that decreased migration, three were BRD2/4 inhibitors, one was a protein methyltransferase inhibitor, and one was an EZH2 inhibitor. Our initial screen supports the hypothesis that a softer chromatin state increases migration, and vice versa. To assess 3D migration, we embedded the cells in a synthetic ECM and observed their migration under treatment with the top drugs. The control cells behaved as they did in the 2D scratch assay. The metastatic cell line was also consistent for the drugs that increased migration. However, some drugs that decreased migration in 2D increased migration in 3D. Our findings suggest that metastatic cancer cells can adapt their migration strategies in response to changes in nuclear stiffness.

LAY SUMMARY

Cancer cells need to move through small spaces during metastasis, which is the movement of cancer cells to a new place in the body different from their original location. One way that they can make it easier for themselves is by changing their own stiffness. We have found that when cancer cells are moving across a flat surface, they are able to move faster when they have a softer state, than a stiffer state, which causes them to move slower. When cancer cells are moving through a 3D environment which is closer to what they do in the body, they are able to change how they move in order to keep moving through the space, regardless of whether they are stiffer or softer. This tells us that metastatic cancer cells are able to switch how they move, something that non-metastatic cells do not appear to be capable of.

Genetics and Epigenetics

Monday, November 3, 2025

The Role of the ING3 Epigenetic Regulator in Neural Stem Cell Differentiation - Poster #258

Presenter: Fatemeh Hashemi, University of Calgary

Other Authors: Li-Fang Chu, Ana Nikolic, Karl Riabowol

ABSTRACT_ID#2647

ABSTRACT

Background: INhibitor of Growth 3 (ING3) is an epigenetic reader and a targeting component of the TIP60 histone acetyltransferase (HAT) complex, which regulates gene expression during cell fate decisions. While TIP60's roles in pluripotency maintenance and neuronal differentiation are well characterized, ING3's function remains unclear. Its involvement in skeletal development and oocyte maturation suggests it may also influence neuronal lineage specification. Neuronal differentiation relies on precise epigenetic control, with TIP60 activating lineage-specific genes. Bivalent genes—bearing both activating (H3K4me3) and repressive (H3K27me3) histone marks—stay poised until triggered to activate. ING3 may direct TIP60 to H3K4me3-enriched chromatin, enabling neurogenic gene activation. **Methods:** We are generating ING3 knockout (KO) and overexpression (OE) models in human induced pluripotent stem cells (iPSCs) using CRISPR/Cas9 delivered via virus-like particles (VLPs) and lentiviral vectors, respectively. RNA sequencing (RNA-seq) will be used to identify affected genes and pathways. To isolate ING3-specific effects from broader TIP60 disruption, we will also target ING3's PHD domain, which recognizes H3K4me3 to help target the TIP60/NuA4 complex. **Preliminary Data and Hypothesis:** Initial findings show that ING3 is expressed in neural progenitor cells (NPCs) but not in undifferentiated iPSCs, suggesting a role in early neural differentiation. This aligns with embryonic lethality in ING3 KO mice at E10.5. ING3 KO may also reduce stress-induced differentiation. We hypothesize that ING3 helps recruit TIP60 to H3K4me3-marked chromatin to activate neurogenic genes. **Conclusion:** This study aims to define ING3's role in neurogenesis and its broader impact on epigenetic regulation in neural development and disease.

LAY SUMMARY

Think of cells as rooms with pianos inside. These pianos represent genes, and the keys are the individual genes that create the melodies of life. Each type of cell, like a brain or skin cell, plays its own unique tune by pressing different combinations of keys, or activating different genes. Just as a pianist selects keys to compose a specific song, cells activate certain genes to become specialized types. For example, brain cells produce a different melody than skin cells because they activate a different gene pattern. Now imagine ING3 as an assistant conductor in this orchestra of cells. ING3 helps guide which genes should be turned on or off—directing the pianist's hands to play the right tune. It works closely with another protein, Tip60, the main conductor, to ensure everything stays in harmony during cell development, especially when forming brain cells. We are investigating what happens when ING3 is removed. Can the brain cell still play its correct melody? If ING3 is essential, losing it could lead to disorganized or “off-key” cellular music, potentially causing brain cells to malfunction. This research may reveal new treatments for brain disorders like Alzheimer's and bring hope to affected individuals and their families.

Genetics and Epigenetics

Sunday, November 2, 2025

Blood-based epigenetic instability linked to cancer, aging, and cardiovascular disease - Poster #259

Presenter: Salman Basrai, Ontario Institute for Cancer Research

Other Authors: Ido Nofech-Mozes, Rajesh Detroja, Fernando Scolari, Mehran Bakhtiari, Andrea Arruda, Tracy Murphy, Scott Bratman, Steven Chan, Mark Minden, Jae-Sook Ahn, Dennis Kim, Robert Kridel, Filio Billia, Sagi Abelson

ABSTRACT_ID#2624

ABSTRACT

Rationale: The abundance, dynamics, and context-dependent heterogeneity of DNA methylation—where a pattern considered abnormal in one cell type may be normal in another—complicate the identification of early methylation changes that drive or signal disease development. This complexity can obscure early markers of increased cancer risk, making it challenging to detect and intervene in carcinogenic processes at their inception. **Approach:** We studied DNA methylation instability by conducting genome-wide analyses to identify CpG sites with highly consistent methylation profiles in healthy blood cells. We examined alterations at these stable loci in a wide variety of hematological cancer cohorts. **Results:** Our findings reveal that hypermethylation of these CpG sites is ubiquitous across myeloid and lymphoid malignancies, correlates with clonal burden and mutation frequency dynamics throughout treatment, and exhibits unique patterns that reflect the cell of origin. In non-cancer cohorts, we observed that methylation levels at epigenetically stable loci become increasingly variable with age—a shift also linked to higher cardiovascular disease risk and lower survival rates. Additionally, our results indicate that highly stable CpGs are enriched in the promoters of age-related genes, frequently overlapping key transcription factor binding sites, suggesting that their disruption contributes to the altered gene expression observed with aging. **Impact:** This study highlights DNA methylation instability as a blood-based biomarker for both cancer and cardiovascular disease and uncovers a mechanistic link between methylation dynamics and the expansion of maladaptive hematopoietic clones. These results have significant implications for early cancer detection and estimating disease risk in healthy individuals.

LAY SUMMARY

DNA methylation – chemical modifications of the nucleotides in DNA – represents a valuable layer of information that can be used to study diseases. However, methylation profiles are highly variable across different cell types and they can evolve over time, making it difficult to identify informative markers of increased cancer risk that are broadly applicable. We studied genome-wide DNA methylation profiles in thousands of blood samples derived from healthy individuals and patients with a wide variety of blood cancers. We identified genomic loci which exhibit consistently low methylation levels in healthy individuals and become abnormally hypermethylated in cancer, aging, and cardiovascular disease. Our findings reveal that this phenomenon of DNA methylation instability is correlated with disease progress in leukemia patients and can be used to track changes in the size of cancer cell populations, similar to somatic mutations. Additionally, DNA methylation instability increases gradually even in healthy aging individuals and is a risk factor for various cardiovascular complications. These results have significant implications for early cancer detection, monitoring residual disease in patients undergoing treatment, and estimating disease risk in healthy individuals.

Genetics and Epigenetics

Monday, November 3, 2025

Intrapatent Genomic Heterogeneity and Evolution of De Novo Metastatic Prostate Cancer - Poster #260

Presenter: Kamal Al Najem Azzam, Vancouver Prostate Centre

Other Authors: Andrew J. Murtha, Edmond M. Kwan, Melissa Koudjanian, Emily Fung, Cecily Q. Bernales, Gráinne Donnellan, Karan Parekh, Wilson Tu, Matti Annala, Peter C. Black, Corinne Maurice-Dror, Gillian Vandekerckhove, Gang Wang, Kim N. Chi, Alexander W. Wyatt

ABSTRACT_ID#2755

ABSTRACT

Prostate cancer (PCa) is initially “castration sensitive” (CS) and is responsive to systemic androgen blockade via hormone therapy, but during treatment can evolve into lethal castration-resistant PCa (CRPC). At first diagnosis, 5-10% of PCa has already spread to other organs and is known as de novo metastatic castration-sensitive PCa (dnmCSPC). These cancers are aggressive, can rapidly progress to CRPC, and represent ~50% of PCa-related deaths. Genomic heterogeneity is often present within and between the prostate and metastases, but its extent in large dnmCSPC populations is unclear. Here, we used patient-matched CS and CRPC samples to identify genomic changes associated with disease progression. Patients were retrospectively identified from a BC Cancer biobank and corresponding tumour tissue and blood plasma samples were subjected to targeted DNA sequencing to derive somatic alterations. 114 patients provided tumor-positive plasma (n=112) or tissue (n=194). 47 patients had both CSPC and CRPC samples, and 78 had paired CSPC diagnostic biopsy samples. In paired CSPC biopsies, we detected 54 heterogeneous mutations, most commonly within PTEN (n=9). 27 and 72 mutations were identified as exclusive to either patient-matched CS or CRPC samples, respectively. CRPC-emergent mutations were frequent in AR (n=18), RB1 (n=8), and TP53 (n=8). Common copy number alterations during the CS-CRPC transition included AR gains (n=15), NKX3-1 loss (n=10), and PTEN loss (n=8). Genomic alterations detected during the CS-CRPC transition emphasize the role of intrapatent heterogeneity in disease progression and resistance, providing potential biomarkers for detecting aggressive subclones and guiding treatment strategies.

LAY SUMMARY

38% of mutations detected in diagnostic biopsies were spatially heterogeneous, highlighting substantial intratumoral diversity at initial disease presentation. 43% of mutations were CRPC-emergent, supporting strong treatment-associated clonal evolution in response to ADT and other systemic treatments.

Genetics and Epigenetics

Sunday, November 2, 2025

Interplay between DOT1L and the Mediator complex regulates glioblastoma stem cell growth - Poster #261

Presenter: Samir Assaf, University of Calgary

Other Authors: Danielle Bozek, Kyle Heemskerk, Amy Banks, Graham MacLeod, Ravinder Bahia, Emilie Cutts, Michael Johnston, Orsolya Cseh, Stephane Angers, Ana Nikolic, Marco Gallo, H. Artee Luchman, Samuel Weiss

ABSTRACT_ID#3044

ABSTRACT

Epigenetic mechanisms that govern normal stem cell fate are dysregulated in glioblastoma stem cells (GSCs). Unraveling the complexity and plasticity of these mechanisms provides an opportunity to harness epigenetics as a tool to reprogram GSC fate and modulate sensitivity to therapeutics. Here, we demonstrate that the histone methyltransferase Disruptor of Telomeric Silencing 1-Like (DOT1L) is a key regulator of GSC stemness and growth through modulation of chromatin accessibility at enhancer elements, notably the cell cycle gene *CyclinD2*. To unravel the underlying epigenetic and transcriptional factors linked to DOT1L regulation in GSCs, we performed a chemogenomic genome-wide dropout screen in the context of DOT1L inhibition. Interestingly, the top hits from this screen were the MED23 and MED24 subunits from the Mediator transcriptional complex. Further investigation identified MED23 as the main driver of GSC growth arrest following DOT1L inhibition by repressing *CyclinD2*, whereas MED24 solely stabilized MED23 incorporation into the Mediator complex tail module. ATAC-seq performed on MED23-knockout GSCs in the context of DOT1L inhibition revealed that MED23 deficiency resists the chromatin accessibility changes induced by the loss of DOT1L epigenetic activity at enhancer elements. Consequently, this enables MED23-knockout GSCs to rescue their growth following DOT1L inhibition by restoring expression of the cell cycle regulator *CyclinD2*. These findings provide critical mechanistic insights into the cooperation between DOT1L and the Mediator complex to regulate GSC growth. Thus, targeting DOT1L in glioblastoma tumors expressing high levels of *CyclinD2* may prove to be beneficial.

LAY SUMMARY

Inhibition of the epigenetic regulator DOT1L promotes glioblastoma stem cell differentiation and growth arrest by restricting chromatin accessibility at enhancer elements corresponding to stemness and cell cycle genes, notably *CyclinD2*. MED23 deficiency resists the chromatin accessibility changes induced by DOT1L inhibition. MED23 is essential for glioblastoma stem cell growth arrest following DOT1L inhibition.

Genetics and Epigenetics

Monday, November 3, 2025

Unravelling the Complicated Epigenetics of Liver Cancer: FocusING on a multi- functional tumor suppressor and epigenetic reader - Poster #262

Presenter: Katayoun Heshmatzad, University of Calgary

Other Authors: mahbod djamshidi, Karl Riabowol

ABSTRACT_ID#2467

ABSTRACT

Liver cancer is the 4th leading cause of cancer death worldwide and the overall survival rate of male and female hepatocellular carcinoma (HCC) patients remains low at ~19%. Epigenetic readers are frequently reported to be dysregulated in HCC leading to poor prognosis and lower disease-free survival. Epigenetic readers are required for precise targeting of epigenetic modifier complexes. Therefore, investigating their mechanism is necessary to fully understand the epigenetic profile and regulation in cancer. The INhibitor of Growth (ING) family of 5 members (ING1-5) encode Plant homeodomain (PHD) containing proteins that are involved in targeting trimethylated histone 3 lysine 4 (H3K4me3) and act as both regulators of chromatin conformation and type-II tumor suppressors (TSGs). ING5, by frequently being recruited by MOZ/MORF histone acetyltransferase (HAT) complex, has been shown to be rarely mutated, but frequently down-regulated or mislocalized in multiple cancers. In vitro experiments have shown that ING5 has diverse functions involved in stemness, cell cycle, and metabolism regulation. In vivo Observations on our ING5 knockout mice colony indicate higher frequency of liver enlargement and pre-malignancy at middle age (~6 months). Utilizing the transcriptome and proteome profile of genetically engineered human liver cancer HepG2 cell line, we will investigate the function and downstream signaling mechanism of differentially expressed genes that might be enriched in cell migration, cell cycle and epigenetic regulation.

LAY SUMMARY

Liver cancer is one of the cancers highly influenced by environmental factors, genetics, and epigenetics. Despite the emergence of effective and systematic treatments, it remains one of the most lethal cancers worldwide. Previous studies have revealed the role of epigenetics as one of the key molecular mechanisms shared by these processes. Our lab discovered a group of genes known as the Inhibitor of Growth (ING) family, which consists of five members and has diverse functions, including roles in differentiation, cell cycle regulation, and apoptosis. We were among the pioneers in generating ING5 knockout (KO) mice. We observed several significant phenotypes, one of which was liver enlargement. Given the role of ING5 as a tumor suppressor and regulator of the cell cycle, and considering the involvement of epigenetics, we focused for the first time on the role of ING5 in hepatocarcinogenesis.

Genetics and Epigenetics

Monday, November 3, 2025

Segregating variants without parental data: progress in the validation of Parent-of-Origin-Aware genomic analysis in hereditary cancer - Poster #264

Presenter: Lilian Cordova, The University of British Columbia

Other Authors: Vahid Akbari, Tiffany Leung, Katherine Dixon, Kieran O'Neill, Alexandra Roston, Eugene Cheung, Chuyi Zheng, Alshanee Sharma, Yaoqing Shen, Janine Senz, Yanni Wang, Daniel Chan, Alexandra Fok, Carol Cremin, Jennifer Nuk, Angela Bedard, Melanie O'Loughlin

ABSTRACT_ID#3247

ABSTRACT

Background: Determining the parent-of-origin (PofO) of a variant is valuable in hereditary cancer, as it informs risk management, variant classification, and identification of at-risk relatives. However, assigning PofO without parental data has long been considered unattainable. To address this, we developed Parent-of-Origin-Aware Genomic Analysis (POAga), which combines chromosome-scale haplotyping with DNA methylation at imprinted regions to accurately assign PofO without external data. **Methods:** To validate POAga, we applied it to blood samples from individuals with pathogenic variants (PVs) in hereditary cancer genes and compared predicted PofO to known segregation to assess concordance and identify limitations. Parental segregation had been previously established for most individuals, while in three prospective cases, additional testing in relatives was performed to confirm POAga's predictions. All samples underwent Strand-seq and long-read sequencing under an REB-approved protocol. **Results:** We analyzed 255 PVs from 250 individuals across the following genes: BRCA2 (n=42), BRCA1 (n=39), MLH1 (n=27), MSH2 (n=29), MSH6 (n=24), SDHD (n=22), PMS2 (n=19), PALB2 (n=14), ATM (n=13), CDH1 (n=9), TP53 (n=4), CHEK2 (n=3), EPCAM (n=2), SDHAF2 (n=2), MUTYH (n=2), and one case each of CDKN2A, POT1, RAD51D, and SDHC. PofO was successfully assigned in 221 PVs, with a concordance rate of 98.6% (218/221). PofO could not be determined in 34 PVs (13.3%), primarily due to polymorphic methylation or regions of homozygosity that limited phasing. **Conclusion:** These findings support the clinical-grade accuracy of POAga for assigning PofO from blood-derived DNA and highlight its potential as a transformative tool in clinical genetics, particularly when parental samples are unavailable.

LAY SUMMARY

We are validating a new method called Parent-of-Origin-Aware Genomic Analysis (POAga). It combines long-read sequencing, DNA methylation at imprinted regions, and chromosome-scale phasing using Strand-seq. What makes this approach innovative is its ability to determine whether a genetic variant was inherited from the mother or the father, even when no parental DNA or relative testing is available. This has been largely unattainable with existing tools. Knowing the parent of origin is especially important in hereditary cancer. For some genes, cancer risk depends on whether the mutation came from the mother or the father. Additionally, this information also supports decisions around risk management, variant classification, and family testing. In many cases, parental samples are not available, and POAga offers a direct way to fill this gap. Our results show that POAga correctly identified the parent of origin in 98.6% of cases across a wide range of cancer-related genes. Only 13.3% of variants could not be assigned a parental origin, mainly due to technical factors like regions of homozygosity (regions inherited identically from both parents) or variability in methylation at imprinted regions.

Genetics and Epigenetics

Sunday, November 2, 2025

Large-scale genetic variant interaction analysis using a computational framework in colorectal cancer - Poster #265

Presenter: Aaron Curtis, Memorial University of Newfoundland

Other Authors: Yajun Yu, Sevtap Savas

ABSTRACT_ID#2860

ABSTRACT

Background: The Atlantic Cancer Consortium (ACC) is a part of the national Marathon of Hope Cancer Centres Network. One of the ACC's projects focuses on multi-variant interactions that may explain tumor features and prognosis in colorectal cancer. **Objectives:** Our objective was to establish a computational workflow to examine genetic variant interactions and apply it to study tumor location (colon/rectum) in colorectal cancer. **Methods:** As the ACC patient genetic data is currently being collected, we aimed to develop the framework using data from 505 colorectal cancer patients recruited to the Newfoundland Colorectal Cancer Registry. The dataset was first screened for interactions using the PLINK fast-epistasis boost method and top results were then examined using logistic regression analysis in R. PUBMED and dbSNP were utilized to further explore select variants. **Results:** 11,098,213,891,086 two-variant interactions were examined from a dataset of 4,711,309 variants. It took approximately two days for computation to complete, with the PLINK fast-epistasis boost function taking most of this time. While no association was significant, a list of candidate interactions and variants were identified, including several non-coding variants of genes that were previously associated with colorectal or other cancers (SORCS2, RNF150, and CD46). **Conclusions:** We applied a practical approach to conduct multi-variant interaction analyses in large-scale genomic data to examine interactions associated with tumor location in a cohort of colorectal cancer patients. We will next explore interactions associated with other clinical features and apply this methodology to data from the ACC.

LAY SUMMARY

Multi-variant interactions are understudied in relation to colorectal cancer tumor features. Computational methods, such as PLINK fast-epistasis boost, can help examine large-scale interactions in genomic datasets. We applied a computational framework, making use of PLINK fast-epistasis boost, to examine interactions of genetic variants associated with tumor location in colorectal cancer. Our work can inspire other groups to study and discover novel interactions in colorectal and other cancers.

Genetics and Epigenetics

Monday, November 3, 2025

Genomic dissection of tumor suppressor gene inactivation from cell-free DNA of 2,212 patients with metastatic prostate cancer - Poster #266

Presenter: Yi Jou (Ruby) Liao, Vancouver Prostate Centre

Other Authors: Sofie H. Tolmeijer, Catherine K. Wang, Tiffany T.Y. Xie, Cameron Herberts, Andrew J. Murtha, Jack V.W. Bacon, Matti Annala, Kim N. Chi, Corinne Maurice-Dror, Alexander W. Wyatt

ABSTRACT_ID#2606

ABSTRACT

Metastatic prostate cancer (mPCa) is a lethal disease marked by defects in key tumor suppressor genes (TSGs; TP53, PTEN, RB1), previously shown to have prognostic relevance. However, the frequency and mechanisms of TSG inactivation remain poorly defined, limiting their potential as therapeutic biomarkers. We analyzed blood samples from 2,212 mPCa patients, primarily from a British Columbia-wide blood biobank, to characterize TSG alterations at allelic resolution. Cell-free and matched white blood cell DNA underwent targeted sequencing, including exonic and intronic coverage of TSGs. We identified mutations, structural variants (SVs), and copy number variants, and estimated circulating tumor DNA (ctDNA) fraction using orthogonal approaches based on mutation allele fraction and copy number profiles. We developed a new computational tool to integrate intragenic alterations and infer allelic status per gene, with “null” designation when all alleles were disrupted and “altered” when any allele was disrupted. Among 1,560 patients with detected ctDNA, somatic TSG mutations were present in 783 (50.2%) patients, SVs in 380 (24.4%), and biallelic copy losses in 120 (7.7%). TP53 was altered in 787 (50.4%) patients and null in 489 patients (31.3%), PTEN altered in 530 (34.0%) and null in 407 (26.1%), and RB1 altered in 283 (18.1%) and null in 230 (14.7%). TP53 alterations were predominantly missense mutations; PTEN and RB1 alterations were often translocations and deletions. Concurrent alterations in all three TSGs occurred in 114 (7.3%) patients, exactly two in 361 (23.1%), and only one in 536 (34.4%). These findings highlight distinct TSG inactivation patterns in aggressive prostate cancers.

LAY SUMMARY

65% of patients with metastatic prostate cancer have at least one genomic alteration in any of the tumor suppressor genes TP53, PTEN, and RB1. 7% of patients with metastatic prostate cancer exhibit co-occurring gene alterations in all three tumor suppressor genes TP53, PTEN, and RB1. TP53, PTEN, and RB1 exhibit diverse mechanisms of gene inactivation in metastatic prostate cancers.

Health Disparities and Equity

Sunday, November 2, 2025

Patient-focused evAluationN Of dispaRities AMong pArticipants in PRecision Oncology studies and Clinical Trials (PANORAMA-PRO) - Poster #267

Presenter: Abdulazeez Salawu, University Health Network

Other Authors: Lindsay Carlsson, Sean Rampersad, Andrew Botros, Daniela Figuera, Paul Reyes, Thaksha Balanathan, Gagan Sran

ABSTRACT_ID#2976

ABSTRACT

Background: Equitable access to clinical trials and precision oncology is crucial for ensuring all cancer patients benefit from advances in care. However, disparities in trial enrollment and genomic research participation persist, especially among historically under-represented racial, ethnic, and socioeconomic groups. In Canada, limited routine collection of sociodemographic data hinders efforts to measure and address these disparities. **Methods:** PANORAMA-PRO is a prospective, mixed-methods study aimed at identifying and addressing sociodemographic disparities in enrollment across clinical trials and precision oncology studies at Princess Margaret Cancer Centre, Toronto. In Part 1, 60 patients from a research clinic completed a validated sociodemographic questionnaire; 20 also participated in semi-structured interviews on attitudes on sociodemographic data collection. In Part 2 (starting June 2025), an adapted questionnaire is being implemented across multiple precision oncology and clinical trial cohorts, with plans to enroll 500 patients and conduct up to 40 interviews exploring attitudes and perceptions of barriers to enrollment. A patient advisory panel will support the development of equity-focused strategies. **Results:** Among 60 participants in Part 1, 42(70%) self-identified as White, 11(18%) as Asian, 3(5%) as Black, and 4(7%) as mixed-race. Only 6(10%) were aged ≥70; 50(83%) were Canadian-born and 53(88%) primarily English-speaking. While participants supported data collection, concerns included privacy issues, unclear purpose and potential misuse of data. **Conclusion:** These pilot data highlight demographic imbalances and diverse patient views on data use. PANORAMA-PRO will inform inclusive strategies to improve equitable enrollment in precision oncology and clinical trials, in collaboration with an ongoing virtual clinical trials navigation pathway.

LAY SUMMARY

Cancer research isn't equally accessible to everyone. Many clinical trials and precision oncology studies don't fully include people from racialized, immigrant, or lower-income communities, partly because we don't routinely collect enough background information (like race or income) in Canada. Patients generally support data collection but have concerns. In a small pilot study, most patients agreed with collecting sociodemographic data, but they were also worried about how the information would be used, particularly regarding privacy and potential discrimination. There are clear gaps in who is participating. In the pilot, 70% of participants were White, and very few were over 70 or from non-English-speaking or immigrant backgrounds. This highlights the need to better reach and support diverse patients. These early findings will help guide future efforts to make cancer research more inclusive, including through partnerships with tools like VIRTUOSO, a virtual clinical trial support program.

Health Disparities and Equity

Monday, November 3, 2025

COVID-19 Impacts on the Breast Cancer Care Pathway among Systemically Marginalized Communities in Ontario - Poster #268

Presenter: Ielaf Khalil, Women's College Hospital

Other Authors: Aisha Lofters, Priya Premranjith, Anastasia Gayowsky, Andrea Covelli, Juliet Daniel

ABSTRACT_ID#2623

ABSTRACT

Purpose: Healthcare system pauses occurred worldwide due to COVID-19, and may have worsened pre-existing disparities in breast cancer. In this population-based, retrospective cohort study, we investigated indicators of breast cancer care (i.e., adherence to screening guidelines, early vs. late-stage diagnosis, and mastectomy vs. breast-conserving surgery) before and after COVID-19 lockdowns in Ontario, with an emphasis on immigrant women. Methods: We had three binary outcomes and corresponding cohorts, and each outcome/cohort was ascertained relative to two time periods: April 1, 2018-March 31, 2020 ("pre-pandemic") and April 1, 2020-March 31, 2022 ("pandemic"): i) up to date on screening, ii) early vs late stage of breast cancer diagnosis, and iii) mastectomy vs breast-conserving surgery at any time after diagnosis for women who were diagnosed at stages I-III during each two-year time period. We conducted descriptive analyses, and used logistic regression, (unadjusted and adjusted), to determine odds ratios for our dichotomous outcomes. Results: Breast screening rates dropped from 59.4% to 51.0%, and the number of women diagnosed dropped from 18,821 to 14,269, in the pre-pandemic vs pandemic period. In multivariable analyses, screening significantly dropped (AOR=0.69 [95% CI (0.69-0.69)]), there was no significant difference for diagnostic stage (AOR =0.99 [95% CI (0.92-1.05)]), and the use of mastectomy vs breast-conserving surgery was higher in the pandemic period (AOR = 1.14 [95% CI (1.08-1.20)]). Women from the Caribbean had lower odds of early-stage diagnosis in the pre-pandemic period despite a screening advantage. Conclusion: Future work should further explore the reasons for these findings and potential system-level solutions.

LAY SUMMARY

1. Breast cancer screening rates in Ontario dropped, and use of mastectomy over lumpectomy rose, during the COVID-19 pandemic.
2. Women from the Caribbean were more likely to be diagnosed with breast cancer at a late stage, even though their screening rates were higher.
3. Women living in lower-income neighbourhoods were less likely to be screened, more likely to be diagnosed at stage III or IV, and more likely to have mastectomy than breast-conserving surgery than those in the highest income neighbourhoods

Health Disparities and Equity

Sunday, November 2, 2025

Improving Digital Cancer Care for Older Black Adults: a Qualitative Study - Poster #269

Presenter: Ambreen Sayani, Women's College Hospital

Other Authors: Paul Wankah, Shivani Chandra, Aisha Lofters

ABSTRACT_ID#2716

ABSTRACT

Objective: Although older Black adults are known to face significant systemic and socioeconomic barriers to healthcare, little is known about their access to and use of digital cancer care. This study aims to better understand the digital cancer care experience of older Black adults, their caregivers, and health care providers to identify strategies that can better support patient-centered digital cancer care. **Methods:** This qualitative study, conducted across 10 Canadian provinces, involved six focus groups with 55 participants. Data were analyzed using a theory-informed thematic analysis, guided by the Synergies of Oppression framework and the Patient-Centered Care model. **Results:** Participant experiences were categorized as barriers to digital access and participation; evolving caregiver roles; autonomy and purpose-based preferences for care delivery; digital accessibility; and effective communication through digital platforms. Key barriers included low digital literacy, unreliable internet in rural areas, language barriers, and concerns about privacy and caregiver involvement. Key facilitators included support from community-based groups, active caregiver involvement, the convenience of receiving care at home, and user-friendly digital technologies. **Conclusion:** Findings underscore the need for a multipronged approach that addresses digital and structural barriers while leveraging community assets to enhance access and usage. Policy and organizational interventions, such as expanded internet infrastructure or culturally sensitive training, are essential to mitigate inequities in digital cancer care. Findings have implications for advancing health equity and improving digital care for other structurally marginalized groups. Tailoring digital cancer care to the unique needs of older Black adults can contribute to more inclusive, responsive healthcare systems.

LAY SUMMARY

Older Black adults have varied experiences with digital cancer care and frequently encounter barriers such as unreliable internet in rural areas, limited digital literacy, and language differences that hinder their ability to effectively engage with virtual health services. Health services organizations should formally recognize caregivers as integral members of the care team by providing them with appropriate training and support, while also ensuring that patient privacy and autonomy are respected. In parallel, partnering with trusted community organizations can enhance outreach to socially isolated patients, improve access to digital tools, and foster greater trust in virtual health services. Health system leaders must enact policies and practices that support a comprehensive, multipronged approach to advancing equitable digital cancer care. This includes expanding stable internet access in rural and underserved areas, integrating interpretation services and culturally appropriate communication strategies—especially for older Black adults who are more fluent in African or Caribbean languages—and delivering targeted digital literacy training for both older adults and their caregivers to reduce access inequities.

Health Disparities and Equity

Monday, November 3, 2025

From principles to practice: Advancing health equity in a national cooperative oncology trial group - Poster #270

Presenter: Anna Johnson, Canadian Cancer Trials Group

Other Authors: Alison Urton, Jessica Sleeth, Dora Nomikos, Pamela Brown-Walker, Patti O'Brien, Susannah Groen, Scott Wilkes, Stephanie Jesshope, Max Sherry, Barbara Graham, Patricia Tang, Nathalie Daaboul, Rohini Venkatesh, Lesley Seymour, Janet Dancey

ABSTRACT_ID#2372

ABSTRACT

The Canadian Cancer Trials Group (CCTG) is committed to advancing health equity, diversity, and improved clinical trial access for all patient populations. In 2021, CCTG launched an Equity, Diversity, Inclusivity, Indigenization, and Accessibility (EDIIA) Action Plan to embed EDIIA principles into its strategic priorities, research agenda, governance, organizational structure, and network operations. As part of this initiative, CCTG developed an EDIIA Policy; integrated EDIIA into the Terms of Reference for Scientific, Oversight, and Support Committees; provided network-wide EDIIA training; and held a trial-focused EDI Workshop. Updates were also made to data dictionaries, generic trial protocols, informed consent forms, and standard operating procedures with an equity lens. Three EDIIA Working Groups, engaging over 50 members from across Canada, contributed recommendations on methods for embedding EDIIA into research priorities, trial design and conduct, and network practices to foster equity of access for patients. Each Working Group had at least one patient representative to ensure the patient voice was reflected in the recommendations. These recommendations will be used by CCTG leaders as they develop and conduct trials, and several recommendations are currently being piloted and monitored on two open CCTG trials. The implementation and success of these practices will be overseen by the new CCTG Health Equity Standing Committee which will also guide and shape CCTG's future EDIIA strategy and priorities. CCTG is a National Program of the Canadian Cancer Society (grant number: 707213).

LAY SUMMARY

To advance health equity, the Canadian Cancer Trials Group (CCTG) launched an Equity, Diversity, Inclusivity, Indigenization, and Accessibility (EDIIA) Action Plan to improve clinical trial access for all patient populations. Activities included developing an EDIIA Policy; providing network-wide EDIIA training; updating trial materials; launching three network-wide EDIIA Working Groups; and hosting a trial-focused EDI Workshop. These activities led to the creation of a new Health Equity Standing Committee which will guide and shape CCTG's future EDIIA strategies and priorities with a focus on fostering equity and access for patients to participate in CCTG trials.

Health Disparities and Equity

Sunday, November 2, 2025

Advancing Equity in Cancer Care Through Community-Engaged Health Equity Assessments (CEn-HEAs) - Poster #271

Presenter: Ambreen Sayani, Women's College Hospital

Other Authors: Alies Maybee, Members of Equity Mobilizing Partnerships in Community (EMPaCT)

ABSTRACT_ID#2952

ABSTRACT

Background Traditional Health Equity Assessments (HEAs) often overlook the insights of those most impacted by systemic inequities. The Community-Engaged Health Equity Assessment (CEn-HEA) framework, developed by Equity Mobilizing Partnerships in Community (EMPaCT), centers lived and living experiences to generate actionable recommendations addressing inequities at downstream, midstream, and upstream levels. Methods CEn-HEA follows a five-step participatory process: creating safe spaces, analyzing inequities, co-developing changes, making recommendations, and tracking accountability. Of 32 CEn-HEAs completed to date, nine were focused on cancer care. Community members and decision-makers engaged to identify inequities and generate actionable recommendations to improve equity. Results Thematic analysis of nine deidentified CEn-HEAs related to care across the cancer continuum (prevention and early detection = 3; treatment and care = 4; and 2 on survivorship) revealed five central equity concerns: (1) digital and linguistic barriers limiting access to care and information; (2) racialized stigma and mistrust in screening processes; (3) unmet psychosocial and follow-up needs; (4) advisory structures lacking cultural safety and meaningful influence; and (5) the need for ethical, community-led data governance to guide how identity and experience are represented and used. Recommendations emphasized reimagining outreach beyond digital platforms, integrating caregiver support into care planning, and embedding trauma-informed approaches to engagement and data practices across the cancer care system. Conclusion CEn-HEA is a scalable, participatory model for embedding equity into cancer systems. Grounded in trauma-informed and relational practices, this approach ensures actionable, equity-driven solutions grounded in the lived experiences of those most affected by health inequities.

LAY SUMMARY

Equity Mobilizing Partnerships in Community (EMPaCTs) Community-Engaged Health Equity Assessment (CEn-HEA) framework centers lived/living experience to identify and address health inequities. Thematic analysis across nine CEn-HEAs revealed barriers in access, trust, survivorship support, engagement, and data governance. CEn-HEA is a scalable model for advancing equity through community-driven, actionable recommendations across the cancer care continuum.

Health Disparities and Equity

Sunday, November 2, 2025

Making cancer research accessible: A Canadian study on direct-to-patient recruitment and lessons in equity

- Poster #273

Presenter: Arabella Helgason, University of British Columbia

Other Authors: Nimmy Sebastian, Dollina Dodani, Rachel Woima, Andrea Neilson, Aline Talhouk

ABSTRACT_ID#3350

ABSTRACT

Introduction: Equitable clinical trial recruitment is essential for generating findings that apply to diverse populations, which can be especially challenging when targeting specific groups. RESToRE, a multi-part study based in British Columbia (BC), aimed to identify postmenopausal, asymptomatic individuals at elevated risk for endometrial cancer (target population) from the general population. Methods: The RESToRE study used a 21-month, mixed-methods, direct-to-patient recruitment model to engage target individuals across BC, beyond clinical pathways. Methods included social media, REACH BC, posters, newspapers, and a recruitment agency. Strategies were iteratively adapted to improve equity and reach. Recruitment and cost-effectiveness were evaluated by method and population (total, target), including estimated labour costs. Results: Of 2446 leads, RESToRE recruited 713 participants, including 120 (16.8%) from the target population, who all had a BMI ≥ 34.9 . Average BMI was highest from the recruitment agency (38.8) and lowest from REACH BC (27.2). Among all participants reporting race/ethnicity, 90.4% identified as white, and Indigenous individuals were the second-most represented group (3.6%). REACH BC was the most ethnically diverse method (14.0%). Most participants were cisgender (99.0%), post-secondary educated (84.1%) and had a family physician (89.9%). Social media achieved the widest regional reach. The recruitment agency had the lowest cost per target participant (CAD 4.29), while newspaper ads were the least cost-effective (CAD 241.99). Conclusions: While the RESToRE study successfully reached its recruitment targets using a flexible, patient-centred approach, participant diversity remained limited. Findings highlight the need for further targeted equity-focused strategies to improve the representation of diverse populations in clinical trials.

LAY SUMMARY

Most participants in the RESToRE study were white, well-educated and had access to a family doctor, highlighting ongoing challenges in reaching diverse populations in clinical research. Recruiting directly from the community, through social media, posters, and online platforms, helped us reach people across British Columbia, but not all groups were equally represented. Some methods, like using a recruitment agency for targeted recruitment or free platforms like REACH BC, were much more cost-effective than traditional advertising, such as newspapers.

Health Disparities and Equity

Monday, November 3, 2025

Advancing Equity-Oriented Cancer Care through Clinical Nurse Specialist Innovation at BC Cancer – Vancouver - Poster #274

Presenter: Jagbir Kaur, BC Cancer

Other Authors: Leah Lambert, Emily Lonsdale, Tracey Rannie, Michael McKenzie, Scott Beck, Annette Browne, Debbie Lam

ABSTRACT_ID#3278

ABSTRACT

Background: Unfair disparities in care and outcomes persist across the cancer continuum, particularly among people with lived experiences of health and social inequities (PWLE-HSI). As such, the Equity-Oriented Model of Cancer Care (E-MOC) project is designed to integrate equity-oriented, team-based care approaches to cancer care delivered at BC Cancer – Vancouver. This project aims to reduce disparities in access, experiences, and outcomes for PWLE-HSI by weaving culturally safe, trauma-informed, and harm reduction-based practices into clinical practice and workflows. Methods: Spanning from 2023 to 2027, the E-MOC project, supported by the Canadian Partnership Against Cancer, draws on the unique ability of Clinical Nurse Specialists' to lead system-level transformation and build capacity for equity-oriented health care. The planning phase (2023-2024) involved community engagement with community-based health and social service providers and intervention co-creation with PWLE-HSI through workshop dialogue and clinical observations. Results: Co-creation and planning efforts resulted in a nurse-led quality improvement project comprised of three components: (1) a knowledge mobilization intervention for staff and clinicians to strengthen knowledge and skills about equity-oriented health care, (2) consultative and at-the-elbow mentorship and support for TBC staff and clinicians at BC Cancer – Vancouver by a Clinical Nurse Specialist with expertise in equity-oriented health care, and (3) interdisciplinary, intersectoral care conferences to plan more equitable, collaborative, and comprehensive person-centred approaches to cancer care for PWLE-HSI. Conclusion: By leveraging advanced practice nursing leadership and engaging PWLE-HSI and community in co-creation activities, our implementation science project presents a uniquely embedded approach to tackling inequities in cancer care.

LAY SUMMARY

Nurses are well-positioned to advance health and healthcare equity in the cancer care system. Authentic partnerships with people with lived experiences of health and social inequities (PWLE-HSI) are essential to closing health equity gaps in cancer care. Our project established a unique collaboration and commitment from health system leaders, oncology staff and clinicians, community-based health and social service providers, and PWLE-HSI to co-create, implement, and evaluate novel approach to equity-oriented cancer care. Co-designed equity-oriented interventions can be tailored to local cancer settings to provide impactful supports for staff an organizations, while advancing person/family-centred improvements for people who come for care.

Health Disparities and Equity

Sunday, November 2, 2025

Women's Experiences with Early-Onset Colorectal Cancer (EO-CRC) - Poster #275

Presenter: Brynn Webber, St. Francis Xavier University

Other Authors: Julia Penny, Safora Johansen, Aisha Lofters, Arlinda Ruco

ABSTRACT_ID#2658

ABSTRACT

Incidence of early onset colorectal cancer (EO-CRC) has been on the rise in many countries including Canada and Australia. Studies have found that EO-CRC patients often experience misdiagnoses, longer wait times for treatment, and a general lack of attention. Additionally, patients who are women face additional barriers to receiving appropriate primary care. Research on women's experiences specifically with EO-CRC remains sparse. To address this gap, the current qualitative descriptive study conducted one-on-one semi-structured interviews with 23 Canadian women who were diagnosed with colorectal cancer before the age of 50. Two independent coders analyzed the data using inductive coding and thematic analysis. Results showed that signs and symptoms prior to an official diagnosis varied for many participants and family physicians often did not suspect they were related to colorectal cancer resulting in misdiagnosis and in some cases, dismissal. While some women were completely dissatisfied with the healthcare system and the healthcare providers they interacted with, others reported positive experiences. Many unique considerations for EO-CRC patients were identified including lack of research and discussion on the lifelong side effects of treatment for young patients. Women interviewed in our study alluded to the lack of discussions related to the impacts of treatment on fertility, especially for those diagnosed in their 20s or 30s. Participants shared a need for more education and advocacy for women with EO-CRC. Our study provides important insights for maximizing the patient experience and quality life of women diagnosed with EO-CRC.

LAY SUMMARY

Women diagnosed with colorectal cancer before age 50 may experience challenges related to dismissal of signs and symptoms or misdiagnosis. There were varied experiences with the healthcare system including both positive and negative experiences related to the pre-diagnosis, diagnosis, and treatment stages. Recommendations for improving the experience of women with EO-CRC include discussions related to the lifelong impacts of treatment, and especially on fertility for women diagnosed in their 20s and 30s.

Health Disparities and Equity

Monday, November 3, 2025

Examining the Supportive Care Needs of Patients with Breast Cancer Who Are Black: a scoping literature review and nominal consensus groups - Poster #276

Presenter: Etienne Oshinowo, Princess Margaret Hospital

Other Authors: Tutsirai Makuwaza, Jennifer Jones, Emily Peterson, Kyle Juliao, Saidah Hack, Melanie Powis, Jennifer Ryan, Michelle Audoin

ABSTRACT_ID#3042

ABSTRACT

Black women experience numerous structural and socioeconomic barriers to equitable cancer care. However, there is minimal research on this population's specific supportive care needs. To address this gap, we performed a scoping review to synthesize the findings of prior assessments of the supportive care needs of patients with breast cancer who are Black to compare them with the findings of a pan-Canadian needs assessment conducted by our team. A systematic search of 7,508 articles from Embase, CINAHL, MEDLINE, and Web of Science yielded 35 relevant articles, which were then thematically coded. A virtual nominal consensus group (NG) was conducted to identify and prioritize the needs of Canadian Black-identifying patients with breast cancer. The findings were validated through interviews and surveys of additional patients and community advocacy groups. The most frequently reported needs in the literature were access to culturally-tailored resources and education (66%; 23/35); which were consistent with our NG findings. Employment, caregiver support, and fertility preservation were all identified in our NG, though only a few articles emphasized these supports (3-9%; 1-3/35). Access to information to make informed choices about clinical trials was seen in the NG but not identified in the literature. The NGs and scoping review emphasized that beyond relevant supportive care, improved cultural awareness and the use of trauma-informed approaches to care are needed to address structural barriers long-term. This work will help to inform the co-creation of a local supportive care program for patients with breast cancer who self-identify as Black.

LAY SUMMARY

3 key messages: There are disparities that negatively affect Black women's experience in the cancer care continuum and there is a need for research that can address these gaps. However, today there has been relatively little research on the unique needs of this population. Our findings are largely consistent with the literature; though, they highlight additional needs that were not previously identified, including access to clinical trials and information to make informed decisions about participation, and the need for care and information continuity; particularly concerning the management of concurrent medical conditions. Beyond relevant supportive care, both the literature and participants in our NGs emphasized the need for improved cultural awareness and the use of trauma-informed approaches to care to address structural barriers in the long term.

Health Disparities and Equity

Sunday, November 2, 2025

More responsive cancer system design: what developmental evaluation can teach us - a practical use case - Poster #277

Presenter: Courtney Petruik, University of Calgary

Other Authors: Lisa Cleveley, Tara Bond

ABSTRACT_ID#2946

ABSTRACT

Background: Cancer system planning often relies on retrospective evaluation approaches. In contexts requiring collaboration across multiple teams and sectors, such as improving cancer diagnosis pathways for unhoused individuals, traditional methods constrain can learning or bypass real-world complexity. Developmental Evaluation (DE) offers an alternative, supporting relationship-centred collaboration, iterative learning, and real-time adaptation. Objectives: This reflection showcases insights from cancer system planners at Cancer Care Alberta (CCA) who engaged an external developmental evaluator while leading the Optimizing Cancer Diagnosis Care for Unhoused People in Alberta (CANDU) project, a Primary Care-led initiative that co-designed a cancer diagnosis model for unhoused patients. Approach: An embedded developmental evaluator supported the team through observation, reflection, and real-time feedback. Planners drew on DE principles such as adaptability, feedback-as-data, and relational accountability. Individual experiences, meeting notes, team surveys & debriefs, evaluator fieldnotes, and project management documents informed reflections. Key Reflections: DE enabled the team to better frame complex problems, stay responsive to emerging insights, and challenge assumptions. Enabling factors included evaluator integration and a learning-oriented culture; challenges included time constraints and discomfort with ambiguity. DE was experienced as both a mindset and a method. Conclusion: DE supported responsive and inclusive project development across a complex setting. For system planners, DE-embedded planning can enhance collaboration, real-time adaptation, and appropriate design. For researchers, this reflection offers practical evidence on applying DE to support cancer system improvement. DE helps ensure designs are shaped by real-world and real-time perspectives, making planning responsive and reflective of actual needs of people, particularly underserved groups.

LAY SUMMARY

- Cancer system planning needs more flexible evaluation approaches. Traditional planning and evaluation methods can be slow to adapt to changing needs, and when solutions are unknown. Developmental Evaluation (DE) supports real-time learning and adjustments during planning and execution—not just after programs are designed or implemented.
- The CANDU project shows how DE works in practice. A developmental evaluator was embedded in the team designing a cancer diagnosis model of care for unhoused patients. Reflections, observations, team feedback, and project documents indicate that DE helped the team reflect, course-correct, and respond to new insights as the model was co-designed.
- DE helped make the planning process more responsive. It improved clarity in problem framing, enabled responsiveness to operational realities, helped assumptions be questioned and, informed decisions at key design points,. It also revealed that communication, flexibility, and openness to continuous learning are key to making DE useful.

Health Disparities and Equity

Monday, November 3, 2025

How are adults with intellectual and/or developmental disabilities represented and included in cancer research: A systematic review - Poster #278

Presenter: Rebecca Hansford, Queen's University

Other Authors: Nicole Bobbette, Christina Godfrey, Laura Mullins, Preeyaporn Srasuebkul, Anna Przednowek, Martin McMahon, Alyson Mahar

ABSTRACT_ID#2492

ABSTRACT

Introduction: Engaging in patient-partnered cancer research is imperative for informing person-centred care priorities. While reviews have documented disparities in cancer-related care, none have explored how adults with intellectual (and/or developmental) disabilities lived experience were involved in research. This mixed-methods systematic review poses the question: How are adults with intellectual disabilities included and represented in cancer research? **Methods:** A systematic review, including terms specific to intellectual disabilities and cancer, will be conducted in Medline, Embase, and CINAHL from inception to July 1, 2025. The search protocol will be registered with Prospero. Review and extraction will be conducted independently by two reviewers. Data extraction will include the methods used to involve people with intellectual disabilities in the research (based on the International Association for Public Participation's Spectrum of Engagement) and the recruitment process employed. We will consult with our cancer advisory board to inform our data analysis. Included articles will be critically appraised. We plan to use a convergent integrated approach that combines qualitative and quantitative findings across cancer care continuum milestones. **Hypothesized Results:** The review is currently in progress. Our team is currently conducting title and abstract screening on 6228 studies. A broad range of studies have been identified. Full text screening, data extraction and quality appraisal will be completed next. **Impact:** The findings will highlight ways that adults with intellectual disabilities are included and engaged in cancer research and will identify gaps in inclusion that may guide future researchers on how to engage with this population in cancer research authentically.

LAY SUMMARY

Key Messages 1. Despite growing cancer advocacy movements, adults with intellectual and/or developmental disabilities are not often included in cancer research. 2. These findings will highlight the ways that adults with intellectual and/or developmental disabilities are or are not included in cancer research. 3. This information could provide future researchers with strategies to meaningfully engage in with this patient population.

Health Disparities and Equity

Monday, November 3, 2025

Association of sociodemographic and remoteness factors with local-area cancer survival in Ontario, 1999-2018 - Poster #280

Presenter: Lindsay Obress, Ontario Health

Other Authors: Todd Norwood, Xinyi Ge, Shabnam Balamchi, Zeinab El-Masri, Simron Singh, Jill Tinmouth, Prithwish De

ABSTRACT_ID#3276

ABSTRACT

Background: Health disparities across demographics, socioeconomic and geographic subgroups affect cancer outcomes. In the absence of individual-level data, this study uses area-level data to estimate the odds of excess mortality (a relative survival measure) associated with socioeconomic indicators and geographic location among people with cancer by local areas in Ontario. **Methods:** We used population-based retrospective cohorts of adult Ontario residents diagnosed with kidney, liver and other cancers, having follow-up time between January 1, 1999, and December 31, 2018. Each person was assigned a local-area (amalgamation of Census Tracts and Census Subdivisions) at time of diagnosis using the Postal Code Conversion File plus and then linked to Ontario Marginalization Index dimensions (material deprivation, residential instability, dependency). A Bayesian flexible parametric spatio-temporal model adjusted for demographic, socioeconomic and geographic variables estimated local-area variation in the odds of excess mortality over successive five-year periods between 1999 and 2018. **Results:** While adjusted excess mortality among people with cancer decreased over the past two decades (survival improved relative to background mortality rates), these decreases have been unequal and geographically clustered. For many cancer types, the largest urban areas had the lowest odds of excess mortality (highest survival) when compared with smaller urban, rural and remote areas. For kidney, liver and several cancer types, increasing material deprivation was associated with higher excess mortality. **Conclusion:** Spatio-temporal modelling of excess mortality demonstrates persistent geographic disparities in cancer survival across Ontario after adjustment for patient demographics, marginalization and remoteness, highlighting the inequalities in cancer outcomes.

LAY SUMMARY

Cancer survival has improved across Ontario over time, but improvement is not equal across all subgroups in the population. Increased marginalization and geographic factors, such as remoteness, are associated with lower survival for several cancer types. Stability in the patterns over time suggest that additional factors (e.g., access to care) may contribute to the disparities

Health Disparities and Equity

Monday, November 3, 2025

Mobilizing Community Health Ambassadors to Support HPV Self-Screening Among Underscreened Populations in Alberta - Poster #282

Presenter: Madison Fullerton, Praxus Health

Other Authors: Gina Vetro, Caitlin Ford, Chelsea D'Silva, Holly Dievert, Bonnie Chiang, Sisi Onobrakpor, Huiming Yang, Jia Hu

ABSTRACT_ID#2346

ABSTRACT

Background: In 2020, the World Health Organization launched a global plan to eliminate cervical cancer by the end of the century. In alignment, Canada aims to achieve this by 2040 through improved access to cervical cancer screening, including HPV self-screening, an approach particularly beneficial for underscreened populations such as immigrants and refugees (i.e., newcomers). This project aimed to explore effective strategies to engage newcomer communities in HPV self-screening across Alberta. **Methods:** Using a community-based health equity approach, we trained 10 Community Health Ambassadors (CHAs) across Alberta in fall 2024. CHAs are trusted community members who support health education and health system navigation in first languages. In January 2025, CHAs were deployed in urban and rural areas to recruit individuals who are: female or a person with a cervix, aged 25–69, unscreened for cervical cancer in the past five years, and are newcomers to Canada within the past 10 years. **Results:** Since deployment, CHAs have engaged newcomer communities through women's health events, public libraries, faith-based organizations, community associations, English language classes, and other resettlement programs. To date, they have reached over 2,000 newcomers, identified approximately 430 eligible participants, and collected around 347 HPV self-screening samples. The CHAs will continue their efforts in the community until August 2025. **Conclusion:** This project demonstrates the importance of culturally relevant, community-led approaches to increase cervical cancer screening among newcomers and potentially other underscreened communities. Findings will inform provincial strategies and contribute to Canada's 2040 goal of eliminating cervical cancer.

LAY SUMMARY

Trusted community members can successfully support culturally safe cervical cancer screening. Through this project, we trained 10 Community Health Ambassadors, newcomers fluent in their community's first languages, to educate peers and distribute HPV self-screening kits. This approach reached over 2,000 individuals and supported nearly 350 newcomers in completing self-collected HPV tests. HPV self-screening is a promising tool to reduce barriers and advance equity in cancer screening. By offering an accessible alternative to clinic-based screening, HPV self-screening helps address cultural, linguistic, and systemic barriers faced by newcomers to Canada. These findings can inform provincial strategies to meet Canada's goal of eliminating cervical cancer by 2040.

Health Disparities and Equity

Sunday, November 2, 2025

Managing Challenges Facing Oncotype-DX testing in Rural Communities: A Mixed-method Study - Poster #283

Presenter: Enam Alsayheen, Nova Scotia Health

Other Authors: Wissam Saliba

ABSTRACT_ID#2867

ABSTRACT

Background: Genomic testing, particularly the Oncotype DX (ODX) test, is a key component in guiding personalized treatment for breast cancer patients. However, in rural communities, ODX is often underutilized, leading to treatment delays and limited patient understanding. **Objective:** This initiative aimed to streamline the ODX ordering process by shifting test initiation from the first in-person Medical Oncology (MO) consultation to the triage stage. The MO contacted patients by phone, explained the test in plain language, and obtained consent for immediate ordering. **Method:** Conducted at a medium-sized cancer center in rural Nova Scotia, this mixed-method study evaluated the impact of the new process. Quantitative data assessed wait times from surgery to treatment initiation. Qualitative data were gathered through semi-structured interviews with 11 breast cancer patients, focusing on their experiences and satisfaction with MO communication. **Results:** Between July 2018 and July 2022, 180 patients underwent ODX testing; 55% via the standard pathway and 45% through the new intervention. The median wait time from surgery to ODX results decreased by 14 days ($p < 0.001$). Time from MO consultation to treatment planning dropped from 24 to 0 days ($p < 0.001$), with 82% of patient ready to discuss their treatment plan during the first consultation. Patients reported high satisfaction with the clarity and empathy of remote communication. **Conclusion:** This intervention significantly reduced delays in treatment planning and improved patient understanding and satisfaction, highlighting the value of early, clear communication and streamlined processes in rural cancer care.

LAY SUMMARY

1. **Streamlined Testing Reduces Delays** The intervention significantly shortened the time from surgery to treatment planning by initiating the Oncotype DX (ODX) test earlier in the care pathway. By having Medical Oncologists order the test during triage via phone consultations, the median wait time from surgery to ODX results was reduced by 14 days. This led to faster treatment decisions, with 82% of patients consenting to chemotherapy during their first oncology consultation.
2. **Improved Patient Understanding and Satisfaction** Patients reported high satisfaction with the new approach, particularly appreciating the use of plain language and the opportunity to discuss the test remotely. This method enhanced their understanding of the ODX test and its role in treatment planning, reducing anxiety during the waiting period and promoting a more patient-centered experience.
3. **Enhanced Access for Rural Populations** The initiative addressed barriers faced by rural and underserved communities, where access to timely genomic testing is often limited. By reducing the need for additional travel and expediting the testing process, the intervention improved equity in cancer care delivery, ensuring that patients in remote areas received timely, informed, and personalized treatment recommendations.

Health Disparities and Equity

Monday, November 3, 2025

Recognizing Endometrial Cancer Risks in Perimenopausal and Postmenopausal Experiences: Insights From Community Qualitative Interviews and Workshop - Poster #284

Presenter: Malak Ibrahim, University of Calgary

Other Authors: Aline Talhouk, Karman Johal, Madeline Dow, Mohamed Bedaiwy, Andrea Neilson, Jerilynn Prior

ABSTRACT_ID#2943

ABSTRACT

Introduction: Endometrial cancer (EC) is the most common gynecologic malignancy in the developed world. If EC is detected early, before metastasis, 5-year survival rates exceed 95%. Conversely, only 20% survive EC diagnosed after metastasis, highlighting the importance of early detection. This study investigated the experiences of perimenopausal and postmenopausal women, and EC survivors in British Columbia to learn about their perceptions of reproductive aging, and potential concerns or experiences with EC. Methods: We interviewed 31 midlife women of diverse backgrounds and hosted a workshop for more in-depth discussion. We summarized relayed experiences and beliefs through a thematic and descriptive analysis of participant stories and workshop feedback. Results: Participants demonstrated a simplistic understanding of midlife changes, facing this phase of life with a “tough-it-out” attitude rather than seeking medical help for arising symptoms. Awareness of EC and EC-specific risk factors, such as obesity, was low. Confusion between cervical and EC was common. Although abnormal bleeding was seen as potentially of concern, many opted to wait before seeking medical help. Workshop participants stressed the need to include awareness about EC in a broader conversation about perimenopause and menopause and suggested strategies for disseminating EC awareness. Conclusions: Women in British Columbia demonstrated low awareness of EC-associated symptoms and risk factors. There is little information to help distinguish when perimenopausal abnormal uterine bleeding is of concern and when to seek help. This highlights the need to enhance knowledge of EC and its risk factors in perimenopause among the public and among health care providers.

LAY SUMMARY

Awareness of EC symptoms and risk factors is low in community women Women want more information to help distinguish when perimenopausal abnormal uterine bleeding is of concern and when to seek help Endometrial cancer is often confused with cervical cancer

Health Disparities and Equity

Sunday, November 2, 2025

Scoping Review: Racial Disparities Reported in Canadian Cancer Care - Poster #285

Presenter: Harkeert Judge, University of Alberta/Cancer Care Alberta

Other Authors: Anna Santos Salas, Anna Pujadas Botey, Paula Robson

ABSTRACT_ID#2351

ABSTRACT

Background: In Canada, promoting health equity for racialized populations within the cancer care system has become an increasingly important focus. This is in part due to recent studies reporting racial disparities in access to cancer care. To explore disparities in cancer care for racialized groups, it is important to understand that race is not a naturally occurring scientific category. Racialization is the social, historical, and political process by which 'race' comes to have meaning for certain groups or communities. **Objectives:** This study aims to: (1) summarize Canadian literature reporting racial disparities in areas of cancer care (e.g. screening, diagnosis, and treatment); and (2) describe the reported factors affecting these disparities among racialized populations. **Methodology:** This scoping review follows the JBI scoping review methodology. The search strategy identifies both published articles and grey literature on this topic. **Findings:** This scoping review is currently in progress and the search results and study selection process will be fully detailed in the final scoping review and presented according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR). We provide a comprehensive synthesis of existing literature reporting racial disparities in Canadian cancer care, including studies that examine whether racial disparities exist and articles describing initiatives to address racial disparities. **Conclusion:** The review will inform future research and health system initiatives to improve cancer care for racialized populations in Canada.

LAY SUMMARY

- This work maps Canadian literature reporting racial disparities in cancer care screening, diagnosis and treatment over the last 10 years. The concept 'racial disparities' refers to differences, inequities, or inequalities in areas of cancer screening, diagnosis, and treatment that are attributed to an individual's ethnicity or race. - Race is a social construct affecting patients' experiences or outcomes upon receiving cancer diagnosis or treatment. Disparities in health care for racialized populations intersect with social, economic, environmental, and historical contexts in Canada. - Through this review we identify gaps in the literature and recommendations for future research that can improve cancer care of racialized populations

Health Disparities and Equity

Sunday, November 2, 2025

Investigating the experiences of Black women with metastatic breast cancer - Poster #287

Presenter: Ilakkiah Chandran, Women's College Hospital

Other Authors: Emily Suthaharan, Rumaisa Khan, Aisha Lofters

ABSTRACT_ID#2357

ABSTRACT

Black women with metastatic breast cancer (mBC) often face systemic inequities and stigma that influence their healthcare experiences and interactions within the healthcare system. Despite being at higher risk for breast cancer-related morbidity and adverse outcomes, their voices are frequently underrepresented in research. This study explored the healthcare experiences of Canadian Black women with mBC to better understand the barriers they encounter and to identify areas for improvement. Using semi-structured interviews and focus groups, Black women who had received a diagnosis of mBC at any point in their lives from across Canada were engaged in a virtual format. An interview guide facilitated discussion, and transcripts were analyzed thematically using an inductive approach by two independent researchers. Five key themes emerged from the data: the necessity and emotional burden of self-advocacy; the impact of exclusion and lack of representation on patient care; the social and emotional toll of living with mBC; cultural stigma and taboo surrounding cancer; and patient-informed recommendations for improving care. These findings highlight the complex ways in which structural inequities, cultural narratives, and gaps in care impact the wellbeing of Black women with mBC. This study calls for more identity-affirming care, increased visibility of Black women in cancer education and research, and better access to culturally relevant resources as addressing these barriers is essential for improving not only the healthcare journey but also the overall quality of life and outcomes for Black women with mBC.

LAY SUMMARY

1. Systemic barriers, including racism and the lack of identity-affirming care, significantly shape the healthcare experiences and outcomes of Canadian Black women with metastatic breast cancer. Understanding these influences is essential to addressing inequities in care.
2. Black women in our study consistently described self-advocacy as a necessary part of navigating the healthcare system. Many felt that without speaking up, they risked receiving inadequate care, yet even with persistent advocacy, some still experienced neglect, highlighting a deep mistrust and systemic failure in patient-provider relationships.
3. Patient-informed recommendations provide actionable ways for primary care providers and health professionals to improve the inclusiveness, equity, and cultural responsiveness of care for Black women and other marginalized individuals with metastatic breast cancer. Including patients in care-related decisions is not only respectful, but is essential to supporting their quality of life and improving long-term outcomes.

Health Disparities and Equity

Sunday, November 2, 2025

Optimizing Equitable Cancer Diagnoses in Canada - Poster #289

Presenter: Leah Stephenson, All.Can Canada

Other Authors: Paula Holmes-Rodman, Michelle Audoin

ABSTRACT_ID#2380

ABSTRACT

Earlier cancer diagnosis means better health outcomes and reduced healthcare costs, but most cancers in Canada are diagnosed at later stages through symptom presentation, not screening. Structurally underserved communities face greater barriers. All.Can Canada (ACC) is a national, multi-stakeholder network for cancer care efficiency, with the aim of optimizing entry into cancer care through swift, accurate, equitable, and appropriately communicated diagnosis. Join a panel about improving equitable, earlier cancer diagnoses. Co-presenters are people with lived/living expertise including a Black woman living with advanced/metastatic breast cancer and a caregiver of a neurodiverse sister with ovarian cancer. The panel will share All.Can Canada's results of mixed methods research on optimizing earlier, more equitable cancer diagnoses in Canada. Inefficiencies within the current state of diagnosing cancer from the point of symptoms through to treatment planning will also be offered, including those linked to barriers for structurally underserved communities. Participants will learn about ACC's findings for an optimal future state and "bright spots" of excellence across Canada that can be scaled to achieve more inclusive, equitable, and earlier cancer diagnosis for all.

LAY SUMMARY

People experience cancer diagnosis in three phases - symptom/suspicion; investigation; and diagnosis (including clinical, pathological, and therapeutic). No matter the type of cancer or determinants of health, people are trying to achieve seven key outcomes in order to achieve a quality diagnosis experience. However, few people have those outcomes. Instead, people's experiences are full of delays, requiring extensive self-advocacy, leading to late diagnosis. This is worse for structurally underserved communities, and can be best understood through an intersectional lens. There is a clear, improved future state for cancer care in Canada and many evidence-based practices to achieve it. Those practices require scale, resource investment, and broader understandings of equity in cancer care.

Health Disparities and Equity

Monday, November 3, 2025

Take Action, Take Control: A genetic testing initiative for cancer risk in Black women - Poster #290

Presenter: Michelle Audoin, Patient Partner

Other Authors: Camille Williams, Maria Madden, Angelina Tryon, Mohammad Akbari, Elysia Bryan, MJ De Coteau, Ariana Del Bianco, Emma Rinaldo, Tameika Shaw, Aisha Lofters, Elaine Goulbourne

ABSTRACT_ID#2887

ABSTRACT

Background. Genetic testing for hereditary cancers is underutilized by racialized groups and those of lower socioeconomic status for numerous reasons including lack of knowledge and awareness, cost, distrust due to historical inequalities, family concerns, and lack of referrals from providers. To help address this inequity, we will: (1) develop culturally relevant educational materials about genetic testing and counselling for Black women; and (2) reduce barriers to accessing genetic testing for a limited number of Black women. **Methods.** We invited patient and community partners to generate ideas for educational resources, developed and publicized a subset of these ideas. Next, we will collaborate with community partners to generate and develop ideas for improving access to genetic testing. **Results & Discussion.** We successfully developed, and promoted via social media, a campaign called “Take Action, Take Control”: a webpage (>1500 views) with resources and FAQs, an informative video (>300 views), and an anonymous survey. Of 40 survey respondents, two-thirds indicated that they know a little or lot more about hereditary cancer genetic testing after visiting the webpage and 82% rated ease of understanding the information as ≥ 4 on a 5-point scale. Respondents also indicated that a barrier to seeking testing is the belief that they are ineligible under their provincial insurance plan and cannot pay out-of-pocket. These early results suggest that our webpage has been useful, and that phase 2 of the project aligns with the community’s needs. We will also continue to refine the webpage based on feedback from the survey.

LAY SUMMARY

- Relationships with patient and community partners are essential for developing initiatives that help address health inequities.
- Additional research is required to understand the factors that affect Canadian Black women's utilization of genetic testing to understand their risk of cancer.
- Additional work is needed to improve healthcare providers' understanding of how Black individuals' lack of knowledge about family history and other cultural factors may affect their eligibility for genetic testing and how these inequities may affect their health outcomes.

Health Disparities and Equity

Sunday, November 2, 2025

EXCEL-Punjabi Protocol: A Culturally Tailored Pilot Exercise Oncology Intervention for Punjabi Speaking Individuals Living with and Beyond Cancer - Poster #291

Presenter: Mannat Bansal, University of Calgary

Other Authors: Imrose Bhullar, Sarbjit Jawandha, Chandan Sangha, Jagbir Kaur, Gurleen Brar, Meghan H. McDonough, Tanvir C. Turin, S. Nicole Culos-Reed

ABSTRACT_ID#3008

ABSTRACT

Introduction: Despite available exercise oncology interventions, there continues to be low uptake for individuals of South Asian heritage, the largest visible minority group in Canada. This pilot 12-week intervention has culturally adapted the Canada-wide EXCEL intervention for the Punjabi speaking population. The main objectives are to assess (1) feasibility and acceptability; (2) implementation markers; and (3) preliminary effectiveness. **Methods:** Participants will include Punjabi speaking individuals of South Asian heritage (optional support persons may also participate), over the age of 18, diagnosed with cancer, on or off treatment, able to safely engage in exercise and the assessments, and able to complete informed consent. The multimodal exercise intervention consists of online or in-person one-hour classes, two times per week, for 12-weeks. Behaviour change is supported with additional educational handouts, and via group discussion facilitated by the instructor pre/post exercise class. Measures at baseline and post-intervention include psychosocial questionnaires and fitness assessments, as well as semi-structured interviews post-intervention. **Data Analysis:** Sociodemographic and medical information, feasibility, and fidelity data will be analyzed using descriptive statistics. Psychosocial questionnaires and fitness assessments will be analyzed using descriptive statistics and general linear mixed models will examine changes in outcomes pre- to post-intervention. Qualitative data will provide further insight into the participant experience and will be analyzed using interpretive description. **Significance:** EXCEL-Punjabi has the potential to support exercise behaviour change in the currently underserved population of South Asian individuals living with and beyond cancer. Results will inform continued cultural tailoring of our exercise oncology resources.

LAY SUMMARY

EXCEL-Punjabi is a 12-week exercise program for Punjabi-speaking individuals living with or beyond cancer and their optional support persons. Specifically, we aim to explore: **Feasibility** – How easy is it to run a 12-week exercise program for Punjabi-speaking individuals living with or beyond cancer? **Implementation** – What resources are needed to run the program? Are facilitators able to deliver it as intended, and are there any challenges raised by participants, trainers, or sites? **Impact** – How does participating in a 12-week exercise program (delivered either online or in-person) affect the mental and physical health of Punjabi-speaking individuals living with or after cancer? We plan to start the program in August 2025, offering both online and in-person options. The program includes twice-weekly group-based exercise classes, focusing on strength training, cardiovascular fitness, balance, and flexibility. All exercises will be adapted to participants' individual needs. In addition to the classes, participants will complete physical assessments and questionnaires before and after the program and may be invited to participate in a short interview about their experience.

Health Disparities and Equity

Monday, November 3, 2025

From Awareness to Uptake: Equipping Community Health Ambassadors to Overcome Structural Barriers in Cancer Screening - Poster #292

Presenter: Sara Shearkhani, Michael Garron Hospital

Other Authors: Kelly Smith, Jeff Powis, Anne Wojtak, Razia Rashed, Mary Hill

ABSTRACT_ID#2728

ABSTRACT

Despite numerous awareness campaigns, cancer screening rates remain disproportionately low among immigrant women living in equity-denied neighborhoods. These women face not only cultural and informational barriers, but also deeply rooted structural challenges — from inaccessible healthcare systems to a lack of culturally appropriate support. The From Awareness to Uptake project aims to bridge this persistent gap between knowledge and action by equipping Community Health Ambassadors (CHAs) with the training, tools, and governance support needed to guide women from awareness to actual cancer screening. Funded by a CIHR Catalyst Grant, this initiative is grounded in partnerships with East Toronto Health Partners (ETHP), Health Access Thorncliffe Park (HATP), Canadian Cancer Society (CCS), TNO-Toronto Neighborhood Organization, Toronto Central Regional Cancer Program (TCRCP), Ontario Health, the City of Toronto, and Michael Garron Hospital (MGH). The project includes two phases: first, the co-design of a practical, culturally relevant intervention with immigrant women and community stakeholders; and second, a small-scale implementation and evaluation in East Toronto's priority neighborhoods to refine and finalize the model. By shifting focus from individual behavior to structural barriers, and by investing in trusted community leadership, this project aims to significantly improve breast cancer screening uptake, reduce disparities, and build sustainable, community-embedded pathways to care. The work is grounded in principles of equity, integrated care, and community co-design. Lessons learned will support other Ontario Health Teams and inform equity-focused cancer prevention strategies across Canada.

LAY SUMMARY

1. Knowing isn't enough — structural barriers matter. Many immigrant women know about cancer screening, but things like not having a family doctor, language barriers, transportation challenges, and fear of being treated unfairly stop them from getting screened.
2. Community Health Ambassadors (CHAs) can bridge the gap. CHAs are trusted members of the community. With the right training and support, they can do more than raise awareness — they can help women book appointments, navigate the system, and follow through on screening.
3. System change starts local. By working closely with community members and health partners, we are designing practical, community-led solutions to remove barriers to breast cancer screening. This small project will teach us how to make the healthcare system work better — not just here in East Toronto, but in other communities across Canada too.

Health Disparities and Equity

Sunday, November 2, 2025

Incorporation of ASCO–Friends of Cancer Research (AFCR-HIV) Eligibility Criteria for People Living With HIV (PLWH) in Cancer Clinical Trials of the Canadian Cancer Trials Group (CCTG) - Poster #293

Presenter: Maria F. Comelles, Canadian Cancer Trials Group

Other Authors: Lynda Balneaves, Maria F. Comelles, Annette E. Hay, Santiago Perez, Tessa Senneker, Anna Johnson, Lisa K. Hicks, Wendy Parulekar, Janet Dancey, Bingshu E. Chen

ABSTRACT_ID#2428

ABSTRACT

Background: In 2017, AFCR-HIV recommended limiting exclusion of PLWH from clinical trials, given improved survival with antiretroviral therapy. We evaluated how CCTG protocols align with these guidelines. **Methods:** All CCTG trials open to accrual or in follow-up were reviewed in 2025Q2. Associations between HIV eligibility and trial characteristics (cancer type, intervention, phase, leading group) were assessed using Chi-square tests. **Results:** Of 136 trials, 81.6% focused on solid tumors, 14.7% on hematology malignancies and 3.7% were mixed. 39.7% were CCTG-led trials. 37.5% were activated between 1999–2017, 47.1% between 2018–2023, and 15.4% between 2024–2025. Interventions encompassed systemic therapy including immunotherapy in 63.2%, combinations in 13.9%, radiation in 8.8%, low-risk in 8.8%, surgery in 3.7% and immunization in 1.5%. Of the 130 interventional trials, 31.6% were phase I/II, 61.5% were phase III and 6.9% were phase II/III. At the time of analysis, 61% were open to accrual; 39% closed with follow-up ongoing. PLWH were included, unmentioned or excluded in 49/136 (36%), 55/136 (40.4%) and 32/136 (23.5%) respectively; with justification in 7/32 (21.9%). PLWH were more likely to be excluded in systemic therapy trials ($p=0.008$), phase I/II trials versus phase III ($p=0.010$) and CCTG-led trials versus non-CCTG-led (31.5% versus 18.3%, $p=0.076$). **Conclusion:** PLWH were eligible for most CCTG trials but some protocols still excluded them without justification. Trial phase and therapy may explain differences in some comparisons. In 2024, CCTG generic protocol eligibility criteria were formally modified to ensure greater consistency in alignment with AFCR-HIV.

LAY SUMMARY

Introduction: Clinical trials are vital for improving cancer treatment, but not everyone has had equal chances to participate. People living with HIV (PLWH) have often been excluded from trials, despite major advances in HIV care. In 2017, ASCO–Friends of Cancer Research (AFCR-HIV) shared guidelines recommending that PLWH should be included in cancer trials when safe and should not be left out without justification. To see if guidelines are being followed, we reviewed the eligibility criteria of 136 trials from the Canadian Cancer Trials Group (CCTG). **Key findings:** Most trials now include PLWH: Over one-third explicitly allow PLWH to participate, and many others don't mention HIV status, giving doctors flexibility to include them when safe. **Reasons for exclusion:** Early-phase and systemic therapy, such as immunotherapy or conventional chemotherapy trials, exclude PLWH more often, likely due to safety concerns. While 23.5% of trials excluded PLWH, only about 22% gave clear reasons. **Positive change underway:** In 2024, CCTG updated its protocol templates to better align with AFCR-HIV recommendations, promoting fairer access. Though some trials still exclude PLWH without clear justification, progress is clear. **Conclusion:** These steps show growing fairness in cancer research, offering PLWH more chances to benefit from cancer clinical trials.

Health Disparities and Equity

Monday, November 3, 2025

Understanding Transitions in Care for Individuals Living with and Beyond Cancer in High Income Versus Low-and-Middle Income Settings: A scoping review - Poster #294

Presenter: Sarina Aryal, University of Calgary

Other Authors: Jaling Kersen, Stefan Kurbatfinsk, Abigail Thomas, Seremi Ibadin, Areej Hezam, Diane Lorenzetti, Khara Sauro

ABSTRACT_ID#2821

ABSTRACT

Background: Low-and-middle-income countries (LMIC) may experience challenges providing high-quality care for individuals diagnosed with cancer compared to high-income countries (HIC). These challenges can be amplified during transitions in care (TiC), when patients move between healthcare providers/settings, which are vulnerable periods in care delivery leading to greater costs and poorer outcomes. The evidence around TiC is varied and comparisons between studies in LMIC and HIC have not been explored. The objective of our study was to map and characterize evidence on TiC by country-level income. **Method:** Six databases were searched to identify literature around TiC and cancer. Country-level income was classified using the World Bank income-level classifications. Data on cancer type, types of TiC, and interventions to improve TiC were abstracted. Data were synthesized using descriptive statistics. **Result:** 799 studies were included; 738 from HIC and 61 from LMIC. The TiC from diagnosis-to-treatment was the most commonly studied TiC in LMIC (2.51%) whereas multiple TiC were most commonly studied in HIC (21.20%). Intervention studies were less common in LMIC (n=8) compared to HIC (n=155). Models of care were found to be the most studied intervention in both HIC (n=64) and LMIC (n=4), followed by survivorship care plans (n=21) for HICs and discharge planning (n=3) for LMIC. **Conclusion:** Despite the large body of evidence on TiC for individuals with cancer, there is limited evidence on TiC in LMIC. This study highlights the need for more research on TiC in LMIC, specifically studies exploring interventions to improve TiC in cancer care.

LAY SUMMARY

The current literature around transitions in care during cancer care is mostly from high-income countries with limited evidence from low-middle income countries. Interventions to improve transitions during cancer care in low-middle income countries is understudied. More research is needed to understand transitions in cancer care in low-middle income countries, to better support high-quality, continuous cancer care in low-middle income countries.

Health Disparities and Equity

Sunday, November 2, 2025

Advancing Equity in Canadian Cancer Clinical Trials Conduct: 3CTN Demonstration Projects - Poster #295

Presenter: Raisa Chowdhury, Canadian Cancer Clinical Trials Network (3CTN)

Other Authors: Michelle Audoin, Diana Kato, Stephen Sundquist, Janet Dancey

ABSTRACT_ID#3050

ABSTRACT

Introduction 3CTN, the Canadian Cancer Clinical Trials Network, comprises 51 cancer centres aligned on aims to improve equitable access to, and performance of academic cancer clinical trials. Despite the potential to provide innovative therapies, trial participation remains limited for many equity-deserving communities due to barriers such as geography, financial hardship, attitudes about research, biases, and cultural or language factors. To begin addressing these barriers, 3CTN funded projects undertaken by member centres to advance equity, diversity, and inclusion (EDI) priorities and seed best practices in trial conduct. **Methods** 3CTN issued a Request for Applications with two funding streams: (1) Advancing EDI best practices, and (2) Applied use of CRAFT - the Canadian Remote Access Framework for Clinical Trials, a pragmatic approach to decentralized clinical trial (DCT) conduct. Eight proposals were received and reviewed by a panel, with oversight from a formed EDI Steering Committee. **Results** Six projects from Ontario, British Columbia, and Nova Scotia were selected and completed by March 2025. Projects addressed trial access and participation through approaches such as financial support for pediatric patient families, DCT implementation, improving community trust and EDI training for staff. Knowledge mobilization of outcomes and learnings derived from awarded projects will be achieved through presentations and discussion among 3CTN member centres. **Conclusion** These projects offered opportunities for fostering knowledge and achieving a better understanding of EDI priorities related to trials access and conduct. The intent is for outcomes to inform scalable, practical solutions and best practices for advancing equity for underserved patients in clinical trials.

LAY SUMMARY

1. Understanding and breaking down barriers: Six projects across Canada tackled obstacles that prevent underrepresented groups from joining and accessing cancer clinical trials. 2. Reaching patients closer to home: Some projects implemented a decentralized clinical trial model (the CRAFT framework) that allow trial patients to access treatment and trial activities closer to home, especially in remote and rural areas. 3. Supporting equitable and inclusive practices: Training staff on equity, diversity, and inclusion (EDI) practices and understanding the barriers and challenges that trial patients and different communities face. These pilot projects are helping shape future best practices for making cancer clinical trials fairer and more inclusive for all Canadians.

Health Disparities and Equity

Sunday, November 2, 2025

Disparities in Geospatial Patterns of Cervical Cancer Screening and Associated Risk factors: Findings from Alberta's Tomorrow Project - Poster #297

Presenter: Sara Nejatinamini, Alberta Health Services

Other Authors: Corin Kuang, Jian-Yi (Jason) Xu, Grace Shen-Tu, Jennifer E. Vena

ABSTRACT_ID#3024

ABSTRACT

Despite the availability of preventive vaccines, routine screening, and effective treatments, cervical cancer (CC) cases in Canada have risen since 2015. Although research has identified factors for CC screening uptake and related disparities, few studies have explored how these vary geographically. This study aims to identify 'hotspot' areas—regions with higher rates of overdue CC screening—among participants in Alberta's Tomorrow Project (ATP) and to explore the contributing factor using a machine learning approach. In this population-based cross-sectional study, we will use self-reported data from an ATP health and lifestyle follow-up survey completed in 2017 (females age<70y, n=18078) linked to data from the Alberta Cervical Cancer Screening Program (ACCSP; 2012-2018) to determine CC screening rates. The survey also captured potential factors associated with screening enablers and barriers. We will perform hotspot analysis to identify geographic clusters with higher or lower rates of overdue CC screening. Additionally, we will apply random forest machine learning to evaluate the relative importance of risk factors associated with these spatial patterns. Finally, we will map spatial clustering. Our preliminary analysis found that self-reported data underestimated overdue CC screening rates compared to ACCSP data (~20% vs. ~35%). Additionally, living in a rural area emerged as the primary factor associated with lower screening uptake. Geospatial data analysis is currently underway, with results to be presented at the conference. Linking geographic patterns of CC screening disparities to their underlying risk factors enables development of more targeted interventions to improve health care access.

LAY SUMMARY

By using ATP health, lifestyle and location data linked with provincial screening records from over 13,000 females in Alberta, the study will map out communities where fewer females are meeting recommended guidelines for screening—and where screening rates are higher than average. We will use machine learning to explore factors like income, education, and access to health care to understand why some areas have lower screening rates and which of these factors have the greatest impact. Comparing cancer screening rates across regions and identifying its related factors helps inform the development of more focused programs and policies.

Health Disparities and Equity

Sunday, November 2, 2025

Facilitators and Barriers to Accessing Telehealth Cancer Services for Underserved Communities in Canada: A Rapid Review - Poster #299

Presenter: Y. Sarah Chae, BC Cancer

Other Authors: Scott A. Lear, Ryan R. Woods, Stuart Peacock, Helen McTaggart-Cowan

ABSTRACT_ID#2402

ABSTRACT

Background: Telehealth supports the provision of cancer care and offers opportunities to improve access to services. However, little is known about the factors that facilitate or hinder access among underserved communities in Canada, who may face unique structural and social barriers to cancer care. Objective: To identify and synthesize facilitators and barriers to accessing telehealth cancer services for underserved communities in Canada. Methods: A rapid review of Canadian empirical studies was conducted using MEDLINE (Ovid), CINAHL, and Web of Science. A combination of medical subject headings and keywords were used to search studies published between 2000 and 2024. Studies were included if they focused on telehealth for individuals with cancer from one of 10 underserved communities identified by the Canadian Cancer Society. Eligible studies reported on facilitators and/or barriers to accessing telehealth anywhere on the cancer care continuum. Data extraction and quality assessment were performed using standardized tools, and studies were synthesized narratively. Results: The search retrieved 374 references, and 18 studies met the inclusion criteria. Most studies were quantitative in design. A range of underserved communities was explored, with the rural (n=6) and advanced cancer (n=5) communities most frequently being studied. Preliminary findings revealed key facilitators as ease of use, provider adaptability, and access to reliable technology. Anticipated barriers may vary by community and include a lack of culturally safe services and limited adaptability to individual or community-specific needs. Conclusions: Findings will inform equity-oriented approaches to telehealth to ensure access and inclusivity for underserved communities in cancer care.

LAY SUMMARY

Some people in Canada face more barriers to cancer care than others - including those living in rural or remote areas, Indigenous peoples, racialized communities, and others identified by the Canadian Cancer Society as underserved in the healthcare system. Telehealth can help make cancer care more accessible, but only when it's easy to use, reliable, and flexible enough to meet people's diverse needs. Some communities still face barriers, such as services that aren't culturally safe or that don't reflect their realities. This research reviewed studies from across Canada to understand what helps or gets in the way of using telehealth for cancer care among underserved groups. These insights can help shape more inclusive, equitable telehealth services - especially for those who already face challenges accessing care.

Health Disparities and Equity

Monday, November 3, 2025

Age and Sex Differences in the Global Burden of Cancer: a population-based study - Poster #300

Presenter: Taylor Hughes, Alberta Health Services

Other Authors: Miranda Fidler-Benauodia

ABSTRACT_ID#2884

ABSTRACT

Background: Descriptive cancer epidemiology often focuses on specific age groups, sexes or cancer sites, limiting comparisons across these variables. We aimed to describe global cancer incidence and mortality by age and sex. **Methods:** Using the GLOBOCAN 2022 database, we quantified overall and cancer-specific incidence and mortality using counts and age-standardized rates (ASRs; per 100,000) stratified by sex and age bands. **Results:** In 2022, an estimated 18.7 million cases and 9.7 million cancer-related deaths occurred. Overall incidence and mortality were similar between sexes in children. For incidence, the overall ASR was higher in females among 15–59-year-olds (1.1- to 2.2-fold), after which there was male dominance. Overall mortality ASRs were greater in males across all ages, except 30–49-year-olds. Among females, incidence and mortality in 15–59-year-olds was highest for breast and cervical cancer, while lung and colorectal cancer were main causes of mortality at 60+ years. In males, incidence was highest in lung, prostate and colorectal cancer, and mortality was highest for lung and liver cancer, which mostly occurred after 40 years age. Developed countries had the highest incidence ASRs across all ages, and higher mortality ASRs for those over 50 years age. There was much variability in cancer incidence and mortality by world region. **Conclusions:** The global burden and profile of cancer varies by age and sex, and further by world region and developmental level. These findings serve as a reference of the heterogeneity in the global cancer burden by sex and age.

LAY SUMMARY

Cancer is a disease of aging and consequently cancer diagnoses and deaths increase with age; however, there is much variability by sex. The regularity of sex-specific and sex dominated cancer diagnoses in young females play a large role in this variability, whereas male-specific and dominated cancers are more common in older ages. Inequities in cancer outcomes were strongest in young people, while the most developed countries had the highest cancer diagnosis rates regardless of age or sex, the least developed countries had the highest mortality rates of males before age 40 and females less than 60. These inequities were observed in cancer overall and further highlighted in the variation of specific cancer outcomes when assessed by age, sex and world region. Our study highlights the wide variability in cancer profile and outcomes by age, sex and country development level and world region, notably highlighting areas of inequity. To improve global cancer outcomes, we need to tailor cancer research, care and control initiatives to specific populations with a focus on equity and global collaboration.

Health Disparities and Equity

Sunday, November 2, 2025

Mapping the Experiences of Sexual and Gender Minorities in Oncology: An Umbrella Review - Poster #301

Presenter: Sophie Lauzier, Université Laval

Other Authors: Nataly Rossana Espinoza Suarez, Kévin Lavoie, Julie Lapointe

ABSTRACT_ID#3089

ABSTRACT

Background: In Canada, approximately 1 million individuals identify as members of sexual and gender minority (SGM) populations. Despite growing awareness, health disparities persist among SGM individuals, including in oncology. A better understanding of the care pathways and lived experiences of SGM individuals affected by cancer is critical for identifying actionable gaps in equity, diversity, and inclusion (EDI) within cancer care systems. **Objective:** This review aims to synthesize knowledge on the care pathways and experiences of SGM individuals living with cancer. **Method:** We conducted an umbrella review that included systematic, scoping, and integrative reviews focused on the cancer care experiences of SGM populations, following the Joanna Briggs Institute methodology. Data were extracted using the Cancer Care Continuum Model, and thematic analysis was used to identify recurring patterns and knowledge gaps. **Results:** A total of 44 reviews were included (21 systematic, 16 scoping, and 7 integrative). Most primary studies originated from Western countries. The literature focuses on the treatment and survivorship phases, with comparatively little evidence related to screening and end-of-life care. Breast, gynecological, and prostate cancers were the most studied. Emerging themes included: structural barriers within healthcare systems, the importance of the patient–clinician relationship, the psychosocial and relational impacts of cancer on SGM patients and their support networks. **Conclusion:** This review, in combination with the findings from our ongoing qualitative study, will help define concrete actions tailored to the Canadian context to promote inclusive oncology care.

LAY SUMMARY

Sexual and gender minority (SGM) people face unique challenges in oncology care. Most research documenting the experiences of SGM individuals is conducted in Western countries, focuses on treatment and survivorship, and centers on breast, gynecological, and prostate cancers. A better understanding will help define concrete actions tailored to the Canadian context, supporting the development of more inclusive cancer care.

Health Disparities and Equity

Monday, November 3, 2025

Examining Real-World Health Related Quality of Life at a pilot centre in Ontario, Canada: feasibility and equity factors - Poster #302

Presenter: Rebecca Mercer, Sunnybrook Research Institute

Other Authors: Teresa C.O. Tsui, Rebecca E. Mercer, Elena J. Zhou, Rahul K. Desai, Shreya Chatterjee, Curtis Y.L. Yeung, Eleanor M. Pullenayegum, Kelvin K.W. Chan

ABSTRACT_ID#2847

ABSTRACT

Introduction: Health-related quality of life (HRQoL) is frequently evaluated using EQ-5D health utilities (HUs) for health technology assessments. Routine collection of EQ-5D HUs supports real-world evaluations of publicly funded cancer treatments. There is limited real-world data examining which factors impact EQ-5D HUs in oncology. Objectives: This Ontario-based study: 1) prospectively measured real-world EQ-5D-3L HUs among patients receiving systemic cancer therapy, 2) evaluated the feasibility of routine EQ-5D-3L collection and 3) examined how demographic and socioeconomic factors influence EQ-5D-3L HUs. Methods: Patients starting publicly reimbursed systemic therapy at the Sunnybrook Odette Cancer Centre were invited to complete the EQ-5D-3L and demographic questionnaires. With a subset of patients, we conducted semi-structured interviews and analyzed their content to identify key themes informing the feasibility of future implementation. We conducted multivariable linear regression to assess the association between demographic and socioeconomic variables with EQ-5D-3L HUs. Results: Of 170 patients enrolled (05/2024 to 02/2025), 94% would definitely or very likely complete future EQ-5D-3L questionnaires. Interview participants expressed satisfaction with the EQ-5D-3L and supported its routine collection. Baseline HUs were consistent with previous cancer studies. Factors associated with lower HUs independent of cancer site were low annual income (<\$30,000), and younger females (<50 years). Implications: This study supports broader EQ-5D-3L implementation using existing infrastructure in Ontario. Our findings suggest the need to account for age, sex, and income in EQ-5D-3L analysis which are pivotal to health technology assessments. Future research can investigate widespread EQ-5D-3L collection across Ontario and additional equity-related associations with HRQoL in oncology.

LAY SUMMARY

We found that patients with cancer are satisfied with routine completion of the EQ-5D-3L questionnaire, a commonly used quality of life measure used in clinical and health economic decisions. This supports broader implementation of the EQ-5D-3L questionnaires using existing infrastructure in Ontario. Our analysis of equity factors and their association with EQ-5D-3L quality of life scores found that people with a lower annual income and younger females experience lower quality of life. This finding supports accounting for age, sex, and income in future quality of life analysis.

Health Disparities and Equity

Sunday, November 2, 2025

Socioeconomic inequalities in receipt of specialized consultation and cancer treatment among Stage IV non-small cell lung cancer patients: A population-based study in Ontario, Canada - Poster #305

Presenter: Tara Horrill, University of Manitoba

Other Authors: Laura Davis, Rebecca Griffiths, Katie Goldie, Timothy Hanna, Alyson Mahar

ABSTRACT_ID#2475

ABSTRACT

Introduction: The impacts of socioeconomic factors on cancer outcomes are well documented; however, their role in moderating access to care is less well understood. We examined associations between material resources and receipt of cancer-directed consultations, cancer-directed care, and palliative care among adults with stage IV non-small cell lung cancer (NSCLC). **Methods:** We used a population-based retrospective cohort with administrative health data to study people aged 18+ diagnosed with stage IV NSCLC between 2010-2022 in Ontario. We measured consultations with medical oncologists, radiation oncologists, and thoracic surgeons as well as systemic therapy, radiation, surgery and palliative care in the year following diagnosis, comparing consultation and treatment rates across quintiles of census measures of community material resources. Multivariable cause-specific Cox proportional hazards regression, accounting for death as a competing event, was used. **Results:** We included 32,902 stage IV NSCLC patients. People living in the least materially resourced neighborhoods were significantly less likely to have a cancer-directed consultation with a medical oncologist, radiation oncologist or thoracic surgeon (hazard ratio [HR]=0.91; 95% confidence interval [CI] 0.87-0.94) and significantly less likely to receive any cancer-directed treatment (HR=0.90; 95% CI 0.86-0.94) compared to those with the most material resources. We did not detect a significant difference in the rate of palliative care based on community level material resources (HR=1.04; 95% CI 1.00-1.08). **Conclusions:** We identified inequities in access to cancer-directed consultation and treatment among adults with stage IV NSCLC by material resources. Future research should explore root causes and identify interventions to address these inequities.

LAY SUMMARY

People with advanced lung cancer living in neighborhoods with the fewest material resources (such as income, quality housing, and education) were less likely to see a cancer specialist than those in neighborhoods with more material resources, but only in urban areas. People with advanced lung cancer living in neighborhoods with the fewest material resources were less likely to receive treatment for their advanced lung cancer than those in neighborhoods with more material resources, but just as likely to receive palliative care. Although our findings are similar to those in other countries with publicly funded healthcare systems, further research to understand the reasons underlying these inequities is needed.

Health Disparities and Equity

Monday, November 3, 2025

Impact of hysterectomy correction on estimated cervical cancer screening rates and cervical cancer incidence rates: A systematic review - Poster #306

Presenter: Yuba Raj Paudel, AHS

Other Authors: Nana Ama Adjabeng, Anjola Adeboye, Kamala Adhikari

ABSTRACT_ID#3058

ABSTRACT

Introduction The main purpose of this systematic review was to synthesize evidence on the impact of hysterectomy correction on the estimation of cervical cancer screening participation rate (CCSPR) and cervical cancer incidence rates (CCIR).

Methods We conducted a search using five databases and grey literature. Two reviewers independently conducted title and abstract screening, followed by full text review. We used a pre-determined and piloted data extraction tool. We used Joanna Briggs Institute's (JBI) quality appraisal tools for evaluating quality of included studies. We narratively synthesized data and compared corrected estimates based on socio-demographic factors. **Results** We included 21 studies in the review. Overall, correction for hysterectomy increased CCSPR (increase range: 0.7% to 13%) and CCIR compared to the uncorrected rates (increase range: 5% to 59%). The increase in CCSPR was greater among people aged >50 years compared to women aged <50 years. Similarly, the increase in CCIR was greater in those aged >60 years compared to those < 60 years such that older age categories had a comparable rate after correction. Hysterectomy correction also shifted the age for peak CCSPR and CCIR. Few studies showed Black, Hispanic, Indigenous women; and women with lower socio-economic status had high CCIR even after hysterectomy correction. **Conclusion** More longitudinal studies assessing CCSPR and CCIR in the same population are needed to inform the implementation of effective strategies to improve the use of cancer screening services. Future studies need to report hysterectomy corrected CCSPR and CCIR from an equity perspective.

LAY SUMMARY

Exclusion of individuals with total hysterectomy due to benign reason is important to accurately estimate cervical cancer screening participation (CCSPR) and cervical cancer incidence rate (CCIR). Limited published studies have estimated hysterectomy corrected CCSPR and CCIR. Reporting uncorrected rates masks the underlying inequalities in screening participation and cancer incidence across sub-populations and may lead to misaligned resource allocation. Future studies need to report hysterectomy corrected CCSPR and CCIR from equity perspective to inform program planning and decision making.

Health Disparities and Equity

Sunday, November 2, 2025

Facilitating equity-oriented care in organizations delivering cancer care: Insights from a qualitative study - Poster #307

Presenter: Tara Horrill, University of Manitoba

Other Authors: Jess Crawford, Scott Beck, Amber Bourgeois, Jagbir Kaur, Leah Lambert, Michael McKenzie, Annette Browne, Kelli Stajduhar

ABSTRACT_ID#2785

ABSTRACT

Background: People experiencing structural marginalization often have advanced cancer diagnoses and higher cancer-related mortality resulting, in part, from poor access to care. A key pathway to addressing health outcome disparities includes integrating equity-oriented approaches to cancer care (EOCC), referring to care that is tailored and seeks to reduce the impacts of structural inequities (e.g., poverty, systemic racism). Implementing equity-oriented approaches can help address access, quality of life and care gaps. However, despite growing interest in EOCC, little is known about the organizational factors required to support the delivery of EOCC; our study addressed this gap. **Methods:** Informed by social justice and intersectional perspectives, and critical ethnographic methods, we employed a participatory and integrated knowledge translation approach. We conducted semi-structured interviews with health and social service providers and key informants, observations of service providers in oncology settings, and interviews and focus groups with people experiencing structural marginalization, in collaboration with a community partner. **Findings:** Our findings point to three dynamic and intertwined dimensions within organizations delivering cancer services that support an equity-oriented approach to care: identifying core values for creating an equity-oriented culture (e.g. relationality, adaptability), building a shared commitment to EOCC, and taking action towards EOCC. **Implications:** Our findings highlight the important role of shared organizational values and formalized commitments to equity, providing direction for integrating equity-oriented care at the organizational level. Findings also emphasize the importance of attending to organizational contexts as playing a key role in supporting clinicians to provide tailored, person-centered, and culturally safe-r care.

LAY SUMMARY

Organizations delivering cancer services can make important contributions to health equity. Equity-oriented approaches to care are one pathway to addressing challenges of access to cancer care, experiences of cancer care, and cancer outcomes. This student demonstrated the importance of organizational values in guiding equity work and making meaningful organizational commitments to equity. Organizational values and commitments to health equity create the conditions in which healthcare providers can deliver care that is patient-centered and equity-oriented. Values and commitments to equity need to be paired with tangible actions within the organization and meaningful accountability measures.

Health Disparities and Equity

Monday, November 3, 2025

Hysterectomy-corrected cervical cancer screening participation in Alberta and its variation across sub-populations - Poster #308

Presenter: Yuba Raj Paudel, AHS

Other Authors: Gary Teare, Kamala Adhikari

ABSTRACT_ID#3059

ABSTRACT

Introduction: Inadequate exclusion of individuals with a history of hysterectomy can underestimate cervical cancer screening participation rate (CCSPR). We aimed to improve the accuracy of Alberta's provincial estimates for CCSPR by excluding women with total hysterectomy due to benign reasons from the screen eligible population. We also aimed to discover disparities in CCSPR across subpopulations. Methods: We identified women with total hysterectomy due to benign reasons using health administrative data. We calculated hysterectomy corrected cervical screening participation rates. We stratified the population into two age strata (25-49 and 50-69) while estimating screening participation rate by geography, migration status and ethnicity. Findings: Correction for hysterectomy increased CCSPR by 3% among women aged 25-69 years (uncorrected: 57.3%, corrected 60.4%). Upon correction, peak participation shifted from 30-39 years to 40-59 years. The correction for hysterectomy narrowed disparity by urban/rural place of residence. Among those aged 25-49 years, inter-provincial migrants (Corrected: 55.6 vs uncorrected: 54.3%) and international migrants (Corrected: 58.6% vs uncorrected: 58.0%) had lower screening participation than non-immigrants (Corrected: 67.0% vs uncorrected: 64.4%). Similarly, the Asian population (Corrected: 57.6% vs uncorrected: 57%) and the Black population (Corrected: 60.2% vs uncorrected: 58.5%) showed a lower screening participation compared to the White (Corrected: 62.0% vs uncorrected: 59.9%) and the Hispanic population (Corrected: 63.1% vs uncorrected: 61.8%). Conclusion: Applying hysterectomy correction improved provincial estimates for screening participation. The findings provide data-driven insights needed to refine analytics and screening plans, enhance equity, and optimize outreach efforts to improve screening uptake in the province.

LAY SUMMARY

Exclusion of individuals with total hysterectomy due to benign reason increased estimates for screening participation rate by 3% overall. Available data in the province can be used to estimate the hysterectomy corrected screening rate. Women aged 40-59 years show higher screening participation after correction compared to women <40 years reflecting a true scenario of participation after correction for prevalence of hysterectomy. Hysterectomy corrected screening participation rate provides important information for planning of interventions to enhance screening uptake and reduce inequity.

Health Disparities and Equity

Sunday, November 2, 2025

A flexible parametric relative survival model with spatio-temporal random effects to evaluate inequalities in population-based cancer survival in Ontario - Poster #309

Presenter: Todd Norwood, Ontario Health (Cancer Care Ontario)

Other Authors: Shabnam Balamchi, Xinyi Ge, Lindsay Obress, Zeinab El-Masri, Simron Singh, Jill Tinmouth, Prithwish De

ABSTRACT_ID#2978

ABSTRACT

Background: Cancer survival is an important measure of treatment effectiveness but estimating local variation to identify inequalities remains challenging. We developed a statistical model to evaluate how patient demographics, diagnostic period, and socioeconomic factors contribute to local disparities in population-based cancer survival. Methods: We used five-year relative survival to capture population-based all-cause mortality among people with cancer, adjusted for expected survival via the period method. We extended a previously developed flexible parametric relative survival (excess mortality) model by incorporating spatial and temporal random effects using a Leroux conditional autoregressive structure. Local areas were defined by amalgamating Census Tracts and Census Subdivisions across Ontario. We compared proportional hazards and proportional odds specifications for the baseline cumulative hazard using model fit statistics. Model selection evaluated baseline cumulative hazard complexity (splines) and covariate inclusion to balance fit and parsimony. Full Bayesian inference was implemented using OpenBUGs software, with sensitivity analyses for priors. The approach was applied to nine cancer types (1999–2018); colorectal cancer is presented as a case study. Results: The proportional odds specification with ≥ 2 spline terms consistently outperformed proportional hazards. Covariates for increasing rurality/remoteness and material deprivation were consistently associated with higher excess mortality. Spatial clustering of areas having a higher adjusted odds of excess mortality persisted across time. For colorectal cancer, the final model included five splines and showed strong local area correlation ($p = 0.90$, 2014–2018) in adjusted excess mortality odds. Conclusions: This spatio-temporal model quantifies inequalities in cancer survival, providing granular, geographic information for targeted cancer control.

LAY SUMMARY

Recent statistical advancements enable population-based estimation of relative survival among people with cancer at a local level. We propose a flexible spatio-temporal relative survival model that identifies local variation in excess mortality and evaluates its associations with rurality and marginalization. Model results (a) showed that complex baseline mortality hazards during five-year follow-up times were typical and (b) provided reliable local area estimates of adjusted relative survival for common (e.g., colorectal) and less common (e.g., kidney) cancers.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

A population-based study of the diagnosis to treatment initiation duration and overall survival for stage I non-small cell lung cancer treated with stereotactic body radiation therapy - Poster #311

Presenter: Cassidy Laub, Queen's University

Other Authors: Paul Nguyen, Matthew Jalink, Timothy P. Hanna

ABSTRACT_ID#3070

ABSTRACT

Background: In Ontario, lung cancer is amongst the most diagnosed cancers and leading cause of cancer-related deaths. For medically inoperable stage I non-small cell lung cancer (NSCLC), stereotactic body radiation therapy (SBRT) is recommended. However, the optimal diagnosis to treatment initiation (DTI) duration for SBRT remains unclear. This study investigated the association between DTI duration and overall survival (OS) in patients with stage I non-adenocarcinoma NSCLC treated with SBRT in Ontario. **Methods:** A population-based retrospective cohort study was conducted using linked ICES health administrative datasets. The cohort included Ontario patients with pathologically diagnosed stage I NSCLC between January 1, 2010, and June 30, 2019, who received curative SBRT. Patients with adenocarcinomas were excluded due to histological diversity not controllable within the available data. Kaplan Meier analysis and multivariable Cox proportional hazards models were used. Cubic splines were used to assess linearity of the exposure in the Cox model. **Results:** Our cohort included 532 patients with a median DTI duration of 61 (interquartile range: 46-79) days. Kaplan Meier analysis revealed a trend showing longer DTI durations were associated with worse OS. There was no evidence of a more complex non-linear relationship between DTI duration and OS. Multivariable Cox models indicated every 1-week (aHR=1.04 (95% CI=1.01-1.08)), and 4-week (aHR=1.19 (95% CI=1.03-1.38)) increase in DTI duration was associated with worse OS. Findings were robust in sensitivity analysis. **Conclusions:** These findings suggest that the DTI duration should be as short as reasonably achievable for stage I non-adenocarcinoma NSCLC patients receiving SBRT.

LAY SUMMARY

This study is the first to investigate the association between time from diagnosis to treatment initiation and overall survival in patients with stage I non-adenocarcinoma non-small cell lung cancer treated with stereotactic body radiation therapy. We found that longer diagnosis to treatment initiation durations were associated with worse overall survival in this population. We recommend that stage I non-adenocarcinoma non-small cell lung cancer patients should initiate stereotactic body radiation therapy as soon after diagnosis as reasonably achievable.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Mixed Methods Evaluation of Implementation Outcomes, Barriers, and Facilitators of an Electronic Patient-Reported Outcome System for Cancer Rehabilitation - Poster #312

Presenter: Christian Lopez, Princess Margaret Cancer Centre

Other Authors: Sarah Neil-Sztramko, Kristin Campbell, David Langelier, Gillian Strudwick, Jackie Bender, Jonathan Greenland, Tony Reiman, Jennifer Jones

ABSTRACT_ID#2681

ABSTRACT

Background: REACH is an electronic patient-reported outcome (ePRO) system that remotely monitors patients' rehabilitation needs during and after treatment, delivering support including links to self-management education and community programs, and recommendations for further evaluation. We aimed to evaluate the implementation of REACH at four Canadian centres for four disease sites (breast, colorectal, head and neck, and lymphoma) and understand barriers and facilitators to implementation. Methods: A 16-month formative mixed methods evaluation was conducted, guided by the Implementation Outcomes Framework and the Consolidated Framework for Implementation Research. Quantitative data included system analytics and patient experience surveys. Qualitative data were collected through focus groups with patients and interviews with clinic leadership and point-of-care staff. Results: Of 722 patients registered, 610 (67% female, 44% breast, median age 57 (21–91), 45% receiving treatment) consented for research. REACH was feasible (median assessment time: 3 minutes; 73% of patients reported ease of use) and showed moderate engagement (99% of patients completed ≥ 1 assessment; 39% of all assessments completed; 68% viewed ≥ 1 recommended resource). Key themes influencing implementation were organizational capacity to prioritize new initiatives, alignment and integration with digital systems, onboarding processes ensuring timely and clear introduction of REACH within clinical workflows, and perceptions of REACH's value and usability. Conclusions: Immediate delivery of tailored resources after ePRO completion was highly valued; however, implementation was hindered by limited integration with organizational decision-making, digital systems, and patient education during clinical encounters. Embedding REACH into a comprehensive digital platform at each centre is needed to support sustained patient engagement.

LAY SUMMARY

REACH is an online tool that helps people living with and beyond cancer report their symptoms and rehabilitation needs from home. After filling out short questionnaires, patients get immediate access to a personalized resource library tailored to their needs. This library provides links to 1) education resources such as videos, websites, and reading materials; 2) programs such as online or in-person classes and workshops led by trained professionals; and 3) suggestions to follow-up with a health care provider for further evaluation or referral. Over 600 patients across four cancer centres used REACH and nearly all completed at least one questionnaire. Most found it easy to use, and many appreciated having a personalized, central place where they could find resources to help with their rehabilitation and symptom management. Successful implementation depended on how well REACH fit into each clinic's existing systems and workflows. Challenges included limited involvement and coordination with clinic leadership, organizational planning, and other patient digital tools, as well as limited time during appointments to introduce REACH to patients.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Drugs for Advanced Renal Cell Carcinoma: A Treatment Pattern Analysis - Poster #314

Presenter: Christie Farrer Rogers, University of Calgary

Other Authors: Reka Pataky, Qi Guan, Mariet Mathew Stephen, Nicola Bai, Katharina Forster, Winson Cheung, Samara Strub, Safiya Karim, Steven Yip, Mina Tadrous, Stuart Peacock, Kelvin Chan

ABSTRACT_ID#3000

ABSTRACT

Background: Renal cell carcinoma (RCC) is the most common type of kidney cancer and is often diagnosed at advanced stages, making treatment challenging. While recent clinical trials show the benefits of combining immune checkpoint inhibitors with VEGF inhibitors, real-world data on their use and treatment durations remain limited. Understanding these patterns is crucial for optimizing clinical guidelines and planning healthcare resources. **Methods:** This study examined systemic treatment patterns for advanced RCC in three Canadian provinces: Ontario, Alberta, and British Columbia. We used population-based data from 2017-2023 to analyze treatment sequences, durations, and dosing for patients receiving publicly funded therapies. **Results:** Amongst 2,224 patients, the average age at diagnosis was 67 years and the majority of patients were male. Since their introduction, combination ipilimumab plus nivolumab and combination axitinib plus pembrolizumab have dominated as the first treatment option (first-line treatment) for advanced RCC, almost entirely replacing the older drugs: pazopanib and sunitinib. The average duration of first-line treatment ranged from 10 to 13 months, while second- and third-line therapies were generally shorter, ranging from six to eight months. **Discussion:** These findings help illustrate how patients move through treatment lines, offering insights for funders on evolving therapy use. As new drugs enter the market, this data can inform budget planning and policy decisions. These results may also support conversations between funders and clinicians when new therapies become available. Finally, the results highlight the need for further research into the clinical and economic implications of ongoing shifts in the advanced RCC treatment landscape.

LAY SUMMARY

Renal cell carcinoma (RCC) is the most common type of kidney cancer and is often diagnosed at advanced stages which makes treatment challenging, therefore understanding drug treatment patterns is crucial for optimizing clinical guidelines and planning healthcare resources. We looked at treatment patterns in 3 Canadian provinces over a 7 year period to find patterns in drug utilization. Our findings help illustrate how patients move through treatment lines, offering insights for funders on evolving therapy use. As new drugs enter the market, this data can inform budget planning and policy decisions. These results may also support conversations between funders and clinicians when new therapies become available. Finally, the results highlight the need for further research into the clinical and economic implications of ongoing shifts in the advanced RCC treatment landscape.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Dynamic Prediction of Length of Active Treatment in Cancer Care Using Deep Learning Models in Alberta, Canada - Poster #315

Presenter: Kenneth Lai, Alberta Health Services

Other Authors: Grace Heemeryck, Cheliger Cheliger, Maeve Wickham, Emily Walker

ABSTRACT_ID#2949

ABSTRACT

Background: With treatment advances and improved survival, time in cancer care is increasing. Accurate prediction of care duration is critical for effective health system planning. We dynamically quantified predictors of active treatment duration (ATD) across major tumour groups in Alberta, Canada, using electronic medical records (EMR). Method: We leveraged Dynamic DeepHit, a deep-learning survival model. All incident cancer cases between 01/01/2010-15/02/2024 were included. ATD was defined as the interval between the first oncologist visit and the last cancer-related encounter (treatment/consultation), death, or end of the study period. We included pre- and post-diagnosis features (comorbidities (physician claims, inpatient and outpatient records), imaging/labs, cancer, sociodemographic, and treatment) in time-varying monthly format. The models generated discharge probabilities at prespecified landmarks (baseline, 3/6/12 months). The models' performances were evaluated using the time-dependent concordance index on a held-out test set. Results: We included diagnoses of breast (n=29,465), genitourinary (n= 20,988), gastrointestinal (n=22,936), hematological (n=15,725), and gynecological (n=10,747) cancers. Predicted median ATD (months) ranged from 5.2 (95%CI:5.1-5.3) to 12.6 (95%CI:11.9-14.1) in hematology and gastrointestinal cancers, respectively. Variable importance differed markedly between tumor groups and over time. Cancer characteristics such as stage, grade and cancer site, and survival probabilities for both cancer-related and non-cancer-related causes, stratified by tumor group and calendar year, were the strongest determinants of ATD at baseline. Treatment-related features gained importance at months ≥ 3 . Performance ranged from 0.71-0.82 at baseline and generally improved over time. Conclusions: Models trained on EMR data can produce dynamic, accurate, and site-specific ATD predictions, subsequently informing capacity planning.

LAY SUMMARY

Dynamic Prediction of Treatment Duration: We used a deep learning survival model (Dynamic DeepHit) to dynamically predict active treatment duration (ATD) for cancer patients across five major tumor groups in Alberta, using detailed, time-varying electronic medical record data. Variable Importance Over Time: The most important predictors of ATD at baseline were cancer characteristics (e.g., stage, grade, site) and survival probabilities for the specific tumor group at the year of diagnosis. Treatment-related factors became more influential in predicting ATD after the initial 3 months, with importance varying significantly across tumor types and over time. Model Performance and Planning Utility: The model showed strong predictive performance (concordance index: 0.71–0.82) which improved over time, demonstrating its potential to support healthcare system capacity planning with accurate, dynamic, and site-specific predictions of active care duration.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Defining and measuring the value of genetic testing from patients' perspectives: Developing the Patient-reported Genetic testing Utility InDEx (P-GUIDE) for precision oncology - Poster #316

Presenter: Yiming Wang, The Hospital for Sick Children

Other Authors: Robin Hayeems, Elise Poole, Stephanie Luca, Daniel Assamad, Bowen Xiao, Joyce Yan, Lesleigh S. Abbott, Tristan Charlinski, Avram Denburg, Rebecca J. Deyell, Bailey Gallinger, Catherine Goudie, Anne-Marie Laberge, Sarah L. Sawyer, Rosemarie E. Venier, Anita Villani, Wendy J. Ungar, P-GUIDE Study Team, Linlea Armstrong, Kym M. Boycott, Monica Bronowski, Emily Carelli, June C. Carroll, Lauren Chad, Alison M. Elliott, Fatemeh Emari, Emilie Espagne, Jessica Leclerc-Blain, Iskra T. Peltekova, Becky Quinlan, Maureen Smith, Elisabeth Soubry, Grace Tam

ABSTRACT_ID#2699

ABSTRACT

Purpose: Determining the value of genetic testing requires a set of metrics, including the patient perspective. This study aims to develop the Patient-reported Genetic testing Utility InDEx (P-GUIDE), a novel outcome measure for precision oncology. **Methods:** Informed by the literature, a list of 48 items was generated. Cognitive interviews were conducted with parents of children who received germline and/or somatic genetic test results to provide item feedback. Item relevance and clarity were scored by interviewees and clinical experts to assess content validity. Prospectively, a new sample of parents completed an updated 45-item tool to support item reduction using Bayesian exploratory factor analysis. **Results:** Twenty-one parents were interviewed (95% mothers). Based on interview feedback, 12 items were removed, 10 were added and 33 were revised. Item relevance and clarity scores provided by 21 parents and 9 clinical experts prompted further modification of 15 items. Bayesian exploratory factor analysis of P-GUIDE responses from 83 parents (81% mothers) supported a three-factor solution (posterior probability = 0.917; Bayes factor = 0.642). Based on factor loadings and cross-loadings, 13 items were removed, and one item was added, resulting in a revised 33-item tool. Standardized factor loadings ranged from .54 to .91, and inter-factor correlations were low ($r = -.005$ to $.27$), suggesting well-differentiated constructs. Further item reduction and validity testing are underway. **Conclusion:** Once validated, P-GUIDE will constitute a standardized measure of personal utility for cancer genetic testing, enhancing the voices of parents of children with cancer in health technology assessments, policy, and funding recommendations.

LAY SUMMARY

Patients and their families are interested in new genetic testing technologies that can help determine the genetic cause of and/or the best way to treat childhood cancer. Currently, there is no standardized way to measure how parents value genetic testing for childhood cancer. To address this gap, we are developing an outcome measure called the Patient-reported Genetic testing Utility InDEx (P-GUIDE), for use in pediatric precision oncology. Through multiple rounds of interviews and surveys with parents and clinicians, we are learning that parents value genetic testing in many different ways, such as learning about cancer risk, using the information to help facilitate conversations with healthcare providers, family, and community, and providing peace of mind. Once validated, P-GUIDE will help to inform patient-centered care, policy decisions and funding recommendations related to genetic testing for children with cancer.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Caring for older adults with cancer: Developing a geriatric assessment toolkit for oncology nurses - Poster #318

Presenter: Fay Strohschein, University of Calgary

Other Authors: Allison Wiens, Brandy Vanderbyl, Erika Martinez, Lauren Cosolo, Kristen Haase, Lorelei Newton, Martine Puts

ABSTRACT_ID#3269

ABSTRACT

In Canada, 62% of cancer cases occur in those ≥ 65 years, and 31% in those ≥ 75 years. Geriatric assessment (GA) is recommended to inform decision-making and supportive interventions among older adults undergoing cancer treatment; however, geriatric oncology consultation services in Canada are limited. Oncology nurses are well-positioned to address age-related concerns impacting cancer care but often lack the knowledge and resources to do so, contributing to care delivery issues and ethical concerns. To develop a bilingual resource of previously validated GA tools that oncology nurses can implement in clinical practice, the Canadian Association of Nurses in Oncology (CANO) Oncology and Aging Special Interest Group collaborated with the International Society of Geriatric Oncology (SIOG) Nursing, Allied Health, and Scientists (NAHS) Interest Group, establishing a working group of nurses with clinical/academic expertise in geriatric oncology. Tool selection was informed by current guidelines, usability, and validated translation availability. CANO members provided feedback on initial drafts. CANO education/professional practice committees and SIOG NAHS/publication committees reviewed final drafts. Caring for Older Adults with Cancer: A Toolkit for Oncology Nurses includes GA tools across nine domains, highlighting their relevance within cancer care, and a care-planning pathway, potential nursing interventions, a template to identify local services, and educational resources. Tailored to the Canadian context, this toolkit is a new resource to support translation of evidence relating to assessment/management of age-related concerns into oncology nursing practice. Future directions include development of workshops and a national virtual community-of-practice to support education and implementation, with active evaluation informing ongoing improvement.

LAY SUMMARY

Almost half of new cancer cases occur in Canadians aged 70+ years, a number expected to nearly double in the next 30 years. Oncology nurses are well-positioned to address age-related concerns that can impact cancer treatment and care, but they often lack the knowledge and resources to do so. A working group of nurses with expertise in care of older adults with cancer collaborated to develop a toolkit for oncology nurses with information about how to assess and manage concerns common among older adults with cancer, including those related to patient goals and values, ability to engage in day-to-day activities, mobility and falls, comorbidities, sensory impairment, social support and resources, cognition and memory, mood, nutrition, and medication management. This toolkit is a new resource to translate evidence about the care of older adults with cancer into oncology nursing practice. Use of the toolkit will be supported by educational workshops and national virtual meetings to develop a community of oncology nurses working to promote implementation.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Measuring the cost-savings and survival benefits of the CancerCare Manitoba (CCMB) Quit Smoking Program (QSP) - Poster #319

Presenter: Carrie O'Conaill, CancerCare Manitoba

Other Authors: Katie Galloway, Kristie Morydz, Zeb Aurangzeb, Lin Xue, Kelly Brown, Donna Turner

ABSTRACT_ID#2670

ABSTRACT

Objectives: Smoking cessation after a cancer diagnosis improves survival and treatment effectiveness, yet 20% of patients continue smoking. The Quit Smoking Program (QSP) at CancerCare Manitoba (CCMB), implemented in 2012, provides cessation support to cancer patients. This study examines treatment costs and survival benefits for individuals who accessed QSP versus those who smoked but did not enroll. **Methods:** A retrospective matched cohort study analyzed cancer treatment costs and survival benefits for individuals diagnosed with cancer who accessed QSP from January 2012-June 2018 (n=360) with those who reported smoking but did not access the program (n=357). Costs related to cancer care and all-cause healthcare utilization were assessed. Analyses were stratified by cancer type (thoracic, head and neck, colorectal, and breast cancer). **Results:** Overall, there was a cost-savings of \$4,134 per individual for QSP participants compared to non-QSP participants. The cost savings per individual were highest for individuals diagnosed with thoracic cancer (\$9,583), then breast cancer (\$2,003) and head and neck cancers (\$1,036). There were no cost savings for individuals diagnosed with colorectal cancers (-\$2,187). The probability of survival was higher for QSP participants compared to the probability of survival for non-QSP participants (HR: 1.16, 95% CI: 0.93-1.45, p-value = 0.1883), particularly for those diagnosed with thoracic cancer (HR: 1.29, 95%: 0.99-1.68, p=0.0581). **Conclusion:** Findings suggest significant cost-savings and survival benefits associated with QSP participation reinforcing the value of smoking cessation programs in improving patient outcomes and reducing healthcare costs. Further research with larger samples is needed to build on this research.

LAY SUMMARY

Quitting smoking after a cancer diagnosis can improve survival and lower healthcare costs. Many cancer patients continue to smoke, but those who receive support through the Quit Smoking Program (QSP) see better health outcomes and lower healthcare costs. The QSP saves money and helps patients live longer. People who joined QSP saved an average of \$4,134 each in healthcare costs. Savings were highest for thoracic cancer patients (\$9,583). Survival rates were also higher for QSP participants, especially those with thoracic cancer. This program benefits both patients and the healthcare system. By helping patients quit smoking, QSP improves treatment effectiveness, survival rates, and long-term health, making it a valuable tool in cancer care.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Assessing Quality of Life Among Canadian Multiple Myeloma Patients and Caregivers - Poster #320

Presenter: Gabriele Colasurdo, PeriPharm

Other Authors: Julie Patenaude, Ariane Légaré, Gabriele Colasurdo, Martine Elias, Catherine Beauchemin, Jean Lachaine

ABSTRACT_ID#2926

ABSTRACT

Background: Multiple myeloma (MM) imposes a substantial burden, affecting patients' quality of life (QoL) and that of caregivers, who often lack psychological support. Real-world data on QoL remain limited despite recent therapeutic advances that have altered the disease course. Methods: A cross-sectional, observational study was conducted across Canadian provinces in MM patients and caregivers of MM patients to assess their QoL. Participants were identified through Myeloma Canada's database and data were collected using the PROxy Network web-based platform. All data were self-reported by patients or caregivers and included demographic and disease-related characteristics. Patients completed the EORTC QLQ-C30, EORTC QLQ-MY20, EQ-5D-5L, and ESAS-R. Caregivers completed the CarGOQoL. Results: A total of 305 patients (49.8% female; mean age: 65.6 years) and 104 caregivers (74.0% female; mean age: 61.6 years) participated in the study between October 2024 and February 2025. 27.2% of patients and 32.7% of caregivers reported that the patient had experienced a relapse of MM. Patients reported a mean Global Health/QoL score of 65.68 on the EORTC QLQ-C30. On the EORTC QLQ-MY20, the mean disease symptoms and side effects of treatment scores were 23.22 and 22.36, respectively. The mean total distress score on the ESAS-R was 18.33, and the mean EQ-5D-5L utility value was 0.80. Caregivers reported a mean CarGOQoL index score of 62.14. Conclusion: Advances in MM treatment have extended survival and changed side effect profiles, which may contribute to a moderately impaired QoL in patients. These changes may also impact caregivers, potentially altering the burden of care.

LAY SUMMARY

Canadian patients with multiple myeloma report moderately reduced quality of life, with a similar burden from disease symptoms and side effects of treatment. Caregivers of Canadian multiple myeloma patients also experience impaired quality of life due to the demands and burden of caregiving.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Enhanced Recovery After Surgery (ERAS) Early Mobilization Compliance and Post-Operative Outcomes: A Retrospective Cohort Study Analysis - Poster #321

Presenter: Charanpal Singh, University of Tennessee, Knoxville

Other Authors: Chad Wagoner, Abby Thomas, Joseph Dort, Gregg Nelson, Khara Sauro

ABSTRACT_ID#2717

ABSTRACT

Background Enhanced Recovery After Surgery (ERAS) guidelines include early mobilization (EM), which involves moving as soon as possible after surgery. EM is associated with better outcomes in patients with head and neck cancer. However, its implementation across surgical populations with or without cancer has not been well described. This analysis assessed EM compliance across surgery sites and its association with postoperative outcomes. Methods ERAS-guided surgeries in Alberta, Canada, between 2017 and 2021 were included. EM compliance (Yes/No) was assessed across surgery types (Breast, Colorectal, Gynecology, Head and Neck, Liver, Pancreatic, and Urology). Postoperative outcomes included hospital LOS and complications. A Pearson chi-squared test was used to compare EM compliance between cancer status. Adjusted generalized linear models (GLMs) assessed associations between EM compliance (exposure) and postoperative outcomes, with cancer status as a covariate. Results About 45% (n=3642) of patients received a cancer diagnosis. EM compliance for those with a cancer diagnosis (50%; n=1786) was significantly lower than that of those without a diagnosis (56%; n=2438; $p<0.001$). Both crude and adjusted GLMs showed that EM was associated ($p<0.001$) with reduced hospital LOS (IRR, 0.78; 95% CI, 0.76-0.79) and reduced odds of experiencing complications (OR=0.41; 95% CI, 0.38-0.45). Conclusions EM compliance was lower for those who had received a cancer diagnosis. Regardless of diagnosis, being compliant with EM was associated with shorter hospital stays and lower odds of experiencing complications, highlighting its importance across populations. Multilevel interventions are needed to address patient-level physical function and system-level tracking and reporting for improved compliance.

LAY SUMMARY

Individuals diagnosed with cancer had lower overall compliance with ERAS-recommended early mobilization. Early mobilization compliance for individuals diagnosed with cancer was 50% compared to 56% of individuals without a cancer diagnosis. Early mobility after surgery is associated with a better recovery, regardless of cancer diagnosis. Despite low compliance, early mobilization clearly improves important postoperative outcomes, such as hospital length of stay and complications. There's room to improve. To boost early mobilization, future efforts should focus on both patient-level supports (like improving physical function) and system-level changes (like better tracking and reporting of compliance).

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Identifying a definition of clinical complexity for follow-up visits using unsupervised clustering - Poster #322

Presenter: Grace Heemeryck, Alberta Health Services

Other Authors: Cheliger Cheliger, Maeve Wickham, Kenneth Lai, Ali Manouchehrinia, Emily Walker

ABSTRACT_ID#2920

ABSTRACT

INTRODUCTION Clinical complexity in cancer patients affects variation in the appointment length of follow-up visits, influencing how clinics schedule care and allocate resources. However, no accepted definition of patient complexity during follow-up visits currently exists. We applied unsupervised clustering to clinical data to identify distinct, clinically meaningful patterns of patient follow-up visit complexity. **METHODS** We included breast cancer patients receiving treatment at Cancer Care Alberta facilities from January 2010 to February 2024. Follow-up visits with a medical oncologist were selected if the patient completed a Patient Reported Outcomes form. We applied HDBSCAN's soft clustering algorithm to assign each visit a membership probability across clusters. Clustering variables, selected with healthcare practitioner input, included symptom severity, treatment changes, and number of tests following the visit. Visits with low membership probability (probability<0.5) to the nearest cluster were deemed low-confidence clusters. We used Cohen's effect size to quantify differences in clustering variables between high- and low-confidence clusters. **RESULTS** We analyzed 24433 visits (n=5986) and consulted 36 healthcare practitioners for variable definitions. Preliminary analyses yielded 6 clusters with 54% of visits assigned to low-confidence clusters. Compared to high-confidence clusters, low-confidence clusters had higher symptom severity and more post-visit clinical activity (Cohen's d=0.5-1.0), suggesting these visits have more complexity than their high-confidence counterparts. **CONCLUSION** Clustering revealed distinct subsets of visits that do not conform to common patterns, offering a data-driven path toward defining clinical complexity in cancer follow-up visits. Further analyses and clinical consultations are required for cluster validation and refining the clinical complexity definition.

LAY SUMMARY

Through consultation with 36 healthcare practitioners, we identified variables related to breast cancer patients' clinical complexity during follow-up visits to medical oncologists in Cancer Care Alberta facilities. These included treatment changes (new chemotherapy medications or dose changes), the numbers of diagnostic imaging events, lab tests, and visits to supportive care, and self-reported patient symptoms. We extracted this data from patient electronic medical records. Using HDBSCAN, a density-based unsupervised clustering algorithm, we identified six clusters of follow-up visits for breast cancer patients in active treatment, based on the variables identified by healthcare practitioners. These clusters can be used to create definitions of visit complexity. Each cluster was separated into "high-confidence" and "low-confidence" subsets based on each visit's probability of being assigned to its cluster. Each cluster's "low-confidence" subset had at least three variables with increased values (Cohen's effect size>0.5) in comparison to the high-confidence subset. In all clusters, this included increases in at least one of the patient's self-reported values for anxiety, depression, pain, mobility concerns, and overall symptom severity. This suggests the low-confidence subset of a cluster contains visits for patients with more concerns than the high-confidence subset, offering further insights for complexity of visits within a cluster.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Economic Burden of Radon-Attributable Lung Cancer in Canada - Poster #323

Presenter: David C. Griffin, University of Calgary

Other Authors: Isaiah E. MacDonald, John M. Danforth, Dustin D. Pearson, Sarah Mazher, Rui Fu, Syed Irteza Abbas Shamsi, Mariet M. Stephen, Ned Liu, Matthew T. Warkentin, Eldon Spackman, Darren R. Brenner, Amanda AW. Gibson, Michelle L. Dean, Vishal Navani, Winson Cheun

ABSTRACT_ID#3004

ABSTRACT

Lung cancer is the leading cause of cancer diagnoses in Canada, accounting for 1 in 4 deaths. 2 in 5 cases are attributed to non-tobacco causes such as genetics and/or exposure to radioactive radon gas and combustion particulates. Of these, radon is ubiquitous and long-term inhalation increases lung cancer lifetime risk by 16% per 100 Bq/m³. Residential radon exposures are at historic highs due to building practices and increased telecommuting. High radon exposure is preventable via building modification, and lung cancer screening offers earlier diagnosis for people whose risk can be assessed. However, there are economic challenges to both of these, including costs of building mitigation and for developing technologies to evaluate personalized risk. An economic rationale is required but lacking, as current lung cancer cost estimates are out-of-date due to evolving standards of care. Advanced non-small cell lung cancer (NSCLC) is the most common non-tobacco diagnosis. We calculated the modern economic burden of lung cancer care utilizing current disease management costs. Cost of management of Stage IV NSCLC from diagnosis to end-of-life is \$215,026 per case, contrasted with \$15,366 per case for Stage I. Based on the 2024 Cross Canada Survey of Radon and life activity pattern data, population attributable risk estimates indicate that 4,216 lung cancers per year are caused by radon, resulting in a total cost of radon-attributable NSCLC of \$798,760,102 per year in Canada.

LAY SUMMARY

New oncologic advances have significantly increased the cost of treating NSCLC with prolonged survival and rising prices of novel therapies. Advanced (stage III-IV) lung cancer is associated with the greatest increase in costs, and continues to be most common diagnosis amongst non-smokers who develop lung cancer. Using population attributable risk estimates, we calculated the economic burden of 1 year of NSCLC treatment to be \$798,760,102.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Ranking Decision-Making Criteria for Early Adoption of Innovative Surgical Technologies - Poster #324

Presenter: Haitham Shoman, Concordia University

Other Authors: Nisha Almeida, Michael Tanzer

ABSTRACT_ID#2594

ABSTRACT

There is no decision-making framework in the early-adoption stage of novel surgical technologies, putting the quality of health-care and resource allocation of the health-care-system at risk. Objective: To investigate relevant weighted criteria that decision-makers may use to make informed decisions for the early adoption of innovative surgical technologies. Methods: This multi-institutional decision analytical modeling study used a mixed-methods multicriteria decision analysis (MCDA) and had 2 phases. First, a panel of 12 experts validated decision criteria in the literature and identified additional criteria. Second, 33 Canadian experts prioritized the main criteria (domains) using the composition pairwise-comparison weight-elicitation method (analytical hierarchy process model) and ranked their subcriteria using the direct-ranking elicitation method (Likert scale). Data were analyzed, and response consistency was estimated using consistency ratio. ANOVA was used to assess for significant differences between expert responses. Results: 45 experts were invited with different levels of education and years of experience. Seven domains and 44 subcriteria were identified. The MCDA model found that clinical outcomes had the highest priority vector, at 0.429, followed by patients and public relevance (0.135). Hospital-specific criteria (0.099), technology-specific criteria (0.092), and physician-specific criteria (0.087) were the next most highly ranked. The lowest priority vectors were for economic criteria, at 0.083, and finally policies and procedures, (0.075). There was consensus in the responses (consistency ratio = 0.006), and there were no statistically significant differences between expert responses. Putting these criteria into a framework may help surgeons and decision-makers make informed decisions for the early adoption of new surgical technologies.

LAY SUMMARY

Findings In this decision analytical modeling study, clinical outcomes were the most important criteria to guide surgeons and nonsurgeons in the early adoption of new surgical technologies. Responses from surgeons and nonsurgeons were not statistically significantly different, suggesting a similar priority-setting criteria among all stakeholders. **Meaning** These findings suggest multiple prioritized and weighted criteria, which may be indispensable in making informed decisions in the early adoption of new surgical technologies to judiciously use financial resources and maximize patient outcomes. **Main Outcome and Measure:** Criteria domain weights and subcriteria rankings. Priority vectors, which are priority scores analyzed and prioritized from expert responses, were used to rank criteria domains and subcriteria for decision-making on adopting new innovative surgical technologies.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Travel Distance and Its Impact on Wait Time for Positron Emission Tomography–Computed Tomography in Patients with Cancers - Poster #325

Presenter: Dat Tran, Institute of Health Economics

Other Authors: Xiaoxiao Liu, Alka Patel, Rizwan Shahid, Maki Ueyama

ABSTRACT_ID#2324

ABSTRACT

Objectives: To examine travel distance and its impact on wait time for Positron emission tomography–computed tomography (PET/CT) in patients with lung, lymphoma, and prostate cancers in Alberta. **Methods:** We used Alberta cancer registry and diagnostic imaging database to identify patients with lung, lymphoma, and prostate cancers who had a PET/CT scan during April 2017 and March 2023. Alberta Facilities Distance/Time Look Up Table was used to calculate travel distance from patient’s residence to PET/CT facility. Negative binomial regression was used to assess association between travel distance and wait time for PET/CT. **Results:** The study included 9,503 patients. Lung cancer accounted for 43.4% of patients, followed by lymphoma (37.1%) and prostate (19.5%) cancers. There were more females with lung (55.5%) than lymphoma (42.9%; $p<0.001$) cancers. Mean (SD) age was 66.8 (13.8) years and lymphoma patients were younger (59.6 years) than lung (70.3 years; $p<0.001$) or prostate (72.7 years; $p<0.001$) patients. Diabetes (14.2%) was the most prevalent comorbidity. Median (IQR) travel distance was 21 (12-121) km and was shorter for urban (16 km) than rural (148 km; $p<0.001$) patients, but wait time was similar (median=20 vs. 21 days; $p=0.378$). There were no significant associations between travel distance and wait time (IRR=1.00; $p=0.108$). The results were robust in six subgroup analyses by type of cancer and scan priority. **Conclusion:** There were no associations between travel distance and wait time for PET/CT. Additional research is warranted to examine the potential impact of longer travel distance on overall access to care and patient outcomes.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Cancer System Performance in Canada: Indicator Refresh Process - Poster #326

Presenter: Corinne Daly, Canadian Partnership Against Cancer

Other Authors: Jessica Kitchen, Kim Tran, Farah McCrate, Natalie Fitzgerald

ABSTRACT_ID#2365

ABSTRACT

Objective To reach consensus and identify an updated set of standardized indicators to compare cancer system performance across Canadian jurisdictions. **Methods** From 2008-2018, the Canadian Partnership Against Cancer (CPAC) worked with partners across Canada to release annual cancer system performance reports. After a break, in which other types of reporting were pursued, an indicator refresh was initiated in Fall 2023. Work included scoping reviews, in-person meetings with the System Performance Steering Committee, and numerous virtual engagements with clinician experts, advisory groups, networks and patient, family and caregiver partners. Consensus building exercises and four modified Delphi consensus surveys were used to identify themes spanning the cancer control continuum, and to prioritize indicators for pan-Canadian comparative analyses. **Results** The final list includes approximately 70 core and optional, pilot indicators, as well as equity stratifications spanning the following themes: long-term outcomes, prevention, screening, diagnosis, treatment, palliative and end-of-life care, research, person-centred perspective, and for the first time, survivorship and system sustainability. A parallel engagement and co-creation process is also underway with First Nations, Inuit and Métis partners to report progress towards People's-specific, self-determined priorities. **Conclusion** Data collection is underway, and results will be released starting in 2026 on CPAC's new interactive, customizable data dashboard on partnershipagainstcancer.ca. This pan-Canadian analyses will provide health system leaders, decision makers and researchers with comparable data on key components of the cancer care continuum, highlighting gaps, and offering actionable insights to drive improvements in cancer prevention and care, including for equity-denied and First Nations, Inuit and Métis communities.

LAY SUMMARY

This work aims to identify standardized indicators to measure and compare cancer system performance across jurisdictions and over time. Literature reviews, in-person and virtual engagements, as well as surveys and consensus building activities were used to identify and prioritize indicators across the following themes: long-term outcomes, prevention, screening, diagnosis, treatment, survivorship, palliative and end-of-life care, research, person-centred perspective and system sustainability. A parallel engagement and co-creation process is also underway with First Nations, Inuit and Métis partners to report progress towards People's-specific, self-determined priorities. This pan-Canadian analyses will help to identify gaps in care, promote continuous improvement, and inform evidence-based policy, planning and practice changes to ultimately improve patient access, experience and outcomes.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Challenges and Stakeholder Perspectives in the Uveal Melanoma Referral Process: A Cross-Sectional Mixed Methods Study - Poster #327

Presenter: Emily Laycock, University of Calgary

Other Authors: Ezekiel Weis, Joakim Siljedal, Trafford Crump

ABSTRACT_ID#2610

ABSTRACT

Purpose Uveal melanoma (UM) is a rare but deadly eye cancer with a 15-year mortality rate of 45%. Timely referrals to specialists (i.e. ocular oncologists) are critical, as early detection and treatment are essential to improving patient outcomes. The objective of this study was to characterize the perspectives of various stakeholders involved in the referral process for suspected UM. **Methods** Using a cross-sectional mixed-method design, three stakeholder groups from across North America were surveyed and/or interviewed: ocular oncologists, primary eye care providers (optometrists and ophthalmologists), and patients. **Results** Fifty-one stakeholders participated, including 18 ocular oncologists, 21 primary eye care providers, and 12 patients. Ocular oncologists reported that 38% of UM patients are referred later than ideal, with 22% having a poor prognosis upon initial evaluation. Fifty percent indicated that inadequate referral information, such as lack of high-quality imaging and presence of risk factors, lead to difficulties in triaging patients. Less than 20% of primary eye care providers felt “very confident” in differentiating between low- and high-risk lesions. They expressed uncertainty regarding where to send referrals and noted that there is a shortage of ocular oncologists needed for monitoring patients with suspicious eye lesions. Patients highlighted issues such as delayed diagnosis, and extensive travel and eye care costs. **Conclusion** The results of this study suggest that there are delays in the referral process of uveal melanoma. The outcomes of this research could help inform future interventions aimed at improving this process for all stakeholders.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Integrating a Provincial Cancer Prevention Initiative in Alberta Using a Learning Health System Framework - Poster #328

Presenter: Kamala Adhikari, Alberta Health Services and University of Calgary

Other Authors: Kimberly Manalili, Gary Teare

ABSTRACT_ID#2998

ABSTRACT

Background: The Integrating Prevention into Connect Care for Health (IPiC-Health) initiative embeds a Screening, Brief Intervention, and Referral (SBIR) approach into the Alberta-wide clinical information system ("Connect Care") to address key cancer risk factors: tobacco use, alcohol use, and physical inactivity. This paper outlines how the Reid et al. 2024 framework is applied to facilitate a Learning Health System (LHS) approach for integrating SBIR as routine care in acute and ambulatory settings. **Methods/approaches:** Health system gaps in SBIR implementation are identified, and solutions are co-designed with patients and providers. Implementation science approaches, along with the project staff embedded in clinical sites, guide implementation, regular evaluations, and use of data for improvement. Research team and implementation support system provide ongoing supports (tools, training, technical assistance, and quality improvement) to the clinical team, guided by Interactive systems framework. **Results:** SBIR has been implemented at nine clinical sites, with 150 providers trained in the SBIR process. Over 7,000 patients have been screened in Connect Care on at least one of the three risk factors, with 3601 identified as medium/high risk and 531 receiving brief advice. Ongoing interviews with providers and patients on their SBIR-related experience, regular engagement with clinical sites/leaderships/champions, and rapid assessment reports on the progress of SBIR delivery have created a strong feedback loop and accelerated integration. Strong alignment of research, evaluation, and implementation activities enhanced the responsiveness, feasibility, and effectiveness of the integration. **Conclusions:** LHS approach facilitates continuous improvement and evidence-informed, sustainable integration of prevention-focused cancer care in healthcare settings.

LAY SUMMARY

Strategies targeting key cancer risk factors (tobacco use, alcohol use, and physical inactivity) can help prevent cancer and improve outcomes of those diagnosed with cancer. The Integrating Prevention into Connect Care for Health (IPiC-Health) initiative incorporates the Screening, Brief Intervention, and Referral (SBIR) approach into the Alberta-wide clinical information system ("Connect Care") to address these modifiable risk factors. A Learning Health System (LHS) approach has supported the integration of SBIR into routine care across acute and ambulatory settings by enabling continuous learning and improvement. This has been achieved through implementation supports, regular data use, feedback and adaptation, and ongoing stakeholder engagement.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Economics of prostate cancer in Canada: focus on advanced stages - Poster #329

Presenter: Félix Morin, Université de Montréal

Other Authors: Meïssa Bougchiche, Ivan Yanev, Alice Dragomir

ABSTRACT_ID#2824

ABSTRACT

INTRODUCTION: Prostate cancer (PCa) is the most common cancer and the third leading cause of death from cancer among Canadian men. Since 2012, new treatments with higher costs have been implemented in Canada, increasing the financial pressure of PCa management. This study aims to review the scientific literature identifying management costs of PCa in Canada. **METHODS:** A systematic review was conducted across Embase, MEDLINE, CINHAL, Web of Science and grey. Eligible studies included studies presenting direct or indirect Canadian costs of PCa management. Study selection, data extraction and quality assessment were conducted independently by two reviewers. Costs were standardized to 2024 Canadian dollars using the Statistic Canada's Consumer Price Index for health and personal care. **RESULTS:** 30 studies were retrieved and analyzed with 9 addressing advanced stages. Every study used administrative data, and some supplemented these data with RCT or published literature data. The studies cover every advanced PCa health states (mHSPC, nmCRPC and mCRPC) and cover a wide variety of treatment like ADT (5 studies, cost ranging from \$4,206 for 2 years to \$191,896 for 15 years), radiation therapy (2 studies, cost ranging from \$11,948 for 1 year to \$34,301 for 1 year) or abiraterone (2 studies, cost ranging from \$3,790 for 28 days to \$412,414 for 15 years). **CONCLUSION:** This is the first systematic review of Canadian costs of PCa management since 2013. The literature remains sparse on advanced stages; we therefore recommend more cost studies and economic analysis to be conducted to bridge the knowledge gap.

LAY SUMMARY

This systematic review aims to describe prostate cancer management costs in Canada 30 studies were retrieved and analyzed with 9 of them addressing advanced prostate cancer. There is a need for more cost analyses and economic evaluations on advanced prostate cancer management costs in Canada.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Can alcohol policy prevent cancer? A modeling study estimating the potential impact of alcohol minimum unit pricing and alcoholic beverage labeling on cancer incidence and mortality in Canada - Poster #330

Presenter: Keegan Lawrence, Canadian Institute for Substance Use Research

Other Authors: Adam Sherk, James Clay, Tim Stockwell, Erin Hobin

ABSTRACT_ID#2551

ABSTRACT

Alcohol is a Group 1 carcinogen responsible for a sizeable share of Canada's cancer burden. In our study using the International Model of Alcohol Harms and Policies, we simulated how two interventions—minimum unit pricing (MUP) and alcohol warning labels—could reduce alcohol-caused cancer incidence and mortality in Canada. We used nationally representative survey, sales, and cancer registry data to estimate the baseline cancer counts of cancer incidence and mortality. Then, we tested how five alcohol policies combining two label designs (a cancer-specific warning and a rotating multi-message series) and two MUP thresholds (\$1.75 and \$2.00 per standard drink) could impact cancers of the breast, larynx, pancreas, liver, colorectum, esophagus, and oral cavity. Reductions in alcohol use were drawn from real-world evaluations and modified by income- and sex-specific price elasticities. All scenarios led to reduced consumption and cancer burden. A \$2.00 MUP alone lowered incidence and mortality by over five percent, with the strongest effect seen when combined with cancer labeling, which was estimated to prevent approximately one in fourteen cases and one in seventeen deaths. Benefits were largest among men and people in lower income quintiles, who suffer disproportionately from alcohol-related harms. This suggests such policies could both reduce overall cancer burden and help close persistent health equity gaps. Our findings highlight the value of pairing pricing and labeling interventions for cancer prevention. As Canada considers stronger alcohol regulations, these results offer timely evidence to guide public health policy and legislative action.

LAY SUMMARY

Alcohol causes cancer, but most people don't know it. Alcohol is a known carcinogen, like tobacco or asbestos, yet public awareness is low. Our study shows that warning labels on alcohol containers can help reduce alcohol use and raise awareness. Raising the minimum price of alcohol works. Policies that set a minimum price per drink (called minimum unit pricing) are especially effective at reducing alcohol consumption, particularly among those who drink the most. When fewer people drink heavily, fewer people get alcohol-related cancers. Policy change can prevent illness and save lives. Combining clear cancer warning labels with a \$2.00 minimum price per drink could prevent hundreds of new cancer cases and deaths each year in Canada. These changes would benefit everyone, but especially lower-income Canadians, who are more affected by alcohol harms.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Towards Comprehensive Cancer Prehabilitation: A Qualitative Investigation to Inform the Design of Patient-Centred Programs for Prostate Cancer - Poster #331

Presenter: Hannah Harsanyi, Cancer Epidemiology & Prevention Research, Alberta Health Services

Other Authors: Nicole Slot, Yun Lee, Ashley Housten, M. Eric Hyndman, Lin Yang

ABSTRACT_ID#2554

ABSTRACT

Background: Prehabilitation, which helps patients physically and mentally prepare for treatment, is traditionally investigated among patients undergoing surgery. A direct translation of surgical prehabilitation programming may not apply to patients with cancer, who undergo various treatments beyond surgery. This study aimed to inform the design of prehabilitation programs in oncology settings through exploring patient perspectives on prehabilitation before prostate cancer treatment. Methods: Semi-structured interviews were conducted with men diagnosed with prostate cancer in Alberta, Canada. Interviews were thematically analyzed, and themes were characterized to identify patient-perceived barriers and facilitators to prehabilitation engagement according to the Theoretical Domains Framework (TDF) and Capability, Opportunity, Motivation- Behaviour (COM-B) model. Recommendations for program design were generated following the steps of Intervention Mapping. Results: Interviews were conducted with 34 participants, of whom 26% were managed by active surveillance, 44% underwent primary surgical treatment, and 29% were pursuing non-surgical treatment. Facilitators to engaging in prehabilitation included connecting with other patients/survivors, desire to improve treatment-related side effects, and need for support navigating the psychological impact of diagnosis. Barriers included lack of familiarity with prehabilitation, logistical concerns, and stigmatized perceptions of mental health support. Recommendations informed by these determinants include beginning prehabilitation early, providing personalized, evidence-based prehabilitation interventions, and creating options for virtual participation to increase accessibility. Conclusions: Our findings provide a guide for designing cancer prehabilitation programs which are relevant to and could benefit patients across the range of prostate cancer treatment modalities. Future research is needed to develop evidence-based interventions effective beyond surgical settings.

LAY SUMMARY

Prehabilitation, or prehab, aims to prepare patients before they start treatment with the goal of improving physical and mental health and getting ready for the journey ahead. By interviewing patients diagnosed with prostate cancer, we wanted to create recommendations for designing cancer prehab programs. We talked to patients who were planning to or had undergone different types of treatment, including surgery, radiation therapy, hormonal therapy, or active surveillance, where they were "watching-and-waiting" rather than receiving treatment immediately. We found that patients were generally interested in participating in prehab. They viewed the opportunity to connect with other patients and survivors as an important reason to join. They also wanted to take part in activities that would help to reduce the side effects from their treatment. It was seen as important to communicate about what prehab was, as well as why it is important, as many patients were not familiar with the idea of prehabilitation. Based on the stress of receiving a cancer diagnosis and challenges making decisions about treatment options, we recommend that prehab should start as soon as possible after diagnosis. Our findings will help to design prehab programs which are meaningful and well-received by patients diagnosed with cancer.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Agreement between Canadian Provincial and Territorial Cancer Registry questionnaire and registry data regarding impact of varying date of diagnosis collection methods on Canadian cancer surveillance measures - Poster #332

Presenter: Lorraine Shack, Statistics Canada

Other Authors: Lorraine Shack, Jonathan Simkin, Laci Davies, Ron Dewar, Kim Vriends, Joshua Mazuryk, Gail Gail Gautreau, Jennifer Gushue, Chris Tremblay, Owen Phillips

ABSTRACT_ID#2980

ABSTRACT

Background Accurate data collection is paramount for understanding the cancer burden in Canada. However, variations in date of diagnosis (DOD) data collection among Provincial and Territorial Cancer Registries (PTCR) have been identified by the Canadian Council of Cancer Registries (CCCR) has been identified as an area requiring further study. Purpose To compare PTCR perspectives on variation in DOD collection methods across PTCRs, with resultant analysis of statistical impact on surveillance measures. Understanding the collection and classification issues from different perspectives will significantly enhance the accuracy and comparability of cancer surveillance data across Canada. Methodology A mixed-methods approach was used. Qualitative methods involved a literature review. Structured questionnaire-based interviews with 13 PTCRs took place to clarify DOD practices and identify perceptions regarding impact of these practices and on cancer surveillance outcomes. Quantitative net survival analysis was then conducted at 3-months, one-year and five-year intervals for 11 cancer sites using age-standardized net Canadian Cancer Registry survival data. Predicted net survival was evaluated using life table and relative survival regression models. Confirmatory sensitivity analysis was also conducted. Results Outcomes from interview questionnaires were compared with statistically determined cancer survival estimates. Agreement between perceived impact and actual statistically determined survival estimates was high. Cancer survival estimates for pancreatic, lung and kidney cancer suggest that variations in DOD collection practices partly explain observed survival differences. Conclusion This work supports efforts to improve enhancing the quality of cancer surveillance data. Ultimately, these efforts aim to advance the comparability and reliability of cancer data collection in Canada.

LAY SUMMARY

1. Accurate data collection is paramount for understanding the cancer burden in Canada. However, variations in date of diagnosis (DOD) data collection among Provincial and Territorial Cancer Registries (PTCR) have been identified by the Canadian Council of Cancer Registries (CCCR) as an area requiring further study.
2. Outcomes from interview questionnaires were compared with statistically determined cancer survival estimates. Agreement between perceived impact and actual statistically determined survival estimates was high. It was agreed that variations in DOD collection practices impact net survival estimates in certain cancer sites and contribute to inter-jurisdictional differences. Poor-Prognosis Cancers: For sites like pancreas and lung, DOD collection variations explain a part of the observed survival differences. Cancers with Varied Diagnostic Pathways: For kidney cancer (including renal pelvis), where diagnostic pathways and watchful waiting/active surveillance practices involve longer timeframes to obtain a date of histological verification, survival estimates are influenced by these practices. Sensitivity Analysis (Lung Cancer - Ontario Example): Further confirmed the impact of earliest DOD on the comparability of net survival.
3. This work supports efforts to improve the consistency across PTCRs and enhancing the quality of cancer surveillance data. Ultimately, these efforts aim to advance the comparability and reliability of cancer data collection in Canada.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Ensuring timely patient access to complex cancer therapies: identifying and addressing gaps in health system implementation readiness - Poster #333

Presenter: Judith Glennie, J.L. Glennie Consulting Inc.

Other Authors: Robert Bick

ABSTRACT_ID#2727

ABSTRACT

Finding ways to accelerate patient access to new therapies has been a key focus of both government and private sectors for many years. Health Canada, Canada's Drug Agency, and the pan-Canadian Pharmaceutical Alliance have made efforts to accelerate their processes. Ontario's Premier is concerned about Canada lagging behind other OECD countries regarding timely patient access, making this a priority issue for the Council of the Federation. With complex therapies, achievement of timely patient access needs to consider factors beyond traditional processes. Timely patient access goes beyond the funding decision. Approval of funding for new cancer treatments is only one step in the process. Timely funding can only positively impact patient outcomes if health system integration and actual utilization in patients also occur in a timely manner. The implementation needs associated with complex cancer therapies should be identified early and addressed based on a system-wide planning approach, along with engagement of and collaboration amongst all stakeholders. There is an opportunity to be innovative in addressing challenges related to the integration of complex therapies into the health system, and at the same time ensuring equitable access for patients no matter where they reside. This presentation will provide an overview of research findings that make the case for more proactive implementation planning for complex therapies, so that actual use in patients is not delayed after the provincial funding decision. Insights and potential solutions identified through collaborative engagement with patient groups, health system leaders, and clinicians will be explored.

LAY SUMMARY

Federal and provincial governments are looking at ways to accelerate access to new therapies to eliminate barriers to timely treatment of patients. Provincial funding of new cancer therapies can only positively impact patient outcomes if health system integration and utilization in patients also occur in a timely manner. Solutions are needed to enable proactive health system implementation planning, so that use of new complex cancer therapies is not delayed after provincial funding decisions.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Exploring High Acute Care Utilization Among Individuals Living with and Beyond Head and Neck Cancer - Poster #334

Presenter: Megan Patton, University of Calgary

Other Authors: Mudra Dave, Benny Rana, Paul Ronksley, Kirsten Fiest, Khara Sauro

ABSTRACT_ID#3307

ABSTRACT

Background: Head and neck cancer (HNC) is the 7th most common cancer worldwide. Patients with HNC often face long-term issues from the disease and its treatment, potentially leading to increased healthcare resource use compared to those without HNC. However, the relationship between HNC diagnosis and increased healthcare resource utilization is not well understood. Objective: To explore healthcare resource utilization among individuals living with and beyond HNC compared to those without HNC. Methods: This retrospective cohort study included adult patients with HNC, identified in the Alberta Cancer Registry. To compare those with HNC to those without we identified a cohort of patients of similar age and sex who had at least 1 hospital admission. The cohort was deterministically linked to Discharge Abstract Database, National Ambulatory Care Reporting System, and eCritical to explore healthcare resource use. The primary outcome was high healthcare resource utilization (≥ 3 ED visits or ≥ 30 days in hospital within a year). Logistic regression was used to explore the association between a diagnosis of HNC and high healthcare use, while controlling patient demographic characteristics. Results: A total of 4,511 patients were included in the cohort, with 2,206 (48.9%) diagnosed with HNC. In the crude model, HNC was associated with lower odds of being a high healthcare user (OR: 0.84; 95% CI: 0.7321–0.9551; $p = 0.001$). Conclusion: Patients with HNC had lower odds of being high healthcare users than those without HNC. Further research is needed to explore healthcare delivery gaps, continuity of care, and unmet needs among this population.

LAY SUMMARY

Contrary to assumptions regarding cancer-related healthcare burden, hospitalized patients with HNC had lower odds of being classified as high healthcare users than those without HNC. This finding may point to potential underutilization of services or barriers in access to care for patients with HNC. The definition of high healthcare users in this study may not capture the nuances of healthcare utilization observed across the care spectrum for patients with HNC (e.g., access to primary care and specialist services). This study demonstrates the need for future research to explore healthcare resource utilization among patients with HNC, particularly focusing on exploration of experiences, health systems, and tailored healthcare models that consider the continuity of care for individuals living with and beyond HNC.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Implementation of a Clinical Care Pathway for Triple Negative and HER2+ Breast Cancer Patients - Poster #335

Presenter: Julia Downey, University of Calgary

Other Authors: Rabia Raheel, Roger Tsang, Sasha Lupichuk, May Lynn Quan, Alison Laws

ABSTRACT_ID#3105

ABSTRACT

Background: Neoadjuvant chemotherapy (NAC) is recommended for stage II-III HER2+ or triple negative (TN) breast cancer, yet most patients are referred to surgery before medical oncology (MO), potentially delaying treatment. We hypothesized that a novel multi-disciplinary care model could reduce time to NAC. **Methods:** In March 2024, we implemented a city-wide clinical care pathway that triages stage II-III HER2+/TN breast cancers to expedited MO referral and staging scans before surgical consult. We aimed for 90% initiating NAC ≤ 5 weeks from referral. We used the RE-AIM framework to evaluate pathway delivery in the first year, comparing to a pre-pathway cohort from 2023. **Results:** We identified 111 patients on pathway with 29 participating providers. Successes included excellent adoption by patients and strong adherence to intended consult sequence (95%). Challenges included unavailable biomarkers at time of triage (86%), delaying referrals to MO (median 1.6 weeks, goal ≤ 3 days). Furthermore, pre-consult ordering of investigations was inconsistent (63%). The median time to NAC was 5.6 weeks pre-pathway vs. 5.1 weeks on pathway ($p = 0.01$). The proportion initiating treatment within 5 weeks was non-significantly lower on pathway (42% vs. 32%, $p=0.14$); delays to treatment of ≥ 7 weeks were significantly reduced (16% vs. 30%, $p=0.02$). There was a non-significant trend toward decreasing time to NAC following pathway implementation. **Conclusion:** While the pathway significantly reduced time to NAC, the effect was small, and many patients did not initiate treatment within the 5-week target. Biomarker availability was the biggest barrier, and future efforts will focus on this element.

LAY SUMMARY

- We evaluated a new care pathway intended to shorten the time to chemotherapy for patients with aggressive breast cancer, aiming to improve outcomes by reducing treatment delays.
- The new process slightly reduced time to treatment. However, many patients still experienced delays, often because test results needed to inform treatment weren't available in time.
- Future efforts will focus on reducing barriers to get patients on treatment faster.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Development of a Framework for the Comprehensive Evaluation of Virtual Cancer Care - Poster #336

Presenter: Melanie Powis, Princess Margaret Cancer Centre, University Health Network

Other Authors: Lisa Barbera, Matthew Cheung, Randy Conrad, Mary DeVera, Annemarie Edwards, Sophia Esmail, Helen McTaggart-Cowan, Robert Olson, Stuart Peacock, Ambreen Sayani, Simron Singh, Anum Ali, Jacelyn Salmini, Saidah Hack, Monika Krzyzanowska

ABSTRACT_ID#2753

ABSTRACT

While there has been a rapid uptake of virtual visits in oncology in recent years, understanding of its impact on the quality of cancer care is fragmented due to the high degree of heterogeneity in the evaluation measures used to date, and no existing comprehensive framework to guide the work. Our aim was to develop a comprehensive evaluation framework for virtual cancer care through a process of ideation, synthesis and consensus. We undertook nominal consensus groups stratified by key informants type (knowledge users and policy makers, clinicians, and patients), to generate a list of potential measures to evaluate virtual care from different perspectives. To identify measures from the health equity perspective, we undertook a structured consultation with an established health equity expert patient and caregiver panel. Findings were thematically grouped by an expert panel through a cognitive mapping exercise using a virtual whiteboard. Virtual cancer care-specific initial construct definitions were derived first from a systematic review of the peer-reviewed literature, then iterated on and finalized through cycles of discussion, and voting until consensus was reached. The resultant framework consisted of 17 constructs across three domains: implementation (usability, appropriateness, feasibility [human resources, capacity, infrastructure, medical-legal], sustainability, adoption, fidelity), service (efficiency, safety, effectiveness, patient-centeredness [continuity/coordination, quality of the interaction, supportive care], timeliness, climate) and outcome (satisfaction, experience, acceptability, preferences, clinical outcomes), with equity (utilization, access) and costs as separate, cross-cutting domains. This work will help to guide future virtual cancer care evaluations to better inform policy or guide practice optimization.

LAY SUMMARY

Cancer care is rarely delivered as strictly virtual or in-person, and so the hybrid nature of care must be considered in operationalizing the framework for practice. Additionally, there are patient populations and clinical scenarios for which in-person care is not necessarily the gold standard. Measurement priorities varied by key informant group, highlighting the need to consider the impacts of virtual cancer care from multiple stakeholder perspectives. Our emergent framework was consistent with the Proctor Outcomes for Implementation Research framework adapted for virtual cancer care, but emphasizes the need to consider equity and cost as separate, over-arching domains.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Creating Capacity to Implement Smoking Cessation Support Across Regional Cancer Centres in Ontario: Findings from a Rapid Review, Qualitative Interviews, and Delphi Panel - Poster #338

Presenter: Nadia Minian, CAMH and University of Toronto

Other Authors: Isabella DeVuono, Reza Zandi, Wayne deRuiter, Scott Veldhuizen, Laurie Zawertailo, Michael Chaiton, Lawson Eng, Osnat Melamed, Matthew George, Michelle Halligan, Caroline Silverman, Archie Stewart, Lester Krames, MaryAnn Bradley, Sarwar Hussain, Adina Cor

ABSTRACT_ID#2878

ABSTRACT

Although smoking cessation is a critical component of cancer care, its implementation across oncology settings remains inconsistent. This study identified strategies to improve smoking cessation support across Ontario's Regional Cancer Centres (RCCs) using a sequential mixed-methods design: a rapid systematic review, qualitative interviews with healthcare providers (HCPs), and a modified Delphi panel involving HCPs and patients. From the systematic review, we identified several implementation strategies associated with increased screening, advising, and referral to smoking cessation treatments, including training and education, continuous evaluation, personalized support, and inter-professional collaboration. Interviews with 27 HCPs revealed that in emotionally complex situations, particularly with patients in palliative care, providers often avoid cessation discussions, believing that patients are not interested in receiving support. HCPs also expressed that existing training does not adequately prepare them for sensitive conversations about smoking. Systemic barriers, such as time constraints, role ambiguity in prescribing, and fragmented care pathways, were seen as major impediments to the consistent delivery of cessation interventions. In a two-round Delphi process with 12 panellists, consensus was reached on four priority strategies: integrating referral responsibilities into clinical roles, increasing the number of local cessation champions, providing free onsite pharmacotherapy, and offering direct-to-home medication delivery. These findings offer actionable, consensus-driven strategies that can guide the integration of smoking cessation support across cancer care settings and help improve outcomes for people with cancer who smoke.

LAY SUMMARY

Quitting smoking after a cancer diagnosis can improve treatment outcomes, reduce complications, and increase survival, yet patients are not always offered the support they need to quit. This study aimed to find practical ways to improve how smoking cessation support is delivered in cancer care in Ontario. We reviewed existing research, interviewed healthcare providers, and worked with a group of experts—including providers and patients—to identify the most helpful and feasible strategies for Ontario's Regional Cancer Centres. We learned that: Strategies such as provider training, regular feedback, personalized support, and stronger teamwork across roles are linked to more consistent screening, advising, and referrals to quit-smoking services. Many healthcare providers avoid talking about the benefits of quitting smoking with cancer patients, especially in emotional or palliative care situations, because they worry patients are not open to it or they do not feel confident having those conversations. Healthcare providers want better training and clearer systems so they know whose role it is to address smoking and how to do it in a supportive, respectful way. Experts believe that offering free medications onsite, delivering medications to patients' homes, and integrating smoking cessation into regular clinical roles would improve the system

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Improving Pathways to Cancer Rehabilitation: Refining, Validating, and Implementing the Cancer Rehabilitation and Exercise Screening Tool (CREST) - Poster #339

Presenter: Lauren Capozzi, BC Cancer

Other Authors: Jenna Sim, Jaclyn Lee, Julia Daun, Aude-Marie Foucaut, David Langelier, Fadee Abboud, S. Nicole Culos-Reed, Khara Sauro

ABSTRACT_ID#3130

ABSTRACT

Introduction: Despite over 65% of patients reporting functional impairments related to their cancer or associated treatments, routine screening and indicated rehabilitation referrals are lacking. The Cancer Rehabilitation and Exercise Screening Tool (CREST) was designed to identify impairment and inactivity to support referral to appropriate resources. Refinement and validation of CREST are needed before it is implemented into clinical practice. Thus, this study aims to refine, validate, and identify barriers and facilitators to implementing CREST nationally. **Methods:** A three-phase study is underway. Phase 1 uses a modified Delphi technique with knowledge users (e.g., clinicians, rehabilitation specialists, patient partners, researchers) to come to consensus on the CREST items. Phase 2 will assess construct validity of the refined CREST. Patients will complete the CREST alongside existing exercise, physical function, and activity of daily living questionnaires, and the agreement between measures will be examined. In Phase 3, knowledge users will complete a survey to identify barriers and facilitators to implementing CREST within clinical practice. **Anticipated results:** The Delphi consensus phase is ongoing, with results expected in July 2025. Phases 2 and 3 will occur in early 2026. This study will result in an end-user developed, validated tool (CREST) that is positioned to be implemented within the cancer care system. **Conclusions:** Implementing streamlined screening and referral tools such as CREST into clinical settings can reduce clinician burden and address unmet care needs among those living with and beyond cancer. This study is a critical first step to standardizing and implementing CREST into cancer care.

LAY SUMMARY

Physical impairment and physical inactivity are common side effects among cancer patients however they are often left undiagnosed and untreated. Integration of standardized screening tools may help facilitate access to rehabilitation and exercise resources, addressing disability and participation restrictions. Optimizing the CREST for future implementation may help bridge this gap within current care.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

One Team, My Team: Evaluation of Team-Based Care on Team effectiveness in Cancer Care - Poster #341

Presenter: Jagbir Kaur, BC Cancer

Other Authors: Leah K. Lambert, Farinaz Havaei, Andy Ma, John Larmet, Nassim Adhami, Dan Le, Ryan Woods

ABSTRACT_ID#2583

ABSTRACT

Background: The British Columbia (BC) government is investing in the implementation of team-based care (TBC) within cancer care to (a) establish/expand multidisciplinary care teams (b) optimize scope of practice and (c) increase team consistency. This study assesses the impact of BC Cancer's new provincial team-based model of care on staff and clinicians through five surveys over a two-year period. **Methods:** The online survey was sent to all TBC staff and clinicians across six BC Cancer centres. The survey includes validated measures examining interdisciplinary interactions, workplace culture, team composition, team consistency, team effectiveness, scope of practice, intention to leave, and quality of care and safety outcomes. Quantitative data was analyzed using descriptive and regression analysis, and qualitative data using content analysis. **Results:** We conducted four surveys between May 2023 and March 2025 with an average responses rate of 40%. Team consistency and scope of practice were significant predictors of team effectiveness. More frequent practice below scope was associated with lower ratings of team effectiveness, while greater team consistency was positively correlated. Workforce retention remains strong despite staffing gaps, with reported reasons for staying including a positive team culture, feeling valued, and the ability to practice to full scope. Factors contributing to team effectiveness include effective communication, enhanced knowledge of each team member's scope of practice, and strong interpersonal relationships. **Conclusion:** Team consistency and role optimization are key factors of team effectiveness in TBC implementation in oncology. Opportunities for improvement include aligning roles, responsibilities, and scopes of practice across disciplines.

LAY SUMMARY

Team consistency and role optimization are key factors of team effectiveness in team-based care model implemented in BC Cancer. Positive team culture and the ability to practice to full scope support workforce retention Continued improvements are needed to align roles and responsibilities across scopes of practice to further optimize team-based care in oncology settings

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Sexual Dysfunction and Healthcare Utilization Among Adults After Cancer Treatment: A Linked Administrative Health-Survey Study in Nova Scotia, Canada - Poster #342

Presenter: Patience Nishimagizwe, Dalhousie University

Other Authors: Lynn Lethbridge, Cynthia Kendell, Robin Urquhart

ABSTRACT_ID#2545

ABSTRACT

Objectives: There is a gap in the literature regarding how adult cancer survivors navigate healthcare services to address sexual dysfunction post cancer treatment. This study aims to assess the association between sexual dysfunction and healthcare utilization in Nova Scotia, Canada, among adult cancer survivors. **Methodology** Adult survivors of breast, prostate, colorectal, melanoma, and hematological cancers diagnosed between 2012 and 2014 were identified from the Nova Scotia Cancer Registry (NSCR). Sexual dysfunction data were obtained from the Transition Study Survey, a pan-Canadian study exploring the experiences of cancer survivors. Survey responses were linked to the NSCR and Medical Services Insurance (MSI) physician billing data to capture healthcare utilization. The final cohort included survivors who completed the survey, had linked administrative data, and were alive and cancer-free during the follow-up period. Multivariate Poisson and negative binomial regression models were used to examine the association between sexual dysfunction and the number of primary care and specialist visits, respectively during treatment post follow-up. **Result** During routine follow-up, adults' cancer survivors with self-reported sexual dysfunction had a 9% higher rate of primary care visits compared to those without sexual dysfunction (IRR: 1.07; 95% CI: 1.06–1.13). However, no significant association was observed between self-reported sexual dysfunction and the rate of specialist visits during follow-up care. **Conclusion** The study showed that adult cancer survivors with sexual dysfunction concerns had a higher rate of primary care visits during follow-up care. These findings highlight an important role of primary care providers in addressing sexual health concerns during follow-up care.

LAY SUMMARY

Sexual health is an important aspect of cancer survivorship, as many adult cancer survivors experience sexual dysfunction after treatment, yet this issue is often overlooked during follow-up care. Adult cancer survivors who reported sexual dysfunction had 9% more primary care visits during follow-up, suggesting they may be actively seeking support for their sexual health concerns. The lack of increase in specialist visits highlights the central role of family doctors and primary care providers in addressing sexual health needs among cancer survivors.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Implementing REmote Symptom mOnitoring and maNagement (RESPONd) in Cancer Care Alberta: Using Patient-Reported Outcomes to Personalize Patient Care - Poster #343

Presenter: Marni Armstrong, Alberta Health Services

Other Authors: Claire Link, Chizoba Anyimukwu, Benedicta Asante, Sheena Morton, April Wales, Andrea Deiure, Linda Watson

ABSTRACT_ID#2573

ABSTRACT

Background In Cancer Care Alberta (CCA), Patient-Reported Outcomes (PROs) are collected at clinic appointments as part of standard practice, using a routine questionnaire to assess symptoms and supportive care needs. PROs personalize symptom management and support to meet each patient's unique needs. Recently, CCA launched an implementation study to examine a remote symptom monitoring and management program called RESPONd, providing patients the opportunity to complete PROs from home between appointments and to explore the impact on patient outcomes and system efficiencies. Methods RESPONd is being implemented over 18 months in six CCA clinics in a stepped-wedge design. Study nurses monitor PROs questionnaires completed between clinic visits and contact patients to provide personalized support. PROs information is also utilized during weekly triage discussions in each clinic, where the route of care is tailored for patients based on their symptom complexity. Impact on Practice The study will explore a person-centered, proactive approach to care, examining the effects on patient outcomes and experience, on clinical teams, and health care utilization. RESPONd represents an innovative opportunity to test a model of care in oncology clinics aiming to optimize efficiency and utilization of limited clinic resources, and create a responsive care team that is ready and able to provide personalized symptom management. Discussion This presentation discusses details of the RESPONd study, focusing on the rationale and anticipated outcomes for patients and care teams, and potential implications for the broader health system. Key learnings and data from the initial study phases will be shared.

LAY SUMMARY

Patient-reported outcomes, such as symptom questionnaires, support personalized care by helping to tailor symptom management to each patient's unique needs. This study examines the implementation of a care model that enables remote symptom monitoring between clinic visits. This presentation will highlight the study design, share preliminary findings, and discuss potential implications for the broader health system.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Healthcare providers' perspectives of the challenges and opportunities for colorectal cancer screening after an abnormal FIT - Poster #344

Presenter: Sharon Mah Mah, Provincial Population Public Health

Other Authors: Jawad Chistie, Amanda Cisar, Kamala Adhikari

ABSTRACT_ID#2750

ABSTRACT

Background Colorectal cancer (CRC) is the third most common cancer globally and in Canada. CRC screening reduces CRC incidence, mortality, and treatment costs through early detection and management. The global literature has little evidence on healthcare providers' (HCP) perspectives of the barriers and facilitators of colonoscopy completion after patients receive an abnormal fecal immunochemical test (FIT+). This study aims to understand HCPs perspectives of the barriers and facilitators to colonoscopy completion after FIT+. **Methods** This is a pragmatic descriptive qualitative inquiry employing the snowball method. HCPs (gastroenterologist, clinic managers, nurses, primary care providers – PCPs) were recruited across Alberta between March to May 2025. Interviews were conducted virtually, video and audio recorded, transcribed, and analyzed using NVivo 15. The Consolidated Framework for Implementation Research (CFIR 2.0) informed the interview guide and analysis. **Results** Sixteen HCPs from across Alberta were interviewed. Reported barriers were patients' fears of painful colonoscopy; limited knowledge of FIT+; and socioeconomic challenges (transportation, parking, and hotels). PCP related barriers were ordering inappropriate FIT for symptomatic, elderly or young patients; lack of resources to support patients' colonoscopy navigation. Health system challenges associated with funding resources matching population need for colonoscopy after FIT+. Reported facilitators were PCP advising patients on FIT+, colonoscopy process, and risks. Additional facilitators were Health system standardizing patient education materials and establishing criteria for accessing colonoscopy after FIT+. **Conclusion** Understanding HCP perspectives on gaps, barriers, and opportunities for enhancing care for patients with an abnormal FIT provides guidance to improve diagnostic colonoscopy and CRC outcomes.

LAY SUMMARY

1. Alberta Health Care Providers (HCPs) perspective of colorectal cancer screening (CRCS) offers invaluable insights on the patient, primary care, and health system factors that impact colonoscopy completion after patients receive a positive fecal immunochemical test (FIT).
2. HCPs reported patients' barriers were fear of the colonoscopy process and preparation; they may not understand the meaning of an abnormal FIT; and those living in rural and remote locations face financial costs associated with traveling (gas, hotels, and parking). HCPs also reported that primary providers (PCPs) ordered inappropriate FITs, may not have staff or resources to help patients navigate the colonoscopy procedure.
3. HCPs reported that the facilitators to colonoscopy completion after a FIT+ were PCP advising patients on colorectal cancer screening after a FIT; and the health system dedicating funding resources to colonoscopy for patients with a positive FIT.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

An evaluation of the association between changes to job protection during illness leave and illness absence behaviour - Poster #345

Presenter: Michael Lebenbaum, Canadian Cancer Society

Other Authors: Ioana Nicolau, Stuart Peacock, Jennifer Gillis, Samuel Asare

ABSTRACT_ID#2811

ABSTRACT

Background: There remains a significant gap in research concerning job protections during illness leaves, which is critical to patients with cancer in Canada since several provinces are considering/passing job-protected leave expansions. We examined three major job-protected leave expansions, Quebec (2003), Manitoba (2016), and Alberta (2018). **Methods:** We used the Canadian Labour Force Survey data spanning from 1998 to 2022. Using a difference-in-difference approach, we examined 5-year changes in leave behaviour before and after expansions in job-protected leave in the three provinces compared to changes in provinces with less than 2 weeks of job-protected leave. Leaves due to any cause of illness were examined. We analyzed the prevalence, duration, and distribution of illness/disability absences using ordinary least squares and linear probability models. **Results:** We found that expanding job-protected leave in Quebec was associated with decreases in the overall length of leave by 2.2 (95% CI: -3.2 to -1.5; $P < 0.001$) weeks or 14.0% relative reduction. Similarly, expansions in Alberta and Manitoba were associated with decreases in the overall length of leave by 1.2 (95% CI: -2.1 to -0.3; $P = 0.016$) weeks or 7.4% relative reduction. Results for absence prevalence were mixed (small increase (Quebec) ($p < 0.05$), no significant change (Alberta-Manitoba) ($p > 0.05$). Both expansions were associated with significant increases in absence duration consistent with the policy (i.e., 3-17-week leaves) ($p < 0.05$). **Conclusions:** Our results suggest that job-protected leave expansion may influence leave behaviour without imposing large additional costs for employers or governments. These findings are important for patients with cancer who often require extensive illness leaves.

LAY SUMMARY

There remains a significant gap in research concerning job protections during illness leaves, which is critical to patients with cancer in Canada due to the lengthy leaves from work they may need to take for cancer treatments. We examined three major job-protected leave expansions in Quebec, Manitoba and Alberta using Labour Force Survey data and statistical methods. We found that expanding job-protected leave in Quebec was associated with small decreases in the overall length of leave and small increases in absences prevalence while Alberta's policy was associated with a small decrease in length of leaves.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Healthcare providers' perspective of implementing screening, brief and intervention and referral for lifestyle risk factors to prevent cancer - Poster #346

Presenter: Sharon Mah, Provincial Population Public Health

Other Authors: Nana A. Adjabeng, Kimberly Manalili, Gary F. Teare, Amanda Cisar, Kamala Adhikari

ABSTRACT_ID#2935

ABSTRACT

Background: Physical inactivity, alcohol, and tobacco use contribute to approximately 40% of preventable cancer cases in Alberta. Alberta Health Services is implementing screening, brief intervention, and referral (SBIR) in Connect Care, electronic health record (EHR) system, across clinical settings to support patients' lifestyle behavior choices. The study aims to understand healthcare providers' (HCPs) SBIR implementation barriers and facilitators. Method: We employed a case studies approach and qualitative descriptions. Interviews were conducted virtually, video and audio recorded, transcribed, and analyzed using NVivo 15. The Consolidated Framework for Implementation Research (CFIR 2.0) informed the interview guide and analysis. Results: Seven HCPs were interviewed. Acute care clinics involved nurses supporting surgical / trauma patients. Nurses reported BI opportunities with patients were limited by information overload at discharge time; gaps in communication flow between nurses limited SBI completion. Reported facilitators included consistent interactions with patients; HCP motivation to connect patients to multi-disciplinary teams; smartphrases in EHR enabled documentation. Ambulatory HCPs are physicians and nurses supporting medically complex patients. Reported barriers included patients' hesitancy to talk about alcohol and tobacco; normalized alcohol use reduced patients' perception of alcohol as a health risk; forgetting to document BI in EHR. Facilitators were: screening was a routine practice prior to SBIR implementation; repeat patient visits increased opportunities to understand patients' readiness to change and identify factors relevant to health challenges. Conclusion: SBIR implementation and adaptations are context specific that reflect ambulatory and acute care settings. Understanding HCP perspectives enables us to improve SBIR process for prevention-oriented cancer care.

LAY SUMMARY

1. 40% of cancers in Alberta are connected to preventable lifestyle changes around physical activity, alcohol and tobacco use. Screening, brief intervention and referral (SBIR) for physical activity, alcohol and tobacco use is an effective way to help patients make changes to their behaviors.
2. Alberta Health Services implemented SBIR in different medical clinics. Healthcare providers (HCPs) reported that some of the challenges they faced were related to patients' unwillingness to talk to them about alcohol and tobacco use. However, over time, patients trusted HCPs to talk about their behaviors. Some of the other challenges with implementing SBIR were connected to the HCPs' work environment which reduced opportunities to SBIR.
3. Understanding HCPs perspectives helps us improve all aspects of SBIR and ensure that it meets the needs of patients and HCPs.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

An evaluation of the use of the Incremental Cost-Effectiveness Ratio (ICER) Thresholds in Canadian Cost-Effectiveness Analysis Studies - Poster #347

Presenter: Michael Lebenbaum, Canadian Cancer Society

Other Authors: Manuela Vieira, Sasha Frost

ABSTRACT_ID#2942

ABSTRACT

Background: Incremental Cost-Effectiveness Ratio (ICER) thresholds are important for determining the funding of cancer and other treatments. They represent the maximum amount a government is willing to pay for health benefits. While previous research has examined trends in ICER thresholds over time in the US, only Canada's Drug Agency submissions have been examined in Canada. Therefore, among Canadian published studies, we examined trends over time in ICER thresholds variation across disease categories. Methods: We included Canadian articles published from 1983 to 2024 in the Tufts Cost-Effectiveness Registry, which compiles data from systematic reviews. The dependent variable was the ICER threshold, measured in dollars per quality adjusted life year (QALY). We conducted descriptive analyses to examine trends in ICER thresholds over time and compared cancer versus non-cancer diagnosis. We will also include results from a multinomial logistic regression and other explanatory variables (pharmaceutical versus other interventions, societal versus health care perspectives). Results: Approximately 70% of studies from 1983 to 2010 and 75% from 2020 to 2024 used an ICER threshold of \$50,000 per QALY. There was no increase in use of ICER thresholds of \$100,000 or more per QALY. Studies focused on cancer were more likely to use \$100,000 per QALY threshold (cancer: 53.6%, non-cancer: 21.6%). Discussion: Unlike the US, most Canadian studies have consistently used an ICER threshold of \$50,000 per QALY, with minimal change over time despite decades of inflation. This suggests that many recommendations may have required a significant price reduction to fund cancer and other treatments.

LAY SUMMARY

Incremental Cost-Effectiveness Ratio (ICER) thresholds are important for determining the funding of cancer and other treatments but there is a lack of Canadian studies. We included Canadian articles published from 1983-2024 in the Tufts Cost-Effectiveness Registry and examined ICER threshold by Cancer and Time. Approximately 70% of studies from 1983 to 2010 and 75% from 2020 to 2024 used an ICER threshold of \$50,000 per QALY and studies focused on cancer were more likely to use \$100,000 per QALY threshold (cancer: 53.6%, non-cancer: 21.6%).

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

The Teleconsultation Experience of Quebec Women with Breast Cancer During the COVID-19 Pandemic: a mixed-method study - Poster #348

Presenter: Sophie Lauzier, Université Laval

Other Authors: Madina Bouziani, Michel Dorval, Josée Savard, Amel Baghdadli, Julie Lapointe, Julie Lemieux, Valérie Théberge, Hermann Nabi, Christine Desbiens

ABSTRACT_ID#3092

ABSTRACT

Background: The COVID-19 pandemic had significant impacts on cancer care delivery worldwide. To ensure continuity of care while protecting patients, teleconsultation was utilized on a broader scale. Objective: This study aims to describe the teleconsultation experience during the COVID-19 pandemic among women in Quebec diagnosed with breast cancer. Method: A cross-sectional analysis was conducted using quantitative and qualitative data from a larger study investigating the impacts of the pandemic, including the use of and satisfaction with teleconsultation. Participants diagnosed with breast cancer in the province of Quebec within the five years preceding the pandemic completed an online questionnaire between May and September 2022. Data were analyzed using descriptive statistics, log-binomial and linear regressions. A subsample also participated in individual semi-structured interviews, analyzed using a thematic approach. Results: Among 138 respondents, 104 (75.4%) had at least one teleconsultation (median: 3; range: 1–30), primarily conducted by telephone (86.5%). Overall, 64.5% reported being satisfied with teleconsultation services, while 17.2% expressed dissatisfaction. Patients undergoing active treatment reported more frequent use and higher satisfaction with teleconsultation. In contrast, those who experienced care disruptions due to the pandemic expressed lower satisfaction. Concerning future implementation, 48.0% favoured maintaining teleconsultation post-pandemic, whereas 51.0% were opposed. The qualitative analysis highlighted the need for a more personalized approach based on a hybrid care model and improved access to videoconferencing. Conclusion: This study comprehensively describes oncology patients' experiences with teleconsultation and provides insights to support decision-making regarding its broader integration into healthcare systems.

LAY SUMMARY

Teleconsultation seems to have been widely used and generally well-received during the pandemic. Treatment status and care disruptions influenced satisfaction. From the patients' perspective, the broader integration of teleconsultations should emphasize a personalized approach based on a hybrid care model.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Associations between the caregiver tax credits and caregiver well-being: a population-based cross-sectional study - Poster #349

Presenter: Michael Lebenbaum, Canadian Cancer Society

Other Authors: Amin Mawani

ABSTRACT_ID#2968

ABSTRACT

Background. Caregiving expenses negatively impact the wellbeing of caregivers, including those who care for patients with cancer. Caregiving tax credits have been implemented to help alleviate some of this burden. However, no studies have examined whether receipt of caregiving tax credits is associated with the wellbeing of caregivers. **Methods.** We used the 2018 General Social Survey Caregiving edition, which is cross-sectional, nationally representative, and focused on caregivers aged 15+ years. Covariates included sociodemographic characteristics, caregiving intensity variables, caregiving recipient characteristics, and other characteristics. We used logistic regression to examine the association between covariates and receipt of the caregiving tax credit and the association between the caregiving tax credit and each outcome. **Results.** We found that individuals with greater caregiving intensity were more likely to receive the caregiver tax credit (30+ hours per week: OR= 1.53; 95%CI: 1.13, 2.08) and individuals caring for a patient with cancer were less likely to receive the credit (OR = 0.62; 95%CI: 0.40, 0.97). The receipt of the caregiver tax credit was associated with lower levels of not coping well with caregiving (OR = 0.70; 95%CI: 0.49, 0.98) but failed to find any associations between caregiver tax credit receipt and self-rated mental health or caregiving hardships ($p>0.05$). **Conclusion.** We find that individuals caring for a patient with cancer are less likely to receive the credit and analyses suggested only modest benefits of the caregiver tax credit. Converting the caregiver tax credit to be refundable and expanding the value of the credit may address these findings.

LAY SUMMARY

Caregiving expenses negatively impact the wellbeing of caregivers, including those who care for patients with cancer, and caregiving tax credits have been implemented to help alleviate some of this burden. We used the 2018 General Social Survey focused on caregivers and statistical methods to examine what predicts receipt of the caregiving tax credit and the relationship between the caregiving tax credit and multiple outcomes. Individuals caring for a patient with cancer were less likely to receive the credit and the credit was only associated with improvements in one of the three outcome variables (i.e., coping well with caregiving).

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

A scoping review of interventions, services and models of care to improve access to cancer treatment for people experiencing social disadvantage - Poster #350

Presenter: Tara Horrill, University of Manitoba

Other Authors: Amber Bourgeois, Morgan Stirling, Noor Breik, Max Kleijberg, Janice Linton, Kate Leahy, Kelli Stajudhar

ABSTRACT_ID#2815

ABSTRACT

Background: Timely and equitable access to cancer treatment are important markers of the quality and accessibility of cancer care, yet socially disadvantaged people experience a range of barriers to receiving cancer treatment, significantly impacting cancer outcomes, including survival and quality of life. **Methods:** We conducted a scoping review to identify and summarize interventions, services, and models of care developed, implemented and evaluated with the aim of improving access to cancer treatment for people experiencing varying forms of social disadvantage. We searched Medline, EMBASE and CINAHL for relevant studies published before 2024; 57 studies were included for analysis. **Findings:** Improving equity and access to cancer treatment is an emerging focus, with nearly half of the included studies published since 2020. Most studies employed an observational design and were conducted in the United States. Various types of cancer treatment were addressed; over half focused on surgical treatment, followed by radiotherapy and systemic therapy. Interventions, services or models of care generally fell into three categories: policies to expand access to health insurance, patient navigation services, and interventions to improve understanding and uptake of clinical trials. Financial/economic disadvantage was most commonly targeted, followed by race or ethnicity. Evaluations demonstrated mixed results, with no specific intervention type definitively improving access to cancer treatment. **Conclusions:** Most interventions focused on financial/economic disadvantage, suggesting a gap in evidence-informed interventions to improve access for people experiencing other forms of social disadvantage. Nearly all studies were US-based, indicating opportunities for the development of evidence-informed interventions in other contexts.

LAY SUMMARY

Strategies to improve equitable access to cancer treatment are urgently needed. To date, these have primarily focused on improving health insurance coverage for cancer treatment (financial support), providing navigational support, and providing education on accessing cancer treatment through clinical trials. While important, these strategies do not address many of the systemic and structural barriers impacting socially disadvantaged groups, including inequitable distribution of social determinants of health, systemic racism, or geographical distribution of cancer treatment services. Despite Canada's publicly funded healthcare system, inequities in access to cancer treatment persist. Evidence-informed interventions are needed to improve access to cancer treatment; however, to date, there are few, if any, interventions that have been developed, implemented, and evaluated in the Canadian context for these purposes. Future research to address this gap is needed.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Longitudinal Financial Worry in a Cohort of Patients with Advanced Cancer - Poster #351

Presenter: Philip Akude, University of Calgary

Other Authors: Hannah Douglass-Molloy, Anyharan Sinnarajah, Patricia Biondo, Jessica Simon

ABSTRACT_ID#3339

ABSTRACT

Introduction: This study examines financial worry among patients with advanced colorectal cancer in Canada, highlighting a key dimension of financial toxicity that has implications for health equity and system-level support. **Methods:** We conducted a secondary analysis of the PaCES (Palliative Care Early and Systematic) cohort study, which recruited patients from tertiary cancer centres in Alberta between January 2018 and December 2020. Financial worry was self-reported using the Canadian Problem Checklist, completed monthly for 10 months and every three months thereafter. **Results:** Of 131 enrolled patients (median age 62.3 years), 43.5% reported financial worry at least once during the study period. Among them, 82.4% reported recurrent worry and 31.6% reported persistent concern across multiple time points. Notably, 35.7% of patients who initially reported no financial worry later developed it, while 87.5% of those with initial worry continued to report it. Bivariable analysis indicated that younger age, non-White ethnicity, and male gender were associated with higher financial worry. Only younger age remained significant in multivariable analysis. The average proportion of patients reporting financial concern at any given time was 18.8%. **Conclusion:** Financial worry is both common and persistent in patients with advanced colorectal cancer, particularly younger individuals. These findings underscore the need for routine financial distress screening and the integration of financial support services into cancer care delivery. Targeted policies could help address socioeconomic inequities and reduce the broader system burden of financial toxicity.

LAY SUMMARY

Financial worry is common and persistent among cancer patients: Over 40% of patients with advanced colorectal cancer reported financial worry during their illness, and most experienced it repeatedly over time. This indicates a sustained need for financial support, not just at diagnosis. Younger and racialized patients are more affected: Younger individuals and those from non-White backgrounds were more likely to report financial worry, highlighting a pressing equity issue within cancer care. Routine screening and targeted policy responses are needed: Financial worry often emerges or worsens over time—even in a publicly funded health system like Canada's. These findings support the need for policies that integrate routine screening for financial distress and expand access to financial navigation and support services within oncology care.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Estimating the number of people living with or beyond cancer in Canada - Poster #352

Presenter: Yibing Ruan, Alberta Health Services

Other Authors: Dylan O'Sullivan, Rochelle Garner, Darren Brenner

ABSTRACT_ID#2997

ABSTRACT

Background: Cancer survivors carry higher risk of cardiovascular diseases, immunological disorders, mental health issues, and subsequent primary cancers. Many of these could occur beyond twenty years from their first cancer diagnosis. Information on the number of people living with or beyond cancer is important to the planning of survivor-centered health care and supports. In Canada, the number of cancer survivors is approximated with a 20-year limited-duration prevalence, defined as the number of diagnosed cancers in the previous 20 years among people alive on a specified date. However, this estimator has two limitations. First, as more cancer survivors live beyond twenty years, the 20-year prevalence underestimates the actual number of survivors. Second, the 20-year prevalence is only available for the year 2012-2018, and is unavailable for Quebec. **Methods:** The OncoSim-All Cancers Model (v3.6.5.7) is a microsimulation tool developed by Statistics Canada and the Canadian Partnership Against Cancer. The model is calibrated to the Canadian cancer incidence, mortality, and survival data of 1992-2020. We estimated the prevalence of all cancer survivors with OncoSim and compared to the 20-year prevalence of major cancer sites available at Statistics Canada. **Results:** All cancer survivors in Canada (excluding Quebec) increased from 1.24 million in 2012 to 1.49 million in 2018, and to 1.81 million in 2015, which aligned well with the 20-year prevalence (0.98 million in 2012, 1.20 million in 2018). It also aligned well by major cancer sites, province, age group, and sex. **Conclusion:** OncoSim provides a good estimate of all cancer survivors in Canada.

LAY SUMMARY

Information on the number of people living with or beyond cancer is important for health care planning, social supports, and public awareness. This number is approximated with a 20-year cancer prevalence, which is the number of diagnosed cancers during the previous 20 years among people who are still alive on a specified date, e.g., last day of a calendar year. With more and more patients living beyond 20 years from their first cancer diagnoses, the 20-year cancer prevalence underestimates the actual number of cancer survivors. Using OncoSim, a simulation tool that is tailored to the Canadian cancer incidence, mortality and survival data, we estimated the number of people living with or beyond cancer in Canada at each calendar year up to 2051. Our estimates align well with the 20-year cancer prevalence.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

The cost of a cancer diagnosis in Northeastern Ontario - Poster #353

Presenter: Stacey A. Santi, Health Sciences North Research Institute

Other Authors: Emelie M. Cote, Christopher J. Longo, Carolyn Rideout, Rasathma Ratnayake Mudiyansele, Carla Bossart-Pletzer, Kevin Lam, Stephanie Winn, Tracy Laginski, Deborah P. Saunders, Lacey D. Pitre

ABSTRACT_ID#3294

ABSTRACT

Introduction: Financial difficulties arising from treatment-related costs and out-of-pocket expenses after receiving a cancer diagnosis can have a substantial impact on a patient, their family, and care-person(s). Out-of-pocket costs such as travel expenses, medical devices and services, additional medication costs, and lost income may result in patients opting out of recommended care. Northeastern Ontario spans a large geographic region with a considerable proportion of the population residing in rural and remote regions. **Methods:** To assess the financial toxicity of having a cancer diagnosis in Northeastern Ontario and optimize study enrollment, a digital questionnaire (P-SAFE 7.3.2, adapted) was administered to patients attending the Shirley & Jim Fielding Northeast Cancer Centre in Sudbury, Ontario. The questionnaire was administered at the time of appointment registration, leveraging existing technology used to collect provincial cancer symptom assessment data. **Results:** A total of 674 cancer patients have interacted with the digital questionnaire after 31 weeks of enrollment. The questionnaire completion rate after the first week was 45.4%, demonstrating the engagement and efficacy of the study delivery. Results from the questionnaire data regarding the financial toxicity experienced by cancer patients and care partner(s) in this region will be discussed. **Conclusion:** Administration of a digital questionnaire at appointment registration provides a novel method to capture financial toxicity data from cancer patients at all stages of treatment pathway, including those actively undergoing treatment and those in follow-up care.

LAY SUMMARY

- Out-of-pocket costs and treatment-related expenses due to a cancer diagnosis affects both patients and their care-person(s).
- This study assessed the impact of out-of-pocket costs and additional expenses (such as travel, medical devices, medical services) in Northeastern Ontario, where many patients often travel long distances to receive their cancer care.
- Delivery of a digital questionnaire at the time of appointment registration is an effective method to capture study data.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

What should research say about us? Trans and Gender Diverse People's perspectives on including gender diversity in cancer health services research? - Poster #354

Presenter: Morgan Stirling, University of Manitoba

Other Authors: Tara Horrill, Janice Ristock, Annette Schultz, Nathan Nickel, Alyson Mahar

ABSTRACT_ID#2788

ABSTRACT

Background: Trans and Gender Diverse (TGD) people are routinely excluded in cancer health services research. TGD people's exclusion is driven by many factors, including: absence of gender-specific data, inadequate approaches for measuring gender diversity, and limited engagement with TGD people in cancer research. Addressing the factors limiting TGD people's representation requires ensuring TGD people's voices are heard and prioritized in developing novel approaches for including gender diversity in cancer health services research. Methods: Recruitment of TGD people with cancer care experiences and live in Canada is underway. Focus groups, each consisting of 4-6 participants, are being organized regionally across Canada. The focus group guide was collaboratively developed with a community advisory board. Questions explore TGD people's perspectives on best practices related to: collecting gender identity data; engaging TGD people in cancer research; data governance and using TGD people's data; and training for cancer health services researchers. Data will be analyzed following a Reflexive Thematic Analysis approach. Results: This study is ongoing. Data analysis to be completed by November 2025. Significance/Conclusion: Efforts to improve how gender diversity is included in cancer research must be guided by TGD people. Results from this study will emphasize TGD people's voices in outlining concrete steps researchers, administrators, and decision-makers can take to transform the practices, policies, and infrastructure that have led to TGD people's exclusion in cancer health services research.

LAY SUMMARY

This study addresses the gap in representation and engagement with Trans and Gender Diverse people in cancer health services research and is a step toward improving the quality and availability of evidence to inform care.

Health Services Research, Health Economics, and Implementation Science

Sunday, November 2, 2025

Real-world survival outcomes and adverse events with adjuvant osimertinib in resected EGFR mutation-positive non-small cell lung cancer - Poster #355

Presenter: Vanessa Samuel, Alberta Health Services

Other Authors: Amanda Gibson, Michelle Liane Dean, Yaroslav Musiiets, Erik Nohr, Vishal Navani

ABSTRACT_ID#2823

ABSTRACT

Background: Adjuvant osimertinib (AO) improved disease-free survival and overall survival in patients with resected epidermal growth factor receptor (EGFR) mutation-positive non-small cell lung cancer (NSCLC) in the ADAURA trial. Our study aimed to assess provincial real-world outcomes of AO for patients with EGFR-positive NSCLC. Methods: A retrospective review was conducted using the Glans-Look Lung Cancer Research database in Alberta, Canada. Patients had resected clinical stage IA-IIIa EGFR-positive NSCLC and received AO in Alberta between September 2021 - November 2024. Disease-free survival was reported using summary statistics. Date of last follow-up was November 2024. Results: 25 patients received AO following resection of their NSCLC. Median duration of AO was 9.8 months. One patient (4%) had disease recurrence following AO, albeit this patient only received 12 days of AO due to toxicity. Disease-free survival ranged from 3.9 – 34.2 months. At the time of analysis, 24 patients (96%) were alive, with one death. Of the 29 adverse events (AE) recorded, there were 13 (45%) grade 1, 13 (45%) grade 2, 1 (3%) grade 4, and 2 (7%) AE of unknown grade. In ADAURA, there were 493 (69%) grade 1, 202 (28%) grade 2, and 22 (3%) grade 3 AE. 28% of patients discontinued AO compared to 11% in ADAURA. Conclusion: AEs occurred less frequently compared to ADAURA, although discontinuation of AO due to AE was higher in our experience, implying a poorer tolerability of AO in routine practice. Future studies require longer follow-up periods to better describe time to event endpoints for AO.

LAY SUMMARY

Adverse events occurred less frequently compared to the clinical trial setting, although discontinuation of osimertinib due to adverse events was higher in our experience, 28% vs. 11%, implying a poorer tolerability of osimertinib in routine practice compared to the trial setting. Similar to the clinical trial setting, most adverse events were low grade, suggesting that there may be an unmet need for more proactive symptom control to mitigate adverse events that may lead to discontinuation of the drug. Future studies require longer follow-up periods to better describe time to event endpoints (i.e. disease-free survival) for adjuvant osimertinib in patients with EGFR positive non small cell lung cancer.

Health Services Research, Health Economics, and Implementation Science

Monday, November 3, 2025

Evaluating the Resource Burden of Radiation Therapy: Identifying Priority Disease Sites for Adaptive Radiation Therapy Implementation - Poster #356

Presenter: Vickie Kong, Princess Margaret Cancer Centre, University Health Network

Other Authors: Aydin Visanji

ABSTRACT_ID#2542

ABSTRACT

Adaptive Radiation Therapy (ART) has been shown to improve clinical outcomes by accounting for anatomical and physiological changes during treatment, thereby enhancing the precision of radiation delivery. In contrast, non-adaptive radiotherapy (RT) employs a static plan throughout the course of treatment, potentially compromising accuracy. However, ART implementation introduces a significant resource burden due to increased imaging, plan adaptations, and clinical oversight. As of 2025, fewer than 10 ART-capable treatment devices are available in Canada, highlighting the need for careful prioritization of patients who would benefit most from this resource-intensive approach. To inform such decisions, we analyzed data from 6,721 treatment appointments across 300 patients, with approval from the Quality Improvement Review Committee. Metrics assessed included appointment duration and the frequency and nature of unplanned activities. The median treatment session length was 16 minutes (range: 15–26 minutes). The most common unplanned activities were repeated imaging and treatment plan modification. A total of 250.4 hours was spent acquiring 790 repeated images for 178 patients (59%), primarily those with bladder and cervix cancers. Sixteen patients required plan modifications during treatment, most commonly among those with sarcoma (n=5) and pelvic cancers (n=9). These findings demonstrate that the resource burden of interventions is considerable. Patients with bladder, cervix, and sarcoma cancers may particularly benefit from a planned adaptive approach. Targeted implementation for these disease sites could optimize resource utilization and improve patient experience within the current system limitations.

LAY SUMMARY

1. Limited capacity for Adaptive Radiation Therapy (ART) delivery: The availability of ART-capable equipment in Canada remains low, necessitating careful allocation of this advanced but resource-intensive treatment approach.
2. Repeated imaging and plan modification drive resource burden: Frequent unplanned activities - particularly repeat imaging and re-planning significantly increase treatment time and clinical workload in the current radiation therapy workflow.
3. Disease site prioritization is key to sustainable ART implementation: Focusing ART on disease sites with a high frequency of unplanned activities (e.g. bladder, cervix, sarcoma) can maximize clinical benefit while preserving system efficiency.

Chimeric-Antigen Receptors to Drive Antigen Presentation: Leveraging Dendritic Cell Function to Bolster Anti-Tumour Immunity - Poster #357

Presenter: Cassandra Wood, University of Calgary

Other Authors: Gerone Gonzales, Liam Wilkinson, Yvonne Chen, Johnathan Canton, Song Huang

ABSTRACT_ID#2598

ABSTRACT

Conventional type 1 dendritic cells (cDC1s) play a pivotal role in anti-tumor immunity through their ability to cross-present (XP) exogenous antigens on MHC-I and prime naïve CD8⁺ T cells. The presence of cDC1s in tumors strongly correlates with improved patient outcomes and cDC1-deficient models fail to reject immunogenic tumors. Despite this, cDC1s remain vastly underutilized in current immunotherapies. While CAR-T therapies have demonstrated success in hematologic cancers, their application to solid tumors remains limited by poor tumor infiltration, immunosuppressive microenvironments, and dependence on a single target antigen, leaving them vulnerable to immune evasion. In contrast, endogenous cDC1s initiate immune responses by capturing, processing, and presenting a diverse repertoire of tumor antigens to CD8⁺ T cells. This allows for the generation of broad T cell responses capable of adapting to tumor heterogeneity. Leveraging this natural functionality, the concept of synthetically engineered, CAR-expressing cDC1s is highly compelling. This project introduces CAR-XPs: a new class of Chimeric Antigen Receptors designed to enhance the cancer antigen-presenting capacity of cDC1s by engaging the body's native antigen presentation pathways to stimulate robust, multi-antigenic cytotoxic responses. In vitro testing will evaluate a panel of CAR-XP constructs for their ability to promote antigen uptake and signaling cascades that drive key steps in cross-presentation. To enable translational application, we are optimizing cDC1-specific CAR expression via lentiviral delivery under the cDC1 specific XCR1 promoter and developing adoptive transfer strategies for tumor-bearing mice. This work seeks to expand the immunotherapeutic toolkit by harnessing cDC1s as programmable initiators of broad anti-tumor responses.

LAY SUMMARY

Tumors are not made of one type of cell. They are a mix of many different cancer cells. This diversity, known as tumor heterogeneity, makes it hard for treatments that target a single cancer marker to work well long-term. Conventional type 1 dendritic cells (cDC1s) are a rare but critical part of the immune system that can help address this problem. cDC1s are especially good at taking up many different types of tumor material and presenting them to T cells, helping the immune system recognize and attack a broader range of cancer cells. We are developing synthetic receptors (CAR-XPs) to improve how well cDC1s detect and present tumor antigens. This could strengthen the immune response against tumors with high heterogeneity, where single-target treatments often fail.

Clinical Bacillus Calmette-Guérin Strains Induce Distinct Trained Immunity Profiles - Poster #358

Presenter: Richard Nauman, Queen's University

Other Authors: Peter Greer, Andrew Craig, D. Robert Siemens, Charles Graham

ABSTRACT_ID#3209

ABSTRACT

Bacillus Calmette-Guérin (BCG) immunotherapy remains the standard of care for higher-risk non-muscle invasive bladder cancer (NMIBC), despite an incomplete understanding of its immunotherapeutic mechanism. Trained immunity (TI), a heterologous form of memory acquired by innate immune cells, is thought to be involved. We have previously reported that TI acquisition in peripheral immune compartments is associated with recurrence-free survival post-BCG therapy. Studies on the effects of BCG in the protection against tuberculosis have revealed that different strains of BCG have distinct immunogenic properties; however, a thorough comparison of the TI-inducing properties of different strains of BCG has not been performed. We assessed TI acquisition and secondary responses following administration of BCG-Russia or BCG-TICE. Macrophages from mice treated with BCG-TICE exhibited significantly higher production of several proinflammatory cytokines in response to heterologous stimuli compared to BCG-Russia or vehicle control. BCG treatment led to expansion of hematopoietic stem and progenitor cell (HSPC) populations, however the effect appeared to be independent of strain. Transcriptomic analysis revealed that expression of genes related to several processes differed between the two strains, including antigen presentation, ECM remodeling, and T cell activation. TI is emerging as a crucial component of the mode of action for BCG therapy in NMIBC. Shortages of BCG-TICE have historically posed a clinical challenge, with BCG-Russia often used as a substitute in Canada. Understanding the differential immune responses elicited by these strains informs clinicians about the suitability of BCG-Russia as an alternative to BCG-TICE.

LAY SUMMARY

BCG-TICE elicits stronger innate immune (bone marrow) reprogramming compared to a different strain that is often used as a substitute for TICE. Macrophages, in particular, exhibited distinct cytokine secretion and transcriptomic profiles dependent on BCG strain. This research has direct implications for clinical decision making, suggesting that the immunostimulatory effect of BCG is strain-dependent and certain strains may be inferior to others.

A Quality Improvement Initiative to Mitigate Immunotherapy-Related Toxicities among Patients Receiving Immunotherapy using a Novel Grade 2 Toxicity tool - Poster #359

Presenter: Christian James-McDonald, Schulich School of Medicine & Dentistry

Other Authors: Laurice Arayan, Caroline Hamm, Emmanuel Joran Boujeke, Tina Joseph, Tiffany Gowanlock, Mina Djuketic

ABSTRACT_ID#3134

ABSTRACT

Introduction: Guidelines on the management of immune-related toxicities (IRTs) are difficult to use because of the need to be detailed and extensive. This study aims to develop and evaluate a novel grade 2 immunotherapy toxicity screening tool designed to identify and prevent progression of IRTs before they result in hospitalization and irreversible organ damage.

Methods: The tools were adapted from the CCO Immune Checkpoint Inhibitor Toxicity Management Clinical Practice Guideline (March 2018). A Plan-Do-Study-Act (PDSA) cycle is being employed to test, refine, and implement the tool, with iterative changes based on feedback from healthcare providers and patients. Healthcare providers were engaged through team presentations in staff meetings, and the Patient and Family Advisory Committee was also consulted for feedback. Handouts were distributed to patients and healthcare providers, and displayed in chemotherapy suites and clinics. The intervention's effectiveness is being assessed through surveys to gather perceptions and identify barriers to use. **Results:** In the January 2025 survey, 43 healthcare providers and 28 patients found the tool easy to use and recommended its continued use. Both groups suggested adding symptoms like arthritis, though some reported not receiving the tool. Despite this, the tool was considered helpful by both groups in managing IRTs. **Discussion:** Revisions were made to the tool based on January 2025 survey feedback, and a follow-up survey is ongoing. Continued evaluation will determine its effectiveness in preventing severe IRTs and guide further improvements. **Funding:** Bristol Myers Squibb- Improvement Initiative in Immuno-Oncology Fund.

LAY SUMMARY

Key Message: A novel grade 2 immunotherapy toxicity screening tool was developed to help patients and frontline healthcare providers more easily identify and manage immune-related toxicities (IRTs) associated with immune checkpoint inhibitors in triple-negative breast cancer. Initial feedback from both groups indicated the tool was easy to use and effective, though minor revisions were needed to improve symptom coverage and ensure broader distribution. Continued evaluation and refinement are underway to assess the tool's long-term impact on preventing severe IRT progression and improving patient outcomes.

Farmington virus – a potent oncolytic rhabdovirus for cancer immunotherapy - Poster #360

Presenter: Daniil Vishnevskiy, University of Calgary

Other Authors: Jahanara Rajwani, Victor Naumenko, Kyle Potts, Hayley Todesco, Franz Zemp, Craig Jenne, Douglas Mahoney

ABSTRACT_ID#2733

ABSTRACT

Introduction Viruses are powerful tools in cancer immunotherapy and can be engineered to infect tumor cells and induce oncolysis. In addition to directly killing cancer cells, oncolytic viruses (OVs) stimulate anti-cancer immune responses, enhancing therapeutic effects. Rhabdoviruses are promising OVs due to their rapid replication, strong type I interferon (IFN) response, and low pre-existing immunity in populations. Farmington virus (FARV), a rhabdovirus, shows anti-tumor potential with potent oncolytic activity against human cancer cell lines in vitro. Remarkably, FARV controls tumor growth and enhances survival in animal models even more effectively than vesicular stomatitis virus (VSV) — the most studied oncolytic rhabdovirus (ORV). **Methods** We previously showed that VSV's therapeutic effect relies less on direct cell lysis and more on the induction of anti-tumor CD8+ T cell responses, mediated by type I IFNs released from infected non-tumor cells in the spleen. To understand FARV's superior efficacy, we explore differences in ORVs' interaction with the host and how these differences foster a stronger anti-tumor immunity. **Results and Discussion** After systemic administration, both viruses are retained in tumor tissues but don't show productive infection. FARV, however, is cleared more slowly from blood and tumor than VSV. CD8+ T cells are critical to the therapeutic effects of both viruses, as their depletion abolishes efficacy. FARV induces a stronger, prolonged systemic cytokine response and targets different non-tumor cells in the spleen. Further studies will examine differences in anti-tumor immune responses and how cytokines and infected non-tumor cells shape them.

LAY SUMMARY

A new cancer-fighting virus called Farmington virus (FARV) shows strong ability to slow tumor growth and help animals live longer—even more effectively than a well-known virus used in cancer therapy called vesicular stomatitis virus (VSV). FARV boosts the body's immune response against tumors, especially by activating CD8+ T cells, which are a type of immune cell that can kill cancer cells. Removing these T cells eliminates the virus's benefits, showing how important they are for treatment. We hypothesize that differences in how fast the viruses replicate may affect the immune response—VSV replicates quickly and builds up more viral antigens, which may shift the immune system toward fighting the virus instead of the tumor. In contrast, FARV's slower replication might avoid triggering strong anti-viral immunity, allowing a more balanced and effective anti-cancer immune response.

Immunotherapy

Sunday, November 2, 2025

Anti-cancer immune responses induced by cytokines - Poster #361

Presenter: Alexandra Berger, UHN

Other Authors: Sarah Colpitts, Morgan Zych, Caren Furlonger, Cheryl D'Souza, Jeffrey Medin, Christopher Paige

ABSTRACT_ID#2925

ABSTRACT

We are testing the effects of cytokines on anti-cancer immune responses in order to optimize cancer immunotherapy. A clinical trial (Phase 1 study of autologous acute myelogenous leukemia (AML) cells containing lentivirus engineering expression of IL-12: NCT02483312) is underway. It is based on experimental murine models testing the effects of IL-12, IL-15, IL-18 and IL-21 to elicit long-lasting immune mediated elimination of cancer cells in vivo. Cancer cells are engineered to secrete cytokines using lentivirus vectors and injected ip into mice. Immune cell populations are characterized by flow cytometry during the first two weeks of the immune response. Long term survival is determined by re-challenging the animals with the parent cell line (no cytokine) after 100 days. Our model reveals the interplay between cytokine-driven early innate immune responses and the development of sustained immunity. Conditions that promote fast acting, but limited, NK-cell responses, followed by an expansion of T-cells, leads to both a strong early anti-cancer immune response and long term (~6 months) immune memory. In these cases mice reject a re-challenge of non-cytokine producing parent cells. However, conditions that result in an overabundant innate response, while leading to a strong reduction in cancer burden and a significant increase in survival time to 2-3 months compared to controls (10 days), demonstrate poor long term immunity. Our goal is to design and develop a cytokine-initiated cancer immunotherapy, which orchestrates the innate and adaptive immune system in order to achieve a fine-tuned immune response.

LAY SUMMARY

Anti-cancer immune response induced by lentivirus-engineered cells secreting IL-12, IL-15, IL-18 or IL-21: preclinical models as a basis for ongoing Phase 1 IL-12 study.

Bench-to-bedside development of GCAR1: A made-in-Canada CAR T therapy targeting MiT-fusion driven protein GPNMB - Poster #362

Presenter: Franz Zemp, University of Calgary

Other Authors: Zackariah Breckenridge, Hyojin Song, Gurveer Gill, Hongrui Liu, Yeon Suh, Louisa Guignard, Joanna Pyczek, Laura Mah, Taye Louie, Kristofer Ellestad, Victor Naumenko, Varsha Thoppey Manoharan, Kiran Narta, Ted Verhey, Hayley Todesco, Kyle Potts, Kiril Trpkov, Jennifer Chan, Ramy Saleh, Jan Willem Henning, Nicole Prokopishyn, Sorana Morrissy, Mona Shafey, Douglas Mahoney

ABSTRACT_ID#3302

ABSTRACT

Chromosomal translocations drive oncogenic gene fusions in ~30% of soft-tissue sarcomas (STS) and certain carcinomas and leukemias. Among these, the microphthalmia-associated transcription factor (MiT) family, including TFE3, frequently serves as the 3' fusion partner in pathognomonic genetic rearrangements underlying alveolar soft-part sarcoma (ASPS) and translocation-associated renal cell carcinoma (tRCC). Glycoprotein NMB (GPNMB), identified as a MiT-fusion-driven cell surface protein, exhibits high and consistent expression in ASPS and tRCC, with minimal presence in normal tissues. GCAR1 is a second-generation 4-1BB/CD3ζ chimeric antigen receptor (CAR) T-cell therapy engineered to target GPNMB. Extensive preclinical studies validated its therapeutic potential, leading to clinical translation. A first-in-human, open-label individual patient study (OLIP) evaluated GCAR1 as a single infusion (1×10^6 CAR-T cells/kg) in a Stage IV ASPS patient with progressive pulmonary metastases. The therapy was well tolerated, with only grade 1 transaminase elevations and a grade 1 urticarial rash. GCAR1 expanded in vivo, reaching ~20% of circulating PBMCs by day 14, persisting until day 84. Radiological assessment revealed stable disease in the largest target lesions for six months, with partial and complete responses in the majority of non-target lesions, leading to symptomatic improvement. Following further disease progression 16 months after the initial dose, a second OLIP study administered an escalated dose (5×10^6 CAR-T cells/kg) demonstrated robust CAR-T expansion but mixed clinical response. Correlative studies identified T-cell inhibitory pathways limiting responses, prompting investigations into Atezolizumab (anti-PDL1) as an adjuvant therapy. A multicenter Phase I trial will commence in 2025 to assess GCAR1 in solid malignancies expressing GPNMB.

LAY SUMMARY

GCAR1 is an innovative CAR T-cell therapy designed to fight certain cancers by targeting GPNMB, a protein that becomes highly expressed due to specific genetic changes that occur in particular cancer types. GCAR1 works by harnessing the patient's own immune cells, modifying them to recognize and attack cancer cells that display GPNMB, offering a new potential approach for treatment. The first patient treated with GCAR1 showed that the therapy is likely safe and has strong potential to fight tumors. Research on how GCAR1 worked in this patient has provided valuable insights, helping scientists identify ways to improve the treatment for future patients. GCAR1 will undergo further testing in a Phase I clinical trial taking place across Canada, starting by the end of this year. This study will help researchers better understand how safe and effective GCAR1 is in treating cancer across a diverse group of patients while providing additional findings that will guide further studies to make the therapy even more effective.

Hsp90 inhibitors reprogram the cancer immunopeptidome and enhance tumor immunogenicity - Poster #363

Presenter: Alexa Mevel, University of Alberta

Other Authors: Rebecca Mercier, Isabelle Sirois, Charles Joly-Beauparlant, Arnaud Droit, Etienne Caron, Madison Wickenberg, Christopher Savoie, Zhaoguan Wu, Richard Fahlman, Shelly Braun, Paul LaPointe

ABSTRACT_ID#2649

ABSTRACT

Peptides from degraded proteins are presented to the immune system on the surface of the cell by Major Histocompatibility Complex 1 (MHC1) molecules, making up what is referred to as the immunopeptidome. The immune system recognizes abnormal peptides in the immunopeptidome and induces apoptosis in the cells that are presenting them. The molecular chaperone Heat Shock Protein 90 (Hsp90) is especially important in cancer cells, as it can maintain the folding of the highly mutated and unstable proteins that are characteristic of most cancers. This prevents the degradation of these proteins and subsequent peptide presentation, allowing cancer cells to evade detection by the immune system. When Hsp90 is inhibited, degradation of misfolded proteins increases, MHC1 presentation is upregulated, and the peptides that are presented in the immunopeptidome change. We are now investigating what determines the immunopeptidome of any given cell and how this can influence anti-tumor immunogenicity. To do this, we are taking advantage of the diverse cellular effects of Hsp90 inhibition—including changes in total protein levels, transcription, and protein degradation rates. We have generated four datasets to measure changes in the proteome, transcriptome, protein turnover rates, and the immunopeptidome after inhibiting Hsp90. By using a multi-omics approach to compare changes across each dataset to changes in the immunopeptidome, we can gain insight into the cellular processes that drive peptide presentation. The results from this analysis can give us a better understanding of how to enhance tumor immunogenicity, ultimately promoting tumor recognition and destruction by the immune system.

LAY SUMMARY

Peptides presented in the immunopeptidome change after Hsp90 inhibition Neoantigens are detected in the immunopeptidome after Hsp90 inhibition

Safety and Effectiveness of Immuno-oncology Agents in Patients with Lung Cancer and Pre-existing Autoimmune Disease - Poster #364

Presenter: Siddhartha Goutam, University of Calgary

Other Authors: Arjun Raghavan, Hans Alday, Daniel Meyers, David Dawe, Liam O'Neil, Jeffrey Graham

ABSTRACT_ID#3020

ABSTRACT

Background: Patients with pre-existing autoimmune disease (PAD) have been largely ineligible for lung cancer immuno-oncology (IO) trials. This study aimed to assess the safety and effectiveness of IO therapy in patients with lung cancer and PAD in the province of Manitoba, Canada. Methods: We used a provincial repository of patients with PAD matched to 1-3 patients without PAD based on age, sex and cancer type to isolate patients with a diagnosis of lung cancer. All patients had received at least 1 cycle of IO between Jan 2013 – June 2022. Rates of immunotoxicity, treatment cessation, rechallenge, immunosuppression/DMARD initiation were compared across the PAD and non-PAD cohort. Time to event endpoints of overall survival (OS) and treatment failure (TTF) were analyzed in the subgroup with advanced disease. Statistical approaches used STATA v12.1. Results: 69 patients with PAD were matched to 139 without PAD, with median age being 69, and 64.9% of patients being male. 66.8% had adenocarcinoma. 78.9% had advanced disease. Immunotoxicity in the PAD group was 55.1% vs. 39.4% in the non-PAD group ($p=0.03$). Incidence of flare of the underlying PAD was 20.3%. Controlling for IO type, there was no significant association between PAD status and irAE (OR=1.27, $p=0.43$) or rate of high grade irAEs (17 vs 24%; $p=0.45$). No significant differences were observed in ORR, TTF and OS in patients with advanced disease. Conclusion: 1 in 5 patients with PAD experienced a flare, however PAD status was not correlated with higher rates of irAEs, or differences in oncologic outcomes.

LAY SUMMARY

1. Our findings suggest that patients with pre-existing autoimmune disease can be treated with immuno-oncology agents with similar rates of overall survival and time to treatment failure.
2. Rates of treatment interruption are higher in patients with pre-existing autoimmune disease as they have the added burden of autoimmune disease flare while being on therapy, but the rates of immunotherapy related adverse events are no different between patients with pre-existing autoimmune disease and those without.
3. More granular data within pre-existing autoimmune disease can be achieved with an expanded dataset, and can inform clinical decision making in clinical practice.

Mobilizing effector double-negative T cells for adoptive cellular therapy against acute myeloid leukemia through exercise - Poster #366

Presenter: Heejae Lee, University of Calgary

Other Authors: Jonathan L. Low, Brittany A. Edgett, S. Nicole Culos-Reed, Jongbok Lee

ABSTRACT_ID#3068

ABSTRACT

Introduction: Double-negative T cell (DNT) therapy is an innovative immunotherapy for acute myeloid leukemia (AML), involving the infusion of donor immune cells (i.e., DNTs) into patients for treatment. Effector immune cells with anti-cancer cytotoxicity, such as NK cells, are highly responsive to acute exercise, increasing their number in circulation up to 10-fold. Exercising donors prior to blood collection may produce superior DNT products, however, its effects on DNT therapy has not been examined. Thus, this study aims to investigate the impact of a pre-donation exercise intervention on the effectiveness of DNT therapy for AML. Methods: DNTs were isolated from peripheral blood of 20-35-year-old healthy participants (n=6, 3 females) at rest and after 30-minutes of high-intensity interval exercise. DNT count and memory phenotype were examined using a Spectral Flow Cell Analyzer. After 10-day expansion, the anti-leukemic activity of DNTs collected at rest (DNTRest) and after exercise (DNTEEx) was compared using 2- and 24-hour cytotoxicity assays against human AML cell lines in vitro. Results: Exercise increased the number of DNTs (2.3 ± 0.7 -fold, $p=0.04$), preferentially mobilizing those with an effector memory phenotype (DNTRest=22.7±8.1%, DNTEEx=27.5±6.2%, $p=0.007$). Preliminary data (n=1) showed that this difference in memory composition became more apparent after 10-day expansion (DNTRest=42.4%, DNTEEx =92.5%), and DNTEEx demonstrated superior killing of AML cells at 2 and 24 hours. Conclusion: Acute exercise mobilized effector DNTs, demonstrating increased anti-leukemic activity in vitro. Future work will examine the potential of exercise to enhance the effectiveness of DNT therapy for AML.

LAY SUMMARY

Double-negative T cell (DNT) therapy is a promising safe and accessible treatment option for acute myeloid leukemia, involving the infusion of donor-derived immune cells into patients for treatment. The immune system is highly responsive to exercise, with benefits that could be leveraged to enhance anti-cancer therapeutics. While implementing structured exercise regimens in high-risk patients actively battling cancer is challenging, DNT therapy provides a unique opportunity to indirectly harness the immune-boosting effects of exercise through a pre-donation exercise program performed by healthy donors, potentially producing a superior DNT product that could lead to enhanced treatment outcomes.

Development of CAR-Double-Negative T Cells as an Off-the-Shelf Therapy for B-Cell Malignancies - Poster #368

Presenter: Jessica López Espinosa, University of Calgary

Other Authors: Juan Arteaga, Michele Nawata, Victor Lewis, Kevin Hay, Jongbok Lee

ABSTRACT_ID#2375

ABSTRACT

Introduction: CAR-T cell therapy has shown significant success in treating B-cell leukemias and lymphomas. However, it faces several barriers like manufacturing complexity, limited accessibility, and relapse due to antigen escape. Double-negative T cells (DNTs), a T-lymphocyte subset which lacks CD4 and CD8 markers, possess intrinsic anti-tumour activity and have potential as an off-the-shelf, allogeneic platform for CAR-based immunotherapy. Methods: DNTs were isolated from healthy donors and modified using lentiviral vectors to express CARs targeting CD19 or CD22. Transduced cells were expanded and assessed for viability and CAR expression. In vitro cytotoxicity against B-cell leukemia cell lines was evaluated using killing assays and analyzed by flow cytometry. Cryopreservation studies were conducted to determine post-thaw viability and cytotoxic functionality. Results: Healthy-donor derived CAR-DNTs targeting both CD19 and CD22 demonstrated superior cytotoxicity compared to untransduced DNTs in in vitro killing assays. These cells maintained cancer-killing function after cryopreservation, indicating their suitability for “off-the-shelf” application. Notably, when targeted by CAR19-DNTs, B-cell leukemia cells exhibited downregulation of CD19 without concurrent downregulation of CD22 when targeted by CAR22-DNTs. This phenomenon mimics a known clinical mechanism of antigen-negative relapse, allowing the targeting of CD19^{low} or negative cancers with CAR22-DNTs. Future Directions: We plan to assess the in vivo efficacy, persistence, and safety of single and mixed CAR-DNTs in a B-cell leukemia xenograft model. Additionally, we aim to evaluate the therapeutic potential of combining CD19- and CD22-directed CAR-DNTs to overcome antigen escape in CD19-downregulated B-ALL and enhance treatment durability.

LAY SUMMARY

We’re developing a new type of cancer immunotherapy using “double-negative T cells” (DNTs), which naturally help fight cancer and can be used off-the-shelf from healthy donors. Unlike traditional CAR-T cell therapies, which are made from each patient’s own cells and take weeks to produce, DNTs offer a faster, more accessible alternative. We engineered these DNTs to specifically recognize and kill B-cell leukemia cells by targeting proteins called CD19 or CD22. Our lab tests showed that the modified DNTs were highly effective at killing cancer cells, and importantly, they still worked well after being frozen and thawed—making them practical for widespread use. By targeting both CD19 and CD22, we can potentially prevent cancer from escaping treatment. Some cancers survive by losing the CD19 marker, but our CD22-targeting DNTs still recognized and killed those cells. This means using a combination of the two could lead to longer-lasting and more effective treatment.

Proteomic Profiling of Circulating Extracellular Vesicles to Identify Response Signatures and Resistance Mechanisms in NSCLC Patients Treated with Immune Checkpoint Inhibitors - Poster #369

Presenter: Catherine Taylor, Atlantic Cancer Research Institute

Other Authors: Michelle Davey, Ammar Sabir Cheema, Eric Allain, Nicholas Finn, Mahmoud Abdelsalam, Rodney Ouellette

ABSTRACT_ID#2983

ABSTRACT

Introduction: Immune-oncology has revolutionized cancer treatment but some patients fail to benefit due to tumor-immune evasion. Extracellular vesicles (EVs) are secreted by both tumour and immune cells that participate in the inter-play between cancer and immune cells. Our study used proteomic profiling of circulating EVs collected from NSCLC patients treated with immune checkpoint inhibitor (ICI) to identify novel biomarkers of response and EV-associated immune evasion mechanisms. **Methods:** Plasma was collected from NSCLC patients pre- and post-treatment with ICI. EVs were isolated from plasma using peptide-affinity purification and ~ 3000 proteins in the EV lysate were profiled using Proximal Extension Assay. Differentially expressed proteins were identified using Qlucore. Cox regression analysis was performed in R environment. **Results:** Proteomics analysis identified 140 differentially expressed EV proteins in patients that experienced durable compared to non-durable response to ICI. Pathway analysis revealed enrichment of VEGF signaling, chemotaxis, myeloid cell differentiation, and neutrophil degranulation associated with poor progression-free survival. Durable response to ICI treatment was associated with enrichment of T cell and B cell activation and antigen receptor-mediated signaling. Sex-related differences in signatures of response to ICI were explored. Increased on-treatment EV PD1 and decreased on-treatment EV KRT19 were associated with treatment response. **Summary:** Profiling circulating EVs may improve our understanding of how EVs participate in immune evasion during immunotherapy treatment and may point to new treatment modalities that could improve responses to immune checkpoint blockade in lung cancer patients. Circulating EV protein signatures may reflect the tumour immune microenvironment and be predictive of anti-PD1 response.

LAY SUMMARY

Extracellular vesicles (EVs) are small particles that are released by both cancer cells and the immune cells responsible for fighting cancer. When released by cancer cells, these vesicles play an important role in cancer development, progression, and treatment resistance; however they can also be involved in the immune system's fight against cancer. Our study investigated the proteins that are carried by EVs that are circulating in the blood stream in patients with lung cancer who are being treated with immunotherapy. We were able to identify a number of EV proteins that could help predict whether a patient would respond to treatment with immunotherapy or not. Our study also identified a number of cancer and immune-related pathways related to immunotherapy treatment resistance that could lead to new treatment combinations to improve immunotherapy responses. Using EVs collected from blood may allow us to better understand patients' responses to immunotherapy without the need for invasive tissue biopsies.

Assessing how macrophages influence CAR T therapy in soft tissue sarcoma - Poster #370

Presenter: Kurt Hildebrand, University of Calgary

Other Authors: Karys Hildebrand, Franz Zemp, Carolina Salazar Arcila, Danyel Evseev, Hyojin Song, Ted Verhey, Kiran Narta, Roman Krawetz, John Matyas, Sorana Morrissy, Douglas Mahoney

ABSTRACT_ID#3112

ABSTRACT

Soft tissue sarcomas (STS) are aggressive mesenchymal lineage tumours in desperate need of new treatments. STS generally do not respond to novel immunotherapies, which is thought to be due to the immune suppressive tumour microenvironment (TME) characteristic of many STS. Dismantling the immune suppressive TME is essential for improving the efficacy of immunotherapy in STS. Interestingly, tumour associated macrophages (TAMs) are the most abundant immune population in STS. In other malignancies, TAMs are known to suppress anti-tumour immunity and diminish efficacy of immunotherapy. However, TAMs have received little investigation in STS. It remains poorly understood how TAMs infiltrate the TME in STS. In assessing human STS using bulk RNA sequencing, we found TAM markers are positively correlated with CX3CR1 and CCR2, known monocyte recruitment pathways. We then engrafted murine STS into Ccr2+/RFP;Cx3cr1+/GFP mice, which showed that TAMs express both CCR2 and CX3CR1. However, in TAMs engrafted into CX3CR1 deficient and CCR2 deficient tumours, only CCR2 deficiency reduced TAMs by flow cytometry. Additionally, CCR2 deficiency showed reduced immune suppressive TAMs, increased T cells, and reduced tumour size. This suggests TAMs may be suppressing anti-tumour immunity in STS. We then wanted to assess if CCR2 deficiency enhanced the efficacy of chimeric antigen receptor (CAR) T therapy targeting GD2, a known target expressed in STS. Ccr2-/- mice showed a trend towards improved survival, but this was not statistically significant. Thus, reducing TAMs may be driving immune suppression and resistance to CAR T therapy in STS, however, further validation is necessary.

Sunday, November 2, 2025

You are what you eat –plasma metabolomics reveal amino acid consumption and sensing as actionable biomarkers for optimization of multiple T-cell therapies - Poster #371

Presenter: Devanand Pinto, Dalhousie University

Other Authors: Evelyn Teh, Susanne Penny, Kenneth Chisholm, Natasha Kekre, Kevin Hay, Rob Holt, Brad Nelson, John Kuruvilla, Rob Laister, Samuel Saibil, Tina Nguyen, Scott McComb, Risini Weeratna

ABSTRACT_ID#3321

ABSTRACT

Complete response rates to adoptive cell therapies vary widely, from below 5% in sarcoma to over 90% in some leukemias. Disease severity and an immunosuppressive tumor microenvironment are well-known predictors of response but, being difficult to modulate, are not currently actionable. Patient plasma metabolomic profiling is an emerging technique for the discovery of predictive biomarkers for adoptive cell therapy. Metabolomic was conducted with liquid chromatography coupled to tandem mass spectrometry (LC-MS/MS). We analyzed the plasma from a discovery cohort (n=60) of patient receiving an investigational chimeric antigen receptor (CAR) T cell therapy targeting CD-19 (CLIC-1901). The findings from this analysis were verified using plasma from patients (n=30) receiving a standard of care CAR-T (tisagenlecleucel). Lysophosphatidylcholines, arginine and tryptophan metabolites were common to both cohorts. Overall decreased amino acid levels were correlated with poor disease-free survival. The finding was compared to two other T-cell based therapies; bispecific T-cell engagers and a therapeutic peptide vaccine, which revealed that tryptophan and arginine-pathway metabolites are common to all T-cell directed therapies studied, thus demonstrating the significant robustness of metabolomic profiling. Mechanistic verification of the metabolites identified was performed using CAR-T cells produced using halofuginone (halo), an agonist of the General control nonderepressible 2 (GCN2) agonist. CAR-T cells treated with halo displayed superior expansion in a serial tumour killing assay. Multiomic analysis of these metabolically enhanced CAR-T cells and in vivo functionality will be presented. Overall, this study will demonstrate the utility of functional metabolomics in bedside to bench and back to bedside cell therapy.

LAY SUMMARY

Metabolomic profiles of plasma are predictive of response across several indications and several unique T-cell directed therapies. Metabolic interventions during CAR-T production improve T-cell functionality and fitness. Phase I proof of concept metabolic interventional clinical assessment is underway.

Precise tuning of CAR expression mitigates exhaustion and hyperinflammation in CAR T cell therapy - Poster #372

Presenter: Paul Jerard Layug, Paul Albrechtsen Research Institute- CancerCare Manitoba

Other Authors: Andrew Cuddihy, Huda Alfarekh, Katharina Haigh, Jody Haigh, Yale Michaels

ABSTRACT_ID#2400

ABSTRACT

Chimeric Antigen Receptor (CAR) T therapy has revolutionized treatment for B-cell malignancies. CARs are synthetic proteins that redirect T cell specificity to eliminate cancer cells that express a specific antigen, such as CD19. Despite remarkable clinical success, suboptimal durability and lethal side effects, such as cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS), limit their broader use. One major contributor to these limitations is the supraphysiological level of CAR expression upon transduction, leading to T-cell overactivation, driving rapid dysfunction and hyperinflammation. We set out to identify a “Goldilocks” level of CAR expression where T-cell exhaustion, CRS and ICANS are mitigated, while maintaining effective tumour killing. To fine-tune CD19-CAR expression, we utilized our previously established microRNA silencing-mediated fine-tuners (miSFIT) technology. miSFITs are engineered target sites for endogenous microRNAs, enabling precise control over gene expression. We assessed the effect of CAR levels on T-cell cytotoxicity, exhaustion and inflammation in vitro. Our co-culture assays demonstrated that reducing CAR expression resulted in a decrease in co-expression of exhaustion markers (PD-1 and LAG3), and dampened secretion of pro-inflammatory cytokines (IFN- γ , TNF- α , GM-CSF), without compromising tumour clearance across multiple re-challenges. In vivo, T cells engineered with a miSFIT to express intermediate CAR levels exhibited the best tumour control and significantly extended survival in a leukemic xenograft mouse model. Collectively, these findings demonstrate that CAR expression is a critical determinant of T cell function and that precisely tuning CAR levels with miSFITs could enable the production of safer and more durable CAR T therapies.

LAY SUMMARY

Too much is never good: CAR T cell products are made with too much CAR levels, making them hyperactive. This, in turn, leads to harmful side effects such as cytokine storm, and causes them to crash and deteriorate before finishing the job. We developed a solution, called “miSFIT”: This allows us to control the CAR levels, like a dimmer switch, to a “Goldilocks” zone. This way, CAR levels are neither too high to cause harm nor too low to allow cancer cells to escape, but just right to fight cancer effectively. “Goldilocks” CAR T cells are better and safer therapies: Our preclinical testing indicates that these CAR T cell products performed better and persisted longer, supporting their potential as a better option for cancer patients.

Leveraging Deep Immunopeptidomics to Identify Tumor Antigens from Solid Tumor Tissue Samples - Poster #373

Presenter: Geneece N.Y. Gilbert, University of Calgary

Other Authors: Geneece N.Y. Gilbert, Ted Verhey, Varsha Thoppey Manoharan, Sean Nesdoly, Harmony Smith, Ana Bogossian, Haley Pedersen, Paul Gordon, Jennifer A. Chan, A. Sorana Morrissy

ABSTRACT_ID#2784

ABSTRACT

Cancer immunotherapy is a promising avenue for treating patients who exhaust their current standard lines of care. These therapies work by promoting anti-cancer adaptive immunity. The process of adaptive immunity relies largely upon T cell-mediated recognition of tumor specific or associated peptides presented upon the Major Histocompatibility Complex (MHC). Given longstanding limitations in profiling MHC presented peptides, several computational tools have been developed to instead predict peptide MHC binding. Here, we leverage recent technical advances in proteomics to generate deep immunopeptidomics data from 20 cancer tissue samples and utilize this data to identify tumor antigens. Immunopeptidomics profiling was performed on a Bruker timsTOF Ultra mass spectrometer platform, yielding an average of 19,283 peptides/sample, and a total of 106,079 unique peptides from 12,209 genes across the cohort. Peptides from multiple sources can drive immunogenic recognition, including tumor associated antigens (TAAs), mutated tumor specific antigens (mTSAs), and aberrantly expressed tumor specific antigens (aeTSAs). Here, we built a workflow for personalized immunopeptidomics that incorporates germline variation, variant phasing, and clonality to perform searches for mTSAs, TAAs, and aeTSAs. Initial analyses applied to samples from glioblastoma patients revealed several TAAs abundantly present on patient specific MHC alleles. Next, this work will be applied to aeTSAs and mTSAs to identify a candidate set of high-quality targets for immunotherapy. In addition, this data will be used to comprehensively assess the accuracy of peptide MHC binding prediction tools run on the matched transcriptomes and genomes in this cohort.

LAY SUMMARY

Deep immunopeptidomics data isolated for 20 samples using a Bruker timsTOF Ultra mass spectrometer platform yields on average 19283 peptides per sample, 106079 unique peptides and 12209 unique genes across all samples. Leveraging deep immunopeptidomics allows identification of several highly presented tumor associated antigens in glioblastoma patients, paving the way for improved targeted cancer immunotherapies and a comprehensive assessment of the accuracy of peptide MHC binding prediction tools.

Harnessing Radiation Therapy to Overcome STING Immunotherapy Resistance in Soft Tissue Sarcoma - Poster #374

Presenter: Logan Mahoney, University of Calgary

Other Authors: Carolina Salazar Arcila, Kurt Hildebrand, Arvind Singla, Michael Monument, Karys Hildebrand

ABSTRACT_ID#3165

ABSTRACT

Background STING agonists represent a promising immunotherapeutic approach for soft tissue sarcomas (STS), yet many tumours exhibit resistance due to suppression of innate immune signaling pathways. Strategies to overcome this resistance are critically needed. Radiation therapy (RT) increases cytosolic DNA and type I interferon signaling. We hypothesize that RT may restore STING responsiveness in resistant STS and enhance anti-tumour immunity. Methods We are using two immunocompetent murine STS models: TAO1 (KRAS^{G12D}, Trp53^{-/-}) and KPP1053 (KRAS^{G12D}, Trp53^{-/-}, Pten^{-/-}). TAO1 demonstrates STING pathway activity, while KPP1053 is resistant to STING agonism. Mice bearing established tumours are randomized to E7766 (a clinically relevant macrocycle-bridged STING agonist), RT (1×8 Gy, 3×8 Gy, or 5×6 Gy), or combination therapy. Tumour kinetics and survival are monitored longitudinally. Comprehensive immune profiling is underway, including multiplex cytokine analysis, flow cytometry, and high-dimensional spatial mapping using the Miltenyi MACSima Imaging Platform. Results Findings so far confirm STING monotherapy efficacy in TAO1 and resistance in KPP1053. Ongoing studies are evaluating whether RT, across multiple dosing regimens, enhances tumour control and remodels the immune microenvironment in both models. Spatial analysis will define treatment-specific immune architecture, cellular composition, and organization. Conclusions This ongoing preclinical study investigates RT as a strategy to overcome STING resistance in genetically distinct STS models. By integrating therapeutic outcomes with spatial and immunologic profiling, this work aims to define how RT and STING agonism cooperate to improve anti-tumour immunity in both STING-responsive and STING-resistant sarcomas.

LAY SUMMARY

1. Stimulator of interferon genes pathway-targeted therapies are a promising new class of cancer immunotherapies, but many soft tissue sarcomas (STS) are resistant to them. Our research models show that some tumours fail to activate immune responses through the STING pathway, preventing effective immune-mediated tumour clearance. Understanding and overcoming this resistance is critical for expanding the benefit of STING-based therapies to more patients with STS.
2. Radiation therapy may re-sensitize resistant tumours to STING agonists by enhancing immune activation. Radiation increases cytosolic DNA and type I interferon signaling, which can stimulate STING-related immune pathways. In our preclinical models, we test whether combining radiation with STING therapy slows tumour growth and improves survival compared to either treatment alone.
3. We are also mapping how these treatments reshape the tumour immune microenvironment. Using cytokine profiling, flow cytometry, and spatial imaging, we are identifying how immune cells respond to treatment—where they go, how they interact, and what signals they produce. These insights may guide future strategies to optimize combination therapies in both STING-responsive and STING-resistant sarcomas.

Development of a Lipid Nanoparticle based Anti-BCMA CAR-NK Therapy for Multiple Myeloma - Poster #375

Presenter: Angela Hamie, University of Alberta

Other Authors: Bee Tan, Justine Lai, Carina Debes Marun, Xiuying Hu, Pankaj Tailore, Michael Chu

ABSTRACT_ID#3109

ABSTRACT

Introduction: Chimeric Antigen Receptor (CAR)-T cell immunotherapy has demonstrated promise in treating multiple myeloma (MM), however it carries severe risks such as cytokine release syndrome (CRS), neurotoxicity, and T cell malignancies. Additionally, viral vector based CAR delivery poses concerns of immunogenicity and insertional mutagenesis. CAR-natural killer (NK) cells offer a safer alternative, reduced toxicity and allogeneic "off-the-shelf" use. Furthermore, lipid nanoparticles (LNPs) present a non-viral and cost-effective method for delivering mRNAs, enabling safe and scalable CAR therapy production. **Methodology:** mRNA CAR constructs, consisting of an IgH-recognizing BCMA, were encapsulated in LNPs and transfected into NK-92 cells or primary NK cells isolated from patient and healthy donors. mRNA LNP delivery efficiency was assessed using fluorescent microscopy, flow cytometry and Western blotting. Functional assays will evaluate the cytotoxicity of the modified NK cells against BCMA-expressing MM cell lines. **Results:** A formulation containing lipid 5, β -sitosterol, DSPC, and PEG 2000 (50:38:10:2) demonstrated superior transfection efficiency in NK cells compared to formulations containing SM-102, MC-3, or cholesterol. 70-90% mRNA transfection efficiencies were achieved in NK cells expanded in vitro over 7-10 days. Anti-BCMA CAR expression was confirmed through fluorescent microscopy, flow cytometry, and Western blot. Current studies are evaluating the cytotoxicity of the modified NK cells against MM cell lines. **Conclusions:** Optimized mRNA LNP encapsulation allowed for high transfection efficiencies in primary NK cells, confirming the feasibility of this non-viral approach for CAR engineering. This safer alternative to viral vector-based CAR-T therapy supports the development of allogeneic CAR-NK therapies for MM treatment.

LAY SUMMARY

We are developing a safer and more accessible type of cancer immunotherapy called CAR (Chimeric Antigen Receptor)-T therapy, where a type of cancer fighting immune cells, called T cells, are taken from patients and engineered in the lab with a "CAR" gene that helps them better target and destroy cancer cells. Rather than using traditional T cells, which may cause serious side effects, we are using natural killer (NK) cells—a safer alternative with similar cancer-fighting abilities. Because NK cells can be collected from healthy donors and stored, they offer the potential for a readily available, off-the-shelf therapy that improves patient access. Furthermore, instead of relying on the traditional viruses to deliver the "CAR" gene—an approach that is both costly and potentially risky—we use lipid nanoparticles, which are tiny particles that offer a safer and more scalable delivery method. So far, we have successfully engineered these modified NK cells in the lab and are now testing their ability to target and kill cancer cells in laboratory models, with plans to expand to animal studies. This work brings us closer to developing safer, scalable, and more accessible cancer therapies.

Monday, November 3, 2025

Enhancing Neuroblastoma Treatment with GD2-Targeted CAR-T Cells and Radiolabeled Antibody Therapy - Poster #376

Presenter: Mahlegaha Ghavami Nodeh, UBC

Other Authors: Kevin Ortner, Maryam Aletaha, Cristina Rodríguez-Rodríguez, Erin Tanaka, Annette Patterson, Ramon Klein Geltink, Haniyeh Ghafari Nazari, Justin Bailey, Paul Schaffer, Kirk R. Schultz, Chinten James Lim, Gregor S. D. Reid

ABSTRACT_ID#2687

ABSTRACT

Background & Aim Neuroblastoma (NB), a highly aggressive pediatric cancer of neural crest origin, remains difficult to treat due to tumor heterogeneity and an immunosuppressive microenvironment. Although conventional therapies have improved outcomes in some cases, prognosis remains poor for high-risk patients. GD2-targeted immunotherapy has shown promise, but anti-GD2 monoclonal antibodies alone rarely induce durable remission. This study aims to develop NB xenograft models to evaluate the therapeutic potential of GD2-directed chimeric antigen receptor (CAR-T) cells and radioimmunotherapy using Lutetium-177 (^{177}Lu) and Actinium-225 (^{225}Ac). **Methods** GD2-positive human NB cells were subcutaneously implanted into immunodeficient mice. T cells were isolated from spleens of 129/SvJ mice and activated using CD3/CD28 stimulation. After two days, cells were transduced with a GD2-specific retroviral CAR-T construct. CAR-T functionality was confirmed through in vitro killing assays. In vivo, 1.25×10^6 CAR-T cells were administered intravenously one week after tumor implantation. Separately, the anti-GD2 monoclonal antibody clone 14.G2a was conjugated to DOTA, radiolabeled with ^{177}Lu , and injected into tumor-bearing mice. **Results & Conclusion** CAR-T cells demonstrated ~75% transduction efficiency and induced over 90% tumor cell death in vitro. In vivo, CAR-T treatment significantly reduced bioluminescent signals from GD2-positive tumors compared to controls. Treatment with ^{177}Lu -conjugated anti-GD2 resulted in notable tumor volume reduction. Ongoing studies are assessing the efficacy of ^{177}Lu - and ^{225}Ac -conjugated antibodies, alone and with CAR-T cells. This combinatorial approach, with both modalities showing activity, may enhance inflammation, improve CAR-T infiltration, and yield superior outcomes for high-risk neuroblastoma.

LAY SUMMARY

We are developing a two-pronged treatment for high-risk childhood cancer: Neuroblastoma is a serious cancer that mainly affects young children. Our research combines two powerful tools, engineered immune cells (CAR-T cells) and targeted radioactive antibodies—to attack tumors more effectively. Engineered immune cells (CAR-T cells) can find and kill cancer cells: We use a special receptor to guide the immune cells to a molecule called GD2, which is commonly found on neuroblastoma cells. These CAR-T cells killed over 90% of cancer cells in lab tests and showed strong tumor-fighting ability in mice. Targeted radiation may boost immune therapy results: We also tested a radioactive antibody that delivers radiation directly to tumor cells. This treatment alone shrank tumors in mice. We are now studying how combining both treatments can improve results by creating a more hostile environment for the tumor and helping immune cells work better.

An Inducible System for Rapid Selection of CRISPR-based Genome Editing - Poster #377

Presenter: Jada Curry, University of Calgary

Other Authors: Lou Baudrier, Jethro Langley, Franz Zemp, Fatemeh Mashayekhi, Doug Mahoney, Pierre Billon

ABSTRACT_ID#2907

ABSTRACT

Next-generation CRISPR technologies, like base editing or prime editing, enable multiplex engineering of immune cells creating promising new solutions for cancer patients, as recently demonstrated in refractory cancer patients who entered remission. However, low editing efficiency limits therapeutic applications of multiplex base and prime editing. The inducible caspase 9 (iC9) suicide gene is a safety switch commonly used to eliminate engineered cells in cellular therapies to circumvent potential fatal adverse effects. Here, we repurpose iC9 as an inducible co-selection system that significantly improves multiplex editing efficiency. We demonstrate that co-editing T-cell-specific therapeutic genes, while inactivating iC9 to protect the edited population from apoptosis, results in up to 4-fold enhancement in editing efficiency. Ongoing validation efforts with this system include enhancing the efficiency of high-throughput CRISPR screens, improving anti-tumor efficacy of multiplex engineered CAR-T cells and increasing safety of allogeneic cell therapies. Notably, this innovative co-selection system leverages the FDA-approved iC9 system and non-toxic CID which will facilitate future translations into clinical applications. iC9 as a co-selection system enables high efficiency engineering for primary human T-cells for next-generation cancer immunotherapy.

LAY SUMMARY

- Inducible caspase 9 (iC9) is a tool that can induce cellular death rapidly and efficiently through the addition of a drug. It is currently used to increase the safety of therapies. We repurposed iC9 to be used as a co-selection system. Co-selection systems are based off the knowledge that DNA editing is non-independent: therefore, if a single cell gets one edit it is more likely to get a second edit. Next generation CRISPR technologies can be used to make two edits: one to inactivate iC9 and one to make the edit of interest. This will result in only the edited cells being protected from inducible cellular death.
- We have established iC9 as a co-selection system and validated it in various cell lines, demonstrating up to 4-fold enhancement of precision gene editing across multiple targets and editor types.
- Since iC9 is FDA approved as a safety switch, ongoing work is focusing on applying the co-selection system to enhance gene editing to improve immunotherapy treatments. This includes in vivo experiments to determine the improved functionality of edited CAR-T cells. iC9 has previously been established in T cells, allowing for a clear path to clinical translation.

STING immunotherapy induces immunogenic tumor clearance and protection against disease recurrence in the KP model of soft tissue sarcoma - Poster #379

Presenter: Karys Hildebrand, University of Calgary

Other Authors: Kurt Hildebrand, Jahanara Rajwani, Carolina Salazar Arcila, Michael Monument

ABSTRACT_ID#2577

ABSTRACT

The stimulator of interferon genes (STING) agonist, E7766 can elicit tumor clearance in an orthotopic murine model of soft tissue sarcoma (STS). As lymphocytes are dominant mediators of tumor cell killing and are present in elevated frequencies in E7766 treated STS, we sought to investigate the contributions of CD4+ and CD8+ lymphocytes in the context of tumor clearance and subsequent protection against a model of STS recurrence. C57BL/6 mice bearing STS tumors received a single intratumoral dose of E7766 and were followed for survival in control and CD8+T-cell depleted states. Following survival studies, mice that achieved tumor clearance were re-challenged with an inoculation of STS cells in the contralateral limb. Re-challenged mice were stratified into control, anti-CD8+ and anti-CD4+ antibody depletion groups. Depletion was confirmed via flow cytometry. In a CD8+ depleted state, the therapeutic benefit of E7766 therapy was entirely negated, while non-depleted controls exhibit tumor clearance frequencies of 38%. Following contralateral limb re-challenge, 88% of E7766 surviving mice retained protection against STS. In a CD8+ T-cells depleted state, 100% of survivors continued to demonstrate immunity against STS re-challenge. However, in the context of CD4+ T-cell depletion, 60% of mice developed STS tumors following re-challenge. Ultimately, the translational STING agonist E7766 can elicit immune mediated STS clearance that is initially dependent on CD8+ T-cells, while the adaptive immune protection conferred following STING therapy following re-challenge is largely CD4+ T-cell dependent. Given the anti-tumor efficacy and immunologic protection conferred by E7766, this agent should be considered for STS clinical trials.

Peptide Nanoparticles-Mediated Macrophage Polarization Enhance Anticancer Efficacy of Chemotherapeutics - Poster #380

Presenter: Marya Ahmed, University of Alberta

ABSTRACT_ID#2657

ABSTRACT

Peptide based self-assembled materials are being investigated as a biodegradable option with various biomedical applications such as drug delivery carriers and as biosensors. In this study, we report the mechanism of action of synthetic β -sheet and turn rich macrocyclic HDP that self-assembles in the form of nanoparticles and stimulates naïve and tumor associated macrophages, resulting in the production of pro-inflammatory mediators in macrophage monoculture and in macrophage/triple negative breast cancer (TNBC) co-culture model. Our results show that macrocyclic peptide-based nanomaterials termed as mCA4 engage with toll-like receptors of macrophages, activating downstream pathways in both naïve and IL-4 pre-treated macrophages, and result in the production of pro-inflammatory mediators by the immune cells. The immunomodulatory potential of mCA4 is highly sequence specific and while an inactive analogue prepared by swapping single amino acid in the peptide chain does not lose self-assembly properties of the parent peptide, its impact on immunomodulatory potential is detrimental. The immunomodulatory potential of mCA4 is further confirmed in macrophage monoculture and in macrophage/TNBC co-culture by the proteomics and by western blot analysis. The treatment of macrophage/TNBC co-culture with mCA4 and chemotherapeutics enhanced anticancer efficacies of chemotherapeutics, suggesting immuno-adjuvant like properties of materials for TNBC treatment.

LAY SUMMARY

Synthetic macrocyclic immunomodulatory peptide based self-assembled nanoparticles are non-toxic towards macrophages but engage with the Toll-like receptors and activate downstream signalling pathways in naïve and tumor associated macrophages. The immunomodulatory properties of the peptides are highly sequence specific and substituting single amino acid causes complete loss of immunomodulatory efficacies, without compromising the self-assembly properties of peptide into nanoparticles. Co-delivery of peptide nanoparticles and chemotherapeutics in macrophage/TNBC co-culture show enhanced anticancer efficacies by the combined effect of chemotherapeutics and immunomodulatory mediators production by the macrophages.

Investigating the role of STING signaling in immune and non-immune cellular compartments on survival phenotypes in the KP model of soft tissue sarcoma - Poster #381

Presenter: Karys Hildebrand, University of Calgary

Other Authors: Kiran Narta, Kurt Hildebrand, Carolina Salazar Arcila, Sorana Morrissy, Michael Monument

ABSTRACT_ID#2581

ABSTRACT

The tumor microenvironment (TME) of soft tissue sarcoma (STS) is traditionally abundant in immunosuppressive myeloid lineage cells which is hypothesized to contribute to systemic therapy resistance. Activation of the STimulator of INterferon Genes (STING) pathway is an emerging immunotherapy known to repolarize myeloid lineage cells towards anti-tumor phenotypes. In a murine model of STS, intra-tumoural (i.t.) E7766 administration, which activates both mouse and human STING, resulted in 38% of mice eradicating STS. Herein, we examined in which cell types STING signaling via E7766 administration is required for inducing anti-STs responses. Syngeneic STS tumors were engrafted in STING deficient mice, treated with E7766 (i.t.) and followed for survival. STING signaling in STS was evaluated by generating a STING knockout cell line, which was engrafted in immune competent mice, treated with E7766, and followed for survival. Single cell RNA sequencing (scRNAseq) was used to characterize interferon stimulated gene (ISG) signatures in numerous cell types in the STS TME at 6-hours and 1-week following E7766 treatment. Fifty-seven percent of immune competent mice bearing STING deficient STS tumors and 0% of STING deficient mice exhibited tumor clearance following E7766 treatment. Additionally, scRNAseq revealed that ISG scores were most significantly elevated in myeloid cells of the STS TME. STING signaling in non-malignant host cells is critical to the anti-tumor phenotype induced by E7766. Furthermore, ISG signaling was significantly elevated in myeloid lineage cells, suggesting STING signaling in this traditionally immunosuppressive cell type may be a potential mechanism for E7766 mediated tumor clearance.

Expanding precision cancer immunotherapy targets with transcriptomics - Poster #382

Presenter: Mathieu Quesnel-Vallières, Université de Sherbrooke

ABSTRACT_ID#2651

ABSTRACT

Cancer antigens can arise from genetic alterations, the reactivation of developmentally-regulated genes, or the use of aberrant RNA splice sites. The expression of cancer antigens confers a molecular specificity that can be leveraged to precisely target cancer cells without affecting normal cells. Emerging immunotherapies capitalize on this feature to educate the patient's own immune system against the tumour or to engineer synthetic receptors that activate immune cells upon binding to cancer cells. A major limitation to the application of these novel technologies is the scarcity of known actionable cancer antigens. We developed a cancer antigen discovery pipeline that uses genomic data to identify neoantigens and transcriptomic data to identify antigens derived from aberrant RNA splicing or gene expression. We compare cancer samples to 86 primary normal human tissue and cell types to highlight cancer-specific splicing variations and gene expression in each individual cancer sample. Our platform can identify potential chimeric antigen receptor (CAR)-T cell targets or cancer epitopes to include in therapeutic vaccines. We analyzed pediatric cohorts of acute myeloid leukemia and neuroblastoma to identify CAR-T targets, as well as adult cohorts of cholangiocarcinoma and pancreatic ductal adenocarcinoma to design personalized therapeutic vaccines. Select candidates are undergoing experimental validation to assess their potential as targets for CAR-T therapy in acute myeloid leukemia. Future work will assess the immunogenicity of splicing antigens identified in solid tumours using primary samples and tumour organoids, and the availability of cancer-specific splicing antigens in other high-risk cancer cases.

MHC-I-Expressing Engineered Vaccinia Virus as a Precision Medicine Cancer Vaccine - Poster #383

Presenter: Shae Komant, University of Alberta

Other Authors: Jun Li Wang, Cyril Alex, Nicole Favis, Ryan Noyce, Troy Baldwin

ABSTRACT_ID#2576

ABSTRACT

Oncolytic viral therapy uses viruses to selectively infect and kill cancer cells, while also aiming to induce anti-tumor immune responses. However, the ability of current oncolytics to stimulate anti-tumor responses varies, therefore novel means of inducing immune activity is necessary. Vaccinia virus (VACV) is a promising oncolytic candidate due to its established safety profile and ability to produce potent T-cell responses. Here, we use modified VACV expressing MHC-I with a defined tumor peptide and β 2-Microglobulin as a single chain trimer (SCT), and the T-cell co-stimulatory molecule CD80. We examined if injections of EMT6 breast cancer cells with VAC-SCT can lead to tumor regression in vivo. In the EMT6 model, a previously described tumor specific antigen, E22, was expressed by the SCT and three models were examined: intratumoral virus treatment, bilateral tumor implantations and percutaneous virus treatment. In all models virus treatment led to E22 specific CD8+ T-cell infiltration in the tumor and were found in the spleen. 70% of mice treated with VACV-SCTE22 cleared their tumor after intratumoral virus treatment. When tumors were implanted bilaterally, 27% of animals cleared both tumor sites. Percutaneous administration of virus led to the clearance of tumor in 25% of animals. Further, all mice that cleared primary tumor also cleared tumor implants upon challenge. Finally, we treated EMT6 tumor bearing mice with VACV-SCTE22 in combination with α PDL-1 antibodies which further enhanced animal survival, surpassing 75%. These data highlight the potential to bolster anti-tumor T-cell responses using immune stimulating VACV delivery of a defined peptide-MHC-I complex.

LAY SUMMARY

These data highlight the potential to bolster anti-tumor T-cell responses using Vaccinia Virus as a delivery vector of a defined peptide-MHC-I complex. Engineered Vaccinia Virus Boosts Immune Attack on Tumors: Our modified Vaccinia virus (VACV) was used to deliver a tumor-specific signal, E22 peptide in the context of the MHC-I complex, and a T-cell stimulating molecule directly to breast cancer cells in mice. This led to strong anti-tumor T-cell responses that could be found within the tumors of treated animals. High Tumor Clearance in Mice: 70% of mice completely cleared their tumors after direct injection of modified VACV into the breast tumors. Further, virus administration directly into tumors or delivering the virus through the skin also cleared tumors at distant sites, indicating a strong, systemic immune response. Combination with Immunotherapy Improves Results: When the viral treatment was combined with α PD-L1 antibodies (a common cancer immunotherapy), survival rates improved even further—over 75% of mice survived.

Microbiome-derived hydroxyphenyl propanoates enhance antitumour immunity by potentiating gasdermin D activity in tumour-associated myeloid cells - Poster #384

Presenter: Saif Sikdar, University of Calgary

Other Authors: Dae-Sun Kim, Tie Wang, Sacha Benaoudia, Jahanara Rajwani, Kristofor Ellestad, Pauline Douglas, Ruzena Filandrova, Ted Verhey, Katrina Ellestad, Marija Drikic, Darren Derksen, Ian Lewis, Marco Gallo, Sorana Morrissy, David Schriemer, Kathy McCoy, Franz Zem

ABSTRACT_ID#3176

ABSTRACT

The microbiome plays a critical role in immune health and response to immune-stimulating treatments, including cancer immunotherapy. However, the molecular mechanisms by which commensal microbes influence cancer immunology remain poorly understood. Here, we report a class of microbiome-derived metabolites called hydroxyphenyl propanoates (HPP) that enhance tumour immune surveillance and response to immune checkpoint blockade (ICB) therapy in mice. HPPs function as potentiators of innate immune signalling in tumour-associated myeloid cells by promoting cleavage of the pore-forming protein gasdermin D (GSDMD), a critical effector of inflammasome function. Increased secretion of proinflammatory cytokines, such as IL-1 family members, in turn heightens autocrine and paracrine inflammatory signaling in tumour-infiltrating leukocytes. HPP supplementation increases anticancer T cell function, improves disease control and extends overall survival (OS) in tumour-bearing wildtype but not Gsdmd knockout mice. In humans, GSDMD activation is associated with a favourable response to PD-1 blockade therapy in patients with advanced stage melanoma. Our study uncovers a molecular mechanism of regulation over host antitumour immunity that is modifiable and can be harnessed for improved cancer immunotherapy.

LAY SUMMARY

Specific gut microbes can help fight cancer: A particular composition of the gut microbiome produces a class of compounds called HPPs, which enhance the immune system's ability to eliminate tumour cells. Boosting immune activity in tumours: HPPs uniquely modulate the immune protein GSDMD enabling immune cells to release bioactive cytokines without undergoing cell death. This process activates other cancer-fighting immune cells within the tumour microenvironment. Potential to improve cancer treatment: In both mouse models and human studies, increased activity of this immune pathway correlates with improved outcomes during PD-1/PD-L1 immunotherapy. Notably, HPPs remain effective in hosts with diverse microbiomes and can overcome the negative impact of antibiotics on immunotherapy, highlighting their potential for clinical application in cancer treatment.

Radiation-driven modulation of the tumour microenvironment to boost CAR T efficacy - Poster #385

Presenter: Lisa Zhan, BC Cancer Research Institute

Other Authors: Meredith Clark, Che-Min Lee, Rocky Shi, Pauline Loos, James Piret, Laura Evgin, Kevin Bennewith

ABSTRACT_ID#2944

ABSTRACT

The solid tumour microenvironment (TME) presents significant challenges to effective CAR-T cell therapy. Combining CAR T cells with other modalities, such as ionizing radiation, may improve outcomes in solid tumours. Our research explores the synergistic potential of targeted radiation to enhance CAR-T cell efficacy. We hypothesized that optimizing radiation timing and doses in EGFRvIII-B16F10 tumours in C57Bl/6 mice would enhance CAR-T cell infiltration and efficacy by modulating aberrant tumour vasculature. First, we identified a radiation schedule that improves vascular perfusion. Using immunofluorescence microscopy, we assessed vascular changes after radiation doses ranging from 2 Gy to 12 Gy, at intervals from 4 hours to 3 days post-radiation. Next, we evaluated whether radiation increased CAR-T cell infiltration and anti-tumour activity. Tumour growth delay and survival were measured across treatment groups. Tumour-infiltrating CAR-T cells was assessed via flow cytometry. Our results demonstrated a significant increase in vascular perfusion 4 hours after a single targeted 8 Gy dose. When EGFRvIII-targeting CAR-T cells were administered 4 hours post-radiation, tumour growth was significantly delayed compared to mice that did not receive prior radiation. Additionally, survival was markedly improved in the combination treatment group compared to all other treatment groups. Our findings demonstrate that radiation-mediated modulation of the TME can potentiate CAR-T cell therapy efficacy to maximize anti-tumour response in our tumour model. These findings demonstrate that radiation-mediated modulation of the TME can potentiate CAR-T cell therapy, enhancing anti-tumour response in our model. This strategy provides a promising avenue for improving CAR-T cell efficacy against solid tumours.

LAY SUMMARY

Engineered immune cells cannot efficiently treat solid tumours. Combining immune cells with other therapies such as radiation can improve outcome. We established a radiation schedule capable of increasing immune cell trafficking into the tumour. Delivery of engineered immune cells following radiation resulted in an increase in tumour regression and survival.

Repurposing telmisartan as a novel immunomodulatory adjuvant to enhance immune checkpoint blockade in triple-negative breast cancer - Poster #387

Presenter: Meredith Clark, BC Cancer Research Institute

Other Authors: Lisa Zhan, Che-Min Lee, Rachel Cederberg, Kevin Bennewith

ABSTRACT_ID#2958

ABSTRACT

Triple-negative breast cancer (TNBC) remains a major clinical challenge due to its aggressive progression and limited response to immunotherapies like immune checkpoint blockade (ICB). Addressing this urgent need, we present the first evidence that telmisartan – a widely used and unique antihypertensive drug – can be repurposed as a powerful immunomodulatory adjuvant for cancer immunotherapy. In immunocompetent TNBC models, single-agent telmisartan treatment induced a robust, CD8+ T cell-dependent delay in tumour growth, an effect absent in immunodeficient hosts. Strikingly, telmisartan treatment led to a marked increase in the presence, activation, and cytotoxic function of CD8+ T cells within the tumour microenvironment, as revealed by high-dimensional flow cytometry and ex vivo functional assays. When combined with anti-PD-1 ICB, telmisartan synergistically amplified anti-tumour immunity, resulting in robust tumour control and enhanced activation of effector T cells. Mechanistic studies demonstrated that telmisartan directly reprograms CD8+ T cell activation and effector function, a property not shared by other ARBs. Ongoing studies are leveraging transcriptomic and genetic approaches to unravel the molecular underpinnings of telmisartan's immunomodulatory effects and to identify additional immune cell populations responsive to this therapy. Given telmisartan's established clinical safety and accessibility, these findings lay the groundwork for rapid translation into clinical trials, offering a transformative and cost-effective strategy to overcome immune resistance in TNBC and potentially other immunologically "cold" tumours.

LAY SUMMARY

We have found that a common blood pressure medication, telmisartan, can boost the body's immune response against aggressive triple-negative breast cancer by activating cancer-fighting T cells. When used together with immune checkpoint therapy (anti-PD-1), telmisartan leads to stronger tumour control than either treatment alone, suggesting a promising new combination approach for hard-to-treat cancers. Because telmisartan is already widely used and well-tolerated, these findings could quickly pave the way for new, cost-effective treatment options for patients with advanced breast cancer and potentially other cancers that don't respond well to current immunotherapies.

Capturing live interactions between T cells and AML in the bone marrow microenvironment using intravital imaging - Poster #388

Presenter: Sadaf Mazhab Jafari, University Health Network

Other Authors: Raheleh Niavarani, Xi Lei, Lyndsey De Guzman, Ralph DaCosta

ABSTRACT_ID#3356

ABSTRACT

Acute myeloid leukemia (AML) is an aggressive hematological malignancy characterized by the clonal proliferation of myeloid progenitor cells in the bone marrow. With a poor overall survival rate of only 27% at 5 years, AML poses significant treatment challenges. AML blasts sustain their survival by remodelling the bone marrow microenvironment (BME), creating a leukemia niche that disrupts normal hematopoiesis and promotes leukemogenesis. Despite the critical role of immune cells in tumour progression, the spatiotemporal dynamics of T cells within the AML BME remain poorly understood. Current models do not effectively capture the real-time, spatial changes within the BME throughout disease progression. Understanding the immune landscape in AML is crucial for improving patient outcomes. Specifically, identifying the time point at which the immune environment shifts toward immunosuppressive and anergic T cells could inform the optimization of treatment strategies, such as immune checkpoint inhibitors and T-cell therapies. This highlights the need for advanced imaging techniques, such as intravital microscopy, to study T-cell distribution and migration within the AML tumour microenvironment. In this study, I will use the femur window chamber (FWC) and intravital fluorescence (IVF) microscopy platform to quantitatively assess T-cell infiltration and interactions with AML cells across different stages of AML progression. By tracking the dynamics of CD8+ and CD4+ T cells in real-time, this study aims to identify the critical time point at which the immune shift occurs. The ultimate goal is to provide insights into the optimal timing of therapeutic interventions that restore immune function and improve patient survival.

LAY SUMMARY

This project aims to investigate the time-dependent spatiotemporal dynamics and functional behaviour of CD8+ and CD4+ T cells during the progression of acute myeloid leukemia (AML). Understanding these dynamic immune changes may reveal optimal therapeutic windows for restoring immune function. To enable this, I will employ a novel imaging strategy that combines a femur window chamber (FWC) model with intravital fluorescence (IVF) microscopy, allowing real-time visualization and quantification of T-cell infiltration, distribution, and interactions within the AML tumor microenvironment.

Monday, November 3, 2025

Methotrexate preserves anti-PD1 therapy efficacy in cancer while controlling arthritis in a mouse model of ICI-arthritis - Poster #390

Presenter: Sonia del Rincón, Lady Davis Institute/McGill University

Other Authors: Marie Hudson, Theo Papadopoulos

ABSTRACT_ID#2429

ABSTRACT

Rationale: Immune checkpoint inhibitors (ICIs) have transformed cancer treatment but are often associated with immune-related adverse events (irAEs), including arthritis, affecting patient survivorship. While steroids and immunosuppressants like methotrexate (MTX) are used to manage ICI-arthritis, steroids may compromise the potential life saving effects of ICIs. This study investigates how MTX affects ICI-mediated anti-tumor immunity, with the goal of improving patient quality of life. **Methods:** Tumor-bearing mice were treated with vehicle or MTX ± ICI. Arthritis was induced after tumor formation, and paw inflammation was measured. Tumors were immune-phenotyped, and CD8+ T-cell exhaustion was modeled ex vivo. **Results:** MTX preserved the anti-tumor efficacy of the ICI anti-PD1. Encouragingly, MTX ± ICI reduced accelerated arthritis resolution. Unexpectedly, MTX enhanced memory CD8+ T cell formation and reduced expression of T cell exhaustion markers (TIM3, LAG3). Using ex vivo T cell assays, we showed that MTX reduced CD8+ T-cell exhaustion, which was supported by RNA sequencing data. **Clinical Impact:** We expect our results will ultimately improve patient survivorship. In summary, we established a tumor-arthritis model to study the impact of immunosuppressive therapy on ICI responses. MTX preserved anti-PD1 anti-tumor activity while reducing inflammation, supporting its potential as a treatment for patients with ICI-arthritis.

LAY SUMMARY

The fourth pillar of cancer therapy known as immune checkpoint inhibitor (ICI) therapy can cause toxic side effects that reduce quality of life. Patient survivorship may be impacted by our research study. Our novel pre-clinical mouse model is used to test the effects of drugs that dampen toxic side effects, while maintaining the anti-cancer efficacy of the ICI therapy.

Chimeric antigen receptor macrophages (CAR-M) sensitize glioblastoma models to temozolomide and immune checkpoint blockade combination therapy - Poster #391

Presenter: Shannon Snelling, University of Calgary

Other Authors: Joanna Pyczek, Xueqing Lun, Kristofor Ellestad, Franz Zemp, Laura Mah, Sorana Morrissey, Douglas Mahoney, Jennifer Chan

ABSTRACT_ID#2800

ABSTRACT

Glioblastoma (GBM) is fatal brain cancer in which immunotherapeutic approaches, including immune checkpoint blockade (ICB), have demonstrated little efficacy. Among myeloid phenotypes in GBM, the presence of inflammatory myeloid cells is associated with improved prognosis. Emerging evidence suggests that chimeric antigen receptor macrophages (CAR-M) reverse immunosuppression and sensitize previously resistant peripheral solid tumours to anti-PD-1 ICB. Whether CAR-M can sensitize GBM to ICB, however, has not been explored and is uncertain given the distinct immunologic features of the brain. We have developed syngeneic CAR-M carrying a CAR with novel signaling domains and a scFV against the GBM neoantigen EGFRvIII and have tested the CAR-M in vitro and in vivo. anti-EGFRvIII CAR-M selectively kill EGFRvIII expressing murine GBM cells and secrete inflammatory chemokines and cytokines in vitro. To test CAR-M in vivo, we developed a syngeneic model of GBM (EGFRvIII-OE/CDKN2A-KO/PTEN-KO) which is sensitive to but not cured by temozolomide (TMZ) chemotherapy and is resistant to ICB therapy (anti-PD1 + anti-CTLA4). To test whether CAR-M sensitize GBM to ICB, we delivered intracranial CAR-M or control macrophages (1x10⁶ cells total) on day 14 post-tumour cell implant on the background of TMZ (50mg/kg oral gavage daily day 7-12) and ICB (200ug anti-PD-1 and 200ug anti-CTLA4 intraperitoneally day 7,10,13,16,19). Control macrophage group mice all died by day 40, whereas two CAR-M treated mice survived to day 300 (N=5/group). These results demonstrate that our CAR-M are functionally active in the presence of EGFRvIII expressing cells and have the potential to sensitize GBM to TMZ/ICB therapy.

LAY SUMMARY

Glioblastoma (GBM) is a devastating brain cancer that has no cure. Large phase 3 clinical trials testing single agent immunotherapy for GBM have been disappointing and the field has concluded that combination therapy including immunotherapy will likely be needed to treat GBM. We wanted to know whether a new immunotherapy called chimeric antigen receptor macrophages (CAR-M) can be combined with standard chemotherapy and immune checkpoint blockade immunotherapy to improve GBM treatment. This work discovered that CAR-M therapy in combination with standard chemotherapy and immune checkpoint blockade can prolong survival in mouse models of GBM compared to standard chemotherapy and immune checkpoint blockade alone. We hope in the future that this work will contribute to people living well for longer with brain cancer.

Reovirus as a novel platform for orally available cancer vaccine - Poster #392

Presenter: Tommy Alain, CHEO Research Institute, University of Ottawa

Other Authors: Salar Pashangzadeh, Negar Sharifi, Eknoor Walia, Nikolas Martin, Ki-Hoon Song, Thet Fatica, An-Dao Yang, Nathalie Earl, Julia Petryk, Zakia Djaoud, Huy-Dung Hoang, John Bell, Dong Guk Park, Shawn Beug, Nam-Hyuk Cho, Xiao Xiang

ABSTRACT_ID#2707

ABSTRACT

Reovirus as a novel platform for orally available cancer vaccine The development of vaccines capable of inducing both systemic and mucosal immunity remains a critical goal in immunology, particularly for addressing infections at mucosal surfaces and advancing cancer immunotherapy. Here, we present a proof-of-principle study demonstrating that a genetically engineered Reovirus Type 3 Dearing (T3D)-based vaccine can elicit robust immune responses when delivered via oral or intraperitoneal (IP) routes. By modifying the S1 gene encoding the sigma 1 protein, we replaced the globular head domain responsible for JAM-A receptor binding with antigens, including the receptor-binding domain (RBD) of SARS-CoV-1 and the model antigen ovalbumin (OVA). This engineering enabled the reovirus to present antigens to the immune system, leveraging its natural immunostimulatory properties. Our results show that the vaccine effectively induced antigen-specific IgG and CD8+ T cell responses following IP and oral administration, with the oral route uniquely stimulating mucosal IgA production. Using a B16F10-OVA melanoma mouse model, we demonstrated that the vaccine prevented tumor implantation and inhibited established tumor progression, irrespective of the delivery route. These findings highlight the versatility of this platform for infectious disease prevention and cancer immunotherapy. The ability to deliver the vaccine orally, stimulating both mucosal and systemic immunity, represents a significant advancement in vaccine technology and broadens its potential applications.

LAY SUMMARY

We are proposing an innovative strategy to ingest modified Reoviruses exposing cancer antigens, to both treat and prevent cancer by enhancing anti-tumour immune responses. This approach would be safe, orally available, and effective also when combined with other immunotherapies.

CD4-Targeting CAR-Engineered Double-Negative T Cells for the Treatment of Acute Myeloid Leukemia - Poster #393

Presenter: Shanshan Wang, University of Calgary

Other Authors: Michele Nawata, Juan Arteaga, Laura Mah, Heewon Seo, Colleen Anderson, Kyle Potts, Lynn Savoie, Michelle Geddes, Etienne Mahe, Sonia Cerquozzi, Jennifer Chan, Sorana Morrissy, Franz Zemp, Douglas Mahoney, Jongbok Lee

ABSTRACT_ID#2459

ABSTRACT

Acute myeloid leukemia (AML) is a highly aggressive hematologic malignancy with poor prognosis, particularly in relapsed patients. While chimeric antigen receptor (CAR)-T cell therapy has shown success in other blood cancers, its application in AML remains challenging due to the antigenic similarity between AML blasts and normal hematopoietic stem/progenitor cells (HSPCs), resulting in significant hematopoietic toxicity. Gene set enrichment analysis of AML patient cohorts reveals that elevated CD4⁺ T cell levels in the bone marrow are associated with inferior overall survival. Moreover, single-cell transcriptomic analyses show that CD4 is preferentially expressed on AML blasts but not on HSPCs, making it an attractive CAR target. Targeting CD4 may not only eliminate leukemic cells but also favorably reshape the immunosuppressive bone marrow microenvironment. We employ double-negative T cells (DNTs)—a rare T cell subset lacking CD4 and CD8—as the platform for CD4-targeted CAR (CAR4) therapy. As DNTs are inherently CD4⁺, they are resistant to fratricide and well-suited for targeting CD4⁺ AML. Previous Phase I clinical data support the safety and preliminary efficacy of DNTs in AML, though limited potency was observed in high-tumor-burden settings. To improve therapeutic efficacy, we engineered CAR4-DNTs incorporating CD3ζ paired with either 4-1BB or CD28 co-stimulatory domains. In vitro studies demonstrate that CAR4-DNTs—particularly those with 4-1BB—exhibit enhanced and sustained cytotoxicity against AML cell lines and primary samples compared to untransduced DNTs. Notably, CAR4-DNTs mediated more durable anti-leukemic responses. These findings support further development of CAR4-DNTs as a promising therapeutic strategy for relapsed AML.

LAY SUMMARY

Identifying a new target in leukemia: Our research found that a molecule called CD4 is often found on the surface of leukemia cells in patients with acute myeloid leukemia (AML). Patients with higher CD4 levels tend to have worse survival outcomes. Importantly, CD4 is not present on healthy blood stem cells, making it a promising and specific target for therapy. Engineering safer immune cells for treatment: We are developing a new immunotherapy using double-negative T cells (DNTs)—a rare type of immune cell that naturally lacks CD4 and CD8 proteins. Because they do not have CD4 themselves, DNTs can be safely engineered to recognize and kill CD4-positive leukemia cells without attacking each other or healthy stem cells. Promising early results in the lab: We have created several versions of CD4-targeting CAR-DNTs, each with different internal “signaling modules” to boost their killing ability. In lab studies, these modified DNTs showed strong and sustained killing of leukemia cells, including some that do not express CD4, which was unexpected and encouraging. This suggests they may be effective in a broader group of AML patients, especially those with relapsed or treatment-resistant disease.

Investigating differential immune responses induced by the TICE and Moscow strains of BCG in a murine model of non-muscle invasive bladder - Poster #394

Presenter: Wesley Geerlinks, Queen's University

Other Authors: Victoria Velazquez Becerra, Alison Hermanutz, Priyanka Yolmo, Kartik Sachdeva, Gwenaëlle Conseil, D. Robert Siemens, Madhuri Koti

ABSTRACT_ID#3220

ABSTRACT

High-risk non-muscle invasive bladder cancer (NMIBC) is treated using intravesical Bacillus Calmette-Guérin (BCG) immunotherapy. Despite its known efficacy, over 50% patients experience early recurrence or progression to higher stage. The ongoing shortage of BCG TICE® in North America has led to a renewed interest in understanding strain-associated differences in clinical outcomes. The Moscow strain of BCG is currently being evaluated in an ongoing non-inferiority trial in Canada. Phylogenetic differences among the early (Moscow) and late (TICE) strains of BCG are well established with regard to their T cell epitopes and immunogenicity. This study was conducted to determine whether the two BCG strains cause differential induction of systemic immune responses and ultimately alter recruitment of immune cells to the bladder microenvironment upon intravesical administration. Aging female mice exposed to bladder specific carcinogen were treated with the two strains of BCG via three weekly intravesical instillations. Bulk-RNA sequencing was performed on whole bladders collected at 24-hours and 1 week post 1st instillation. Spatial immune profiling using multiplex immunofluorescence was performed on whole bladder sections following treatment completion. Multiplex cytokine profiling of plasma collected at different time points was performed. Systemic immune profiling was performed on immune cells isolated from bone marrow, blood and spleen at various time points using flow cytometry. Transcriptomic profiling of bladder revealed differential activation of immune-related pathways. Bladders of mice treated with Moscow strain showed higher T cell infiltration. Further studies are required to evaluate BCG strain-associated differences in the bladder immune microenvironment and clinical outcomes of patients.

LAY SUMMARY

Different strains of BCG may differentially activate systemic immune responses upon repeated intravesical instillation. Moscow strain of BCG leads to higher T cell infiltration in the bladder microenvironment compared to the TICE strain, in a pre-clinical model of bladder cancer. BCG strain associated differences in post-BCG bladder immune microenvironment may influence overall treatment outcomes.

Systemic CD4+-Dominant Immune Response Following Liver Resection in Patients with Colorectal Cancer Liver Metastases - Poster #395

Presenter: Shuo Ren, McGill University Health Centre

Other Authors: Oran Zlotnik, Migmar Tsamchoe, Jessica Bloom, Anastasia Tsatoumas, Vered Domankevich, Stephanie K Petrillo, Anthoula Lazaris, Peter Metrakos

ABSTRACT_ID#2688

ABSTRACT

Purpose: Colorectal cancer liver metastases (CRLMs) are a key driver of systemic immunosuppression and a critical determinant of poor prognosis in affected patients. While surgical resection remains the mainstay of treatment, the intricate dynamics of the immune landscape post-resection remain insufficiently characterized. This study aims to delineate the immune reprogramming that occurs following liver resection in CRLMs patients, offering insights into potential therapeutic strategies. **Experimental Design:** Peripheral blood samples were collected from CRLMs patients prior to and following liver resection, once disease-free status was confirmed post-surgery. Immune populations from both adaptive and innate immunity were evaluated via multiparameter flow cytometry, using FlowJo and FlowAI software for analysis. **Results:** Post-surgery, circulating CD4+ T cells, CD28+ CD4+ T cells, and the CD4+/CD8+ T cell ratio were significantly increased. TIM3+ CD4+ T cell levels significantly decreased, while PD-1+ CD4+ T cells exhibited a downward trend, though the change was not statistically significant. No significant changes were observed in the myeloid cell subsets examined pre- and post-surgery; however, elevated levels of S100A9+ M-MDSCs were found in recurrent patients post-surgery. **Conclusions:** Immune reprogramming, characterized by a CD4+ T cell-dominant response and a reduction in the exhausted phenotype, reveals potential mechanisms underlying the reversal of systemic immune suppression following liver metastasis resection. These findings highlight the promise of adoptive CD4+ T cell therapy, particularly when combined with immune checkpoint inhibitors, as a compelling therapeutic strategy to enhance antitumor immunity.

LAY SUMMARY

1. We identify the potential mechanisms driving the reversal of systemic immune suppression following liver metastasis resection. Given the observed systemic immune remodeling, characterized by an increase in CD4+ T cells and alterations in immune checkpoint expression after liver resection, adoptive CD4+ T cell therapy—especially in combination with immune checkpoint blockade—emerges as a promising strategy to enhance immune responses and reduce recurrence in CRCLM patients. This approach underscores the pivotal role of CD4+ T cells, not only as helpers but also in directly driving cytotoxic responses, thereby enhancing anti-tumor immunity after surgery. However, larger-scale clinical trial with extended follow-up is essential to validate these findings and optimize treatment protocols for personalized patient care.
2. In this study, we found that recurrent patients exhibited higher S100A9+M-MDSCs expression following liver resection. This finding aligns with our earlier work , which demonstrated that elevated levels of circulating extracellular vesicle (EV)-associated S100A9, originating from MDSCs, were linked to the poorest prognosis in patients with colorectal cancer liver metastases.

Investigating the Use of PCSK9 Inhibitor Alirocumab for use in Combination Therapy with PD-1 Inhibitors to Treat Melanoma - Poster #396

Presenter: Madison Wickenberg, University of Alberta

Other Authors: Suha Jarad, Dawei Zhang, John Walker, Paul LaPointe

ABSTRACT_ID#2347

ABSTRACT

Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a serine protease responsible for cleavage and endocytosis of the low-density lipoprotein receptor (LDLR). Therefore, PCSK9 inhibitors have been developed for use in patients with high cholesterol. Recent studies have suggested that PCSK9 may also mediate internalization of the major histocompatibility complex 1 (MHC1). Peptides presented in MHC1 provide information to immune cells regarding whether the presenting cell is “foreign” or “self”. MHC1 presentation is highly relevant in cancer cells, as the presentation of novel/mutated peptides allows for T-cell recognition and subsequent destruction of the cancer cells. Inhibiting PCSK9 activity will prevent MHC1 internalization and surface downregulation, leading to increased immune detection of tumours. As a result, we are investigating the use of PCSK9 monoclonal antibody, alirocumab, for use as a cancer immunotherapy. My recent work demonstrates that PCSK9 regulates internalization of MHC1 in melanoma cells, and that this effect is not seen for other immunological receptors. Additionally, I demonstrated that the total protein levels of MHC1 are mediated by PCSK9, corresponding with the concept that PCSK9 causes MHC1 trafficking to lysosomes in a similar manner to its regulation of the LDLR. Preliminary findings also suggest that melanoma cells express an intrinsic level of PCSK9, which may be protective by downregulating MHC1. This emphasizes the need for further research into PCSK9 in cancer, and the potential for alirocumab as an immunotherapy, specifically in combination with existing checkpoint inhibitors.

LAY SUMMARY

Alirocumab, an antibody originally studied for use to treat high cholesterol (hypercholesterimia), has also been shown to decrease tumors in patients. This antibody binds to proprotein convertase subtilisin/kexin type 9 (PCSK9), the protein responsible for downregulating levels of the low-density lipoprotein receptor (LDLR). Recent studies have now shown that PCSK9 also downregulates expression of major histocompatibility complex 1 (MHC1). MHC1 is a receptor important for immune cell recognition of cancer cells. Since alirocumab prevents MHC1 reduction on tumor cells, immune cells can more effectively recognize and target the cancer cells for destruction. The current standard of care treatment for patients with melanoma is immunotherapy, a form of cancer therapy that focuses on boosting immune activation through modulation of various pathways. However, not all patients respond to the most commonly used immunotherapy treatments, so alternatives and combination of multiple treatments are being investigated. Therefore, better understanding the role of PCSK9, and ultimately alirocumab, in melanoma cells is an important area of research as a potential therapeutic option.

The glycosyltransferase ST3GAL4 drives immune evasion in acute myeloid leukemia by synthesizing ligands for the glyco-immune checkpoint receptor Siglec-9 - Poster #397

Presenter: Simon Wisnovsky, University of British Columbia

ABSTRACT_ID#2645

ABSTRACT

Immunotherapy has demonstrated promise as a treatment for acute myeloid leukemia (AML). However, there is still an urgent need to identify new molecules that inhibit the immune response to AML. Most prior research in this area has focused on protein-protein interaction interfaces. While carbohydrates also regulate immune recognition, the role of cell-surface glycans in driving AML immune evasion is comparatively understudied. The Siglecs, for example, are an important family of inhibitory, glycan-binding signalling receptors that have emerged as prime targets for cancer immunotherapy in recent years. In this study, we find that AML cell lines and patient-derived xenografts express ligands for the receptor Siglec-9 at high levels. Integrated CRISPR genomic screening and clinical bioinformatic analysis identified the glycosyltransferase ST3GAL4 as a potential driver of Siglec-9 ligand expression in AML. Depletion of ST3GAL4 by CRISPR-Cas9 knockout (KO) dramatically reduced the expression of Siglec-9 ligands in AML cells. Mass spectrometry analysis of cell-surface glycosylation in ST3GAL4 KO cells revealed that Siglec-9 primarily binds N-linked sialoglycans on these cell types. Finally, we found that ST3GAL4 KO inhibits AML progression in vivo by sensitizing cells to phagocytosis by Siglec-9-expressing macrophages. This work reveals a novel axis of immune evasion and implicates ST3GAL4 as a next-generation target for immunotherapy in AML.

LAY SUMMARY

All cells in our body are coated with sugar molecules. In cancer, the chemical structure of these molecules is dramatically altered. Changes in cell-surface glycosylation can prevent our immune cells from finding and destroying cancer cells. This study shows that acute myeloid leukemia (AML) cells show a distinct change in their cell-surface carbohydrate structures that makes them resistant to killing by immune cells. We also identify a key gene (ST3GAL4) that drives this process in AML by synthesizing these specific carbohydrate structures. When we inhibited the activity of ST3GAL4, it makes AML cells more sensitive to killing by immune cells. This slowed down the progression of AML in mouse models, implying that ST3GAL4 may be an exciting new target for cancer immunotherapy.

Bioresponsive Immunomodulator Nanocomplex for Selective Immunoengineering in Metastatic Lymph Node

- Poster #398

Presenter: Yueyang Deng, McGill University

Other Authors: Mo Chen, Tianxu Fang, Tianwen Luo, Xiaona Cao, Guojun Chen

ABSTRACT_ID#2586

ABSTRACT

Lymph node (LN) metastasis (LNM) frequently occurs in various cancer types and is associated with high aggressiveness, poor prognosis, and low survival rates. However, effective clinical interventions remain limited primarily due to the distinctive immunosuppressive microenvironment found in metastatic LNs. Targeted delivery of immunomodulators and selective immunoengineering in metastatic LNs offers a promising avenue for repurposing these LNs as an effective anti-tumor system while mitigating the risk of unwanted immune activation elsewhere. Here, we develop a bioresponsive LN-targeted immunomodulator nanocomplex designed to selectively reprogram the immune microenvironment in metastatic LNs for LNM inhibition. The immunomodulator nanocomplex can target LNs due to specific chemokine receptor 7 (CCR7) modification and selectively release anti-PD-1 antibodies (aPD-1) in response to glutathione (GSH) that is found elevated in the extracellular matrix of metastatic LNs. In two mouse models, our data suggested that the immunomodulator nanocomplex can selectively activate T-cell mediated antitumor immune responses in metastatic LNs and thus effectively inhibit tumor growth and prolong the survival of animals. This approach holds significant promises for reducing the necessity for complete LN dissection, thereby presenting a valuable therapeutic option for a broad spectrum of cancer patients.

LAY SUMMARY

1. We created an intelligent protein delivery system that specifically targets lymph nodes. This system carries immune-boosting protein and releases it in response to certain chemical signals found in cancerous lymph nodes.
2. The treatment reactivates the body's immune system within these metastatic lymph nodes, helping T cells recognize and fight the cancer more effectively—without triggering widespread immune responses that could harm healthy tissues.
3. In mouse models, this approach slowed tumor growth and extended survival, showing promise as a less invasive alternative to completely removing lymph nodes during cancer treatment in clinic.

Armouring GPNMB-Targeting Chimeric Antigen Receptor (CAR) T Cell Therapy Against the PD1/PDL1 Immune Checkpoint Axis - Poster #402

Presenter: Taye Louie, University of Calgary

Other Authors: Hongrui Liu, Jethro Langley, Laura Mah, Shahrabano Rukunuddin, Franz Zemp, Douglas Mahoney

ABSTRACT_ID#3233

ABSTRACT

GCAR1 is a novel GPNMB-targeting CAR T therapy recently evaluated in a first-in-human trial for a patient with metastatic alveolar soft part sarcoma (ASPS). GCAR1 was well-tolerated, significantly expanded in the blood, and eliminated numerous metastatic lesions. However, larger lesions failed to respond. Given extensive T cell infiltration and target antigen retention through analysis of a post-treatment biopsy, we hypothesized that tumour-driven immunosuppression limited therapeutic efficacy. Further, using a patient-derived xenograft (PDX) mouse model of ASPS, we found an otherwise sub-curative dose of GCAR1 achieved complete responses when administered with adjuvant atezolizumab, a PD-L1 checkpoint inhibitor. Atezolizumab, however, is costly, requires frequent hospital visits, and can potentiate toxic non-specific T cell responses. Given the limitations of atezolizumab, we hypothesized that armouring of GCAR1 to directly resist PD-1/PD-L1 immunosuppression would address these challenges, providing additional opportunities to precisely tailor T cell responses. Specifically, we developed a PD-1 switch receptor with a PD-1 extracellular and CD28 intracellular domain for co-expression in GCAR1. We believe the switch receptor will sequester functional PD-L1 signalling while simultaneously propagating CD28 costimulatory signals, producing a more robust CAR T cell response. To optimize the switch receptor, we developed a platform for functional assessment of iterative designs. This platform includes PD-L1 overexpressing and antigen-negative cancer cell lines, an optimized fluorescence-based triple reporter Jurkat system for measuring T cell activation, and a novel PDX model for studying the switch receptor in other GPNMB-expressing cancers. Using this platform, we aim to develop an optimized version of GCAR1 for clinical implementation.

LAY SUMMARY

Chimeric antigen receptor (CAR) T cell therapy is a cancer immunotherapy where specialized killing cells of a patient's immune system, known as the T cells, are genetically edited to more effectively recognize and fight their cancer. GCAR1 is a novel CAR T therapy developed in our lab that was tested in a first-in-human trial for a patient with aggressive disease, eliminating many metastatic growths. Evidence suggests that the effectiveness of GCAR1 was limited by immunosuppressive signalling mechanisms exploited by the cancer, which shut down the T cell response. In a mouse model, we showed that blockade of a particular immunosuppressive pathway, PD-1/PD-L1, enhances the ability of CAR T cells to eliminate tumours. However, the current approach for disrupting this pathway is financially costly, burdensome on the patient, and has toxicity concerns. We developed a strategy for directly protecting GCAR1 against immunosuppression by the tumour. Specifically, with this strategy, inhibitory signals produced by the tumour are converted into signals that will instead activate our CAR T cells. We are currently testing and refining this strategy for maximal therapeutic efficacy using a platform of experimental techniques developed in the lab.

Advancing CAR-T clinical development with high-parameter CyTOF technology - Poster #403

Presenter: Toby Astill, Standard BioTools

Other Authors: Michael Cohen

ABSTRACT_ID#2899

ABSTRACT

Adoptive immunotherapy using chimeric antigen receptor (CAR)-T cells has revolutionized cancer treatment, showing significant success in B cell malignancies. However, CAR-T therapy faces major challenges in treating solid tumors due to the complex tumor microenvironment, limited cell trafficking, short-lived anti-tumor activity, and potential toxicities. Advancing CAR-T efficacy requires a deeper understanding of its biology to enhance durability and reduce side effects. High-parameter flow cytometry has been instrumental in evaluating CAR-T cells throughout development, from manufacturing to patient monitoring. However, fluorescence-based cytometry is constrained by signal overlap and autofluorescence, limiting the detection of rare populations and reliable functional readouts. CyTOF (mass cytometry) technology overcomes these limitations with minimal signal overlap and no autofluorescence. It also enables flexible workflows using frozen antibody panels and stained samples. In this study, a 40+ marker CyTOF panel was used to assess CAR-T cell phenotype and function in vitro. CD19 CAR-T cells were expanded and co-cultured with Nalm6 tumor cells at various effector-to-target (E:T) ratios for 2–5 days. Cells were stained, barcoded, and acquired simultaneously using CyTOF XT. The panel enabled simultaneous detection of surface, cytoplasmic, and nuclear markers. Key functional states—including activation, proliferation, cytokine production, cytotoxicity, and exhaustion—were evaluated. Results showed that CAR-T cells became activated and functional during early co-culture, but displayed reduced cytotoxicity and signs of exhaustion with prolonged stimulation. Overall, the high-parameter CyTOF panel allows for deep, multiplexed analysis of CAR-T cells, supporting continued clinical development of more effective and safer CAR-T therapies.

LAY SUMMARY

CAR-T cell therapy is highly effective in blood cancers but faces major challenges in solid tumors due to limited persistence, tumor environment complexity, and toxicity risks. CyTOF technology overcomes limitations of traditional flow cytometry by enabling multiplexed analysis with minimal signal interference and no autofluorescence, allowing for better detection of rare CAR-T cell populations. A 40+ marker CyTOF panel enables deep functional profiling of CAR-T cells—revealing activation, cytokine production, and exhaustion dynamics during co-culture with tumor cells—critical for improving clinical performance and safety.

Sunday, November 2, 2025

Gut Microbial Drivers of Response to Bladder-Sparing Therapy in Human Muscle-Invasive Bladder Cancer Enhance Immunotherapy Efficacy and Limit Immunosuppression in vivo - Poster #405

Presenter: Éva Michaud, Research Institute of the McGill University Health Center

Other Authors: Éva Michaud, Sabina Fehric, Cynthia Faubert, Irah King, José Joao Mansure, Wassim Kassouf

ABSTRACT_ID#3029

ABSTRACT

BACKGROUND: Radiation therapy (RT) is a bladder-sparing treatment for muscle-invasive bladder cancer (MIBC), but 30% of patients do not respond, and 50% develop metastasis. Combining RT with PD-1/PD-L1 blockade enhances antitumor responses in preclinical models, but factors influencing its success remain unclear. Patients with a favorable microbiome (*A. muciniphila*, *Bifidobacterium*, *Faecalibacterium*) have improved immunotherapy responses. We thus hypothesized that modulating gut microbiome during MIBC improves responses to RT-based combinations. **METHODS:** Fecal samples from RT responders (R, n=26) and non-responders (NR, n=11) underwent shotgun metagenomics. Fecal microbiota transplantation (FMT) from R (n=3) and NR (n=3) were performed into germ-free mice, then randomized into anti-PD-L1, RT, RT+anti-PD-L1 or untreated groups. Tumors were analyzed by single-cell RNA- and TCR-seq. **RESULTS:** R patients displayed particular enrichment in known promoters of anti-PD-L1 response, such as *Phocaeicola dorei* relative to NR. R-FMT into GF mice resulted in significant increases in predicted survival, tumor growth delay, and counts of effector CD4⁺ T cell infiltration in the RT+PD-L1 arm. Conversely, NR-FMT correlated with increased neutrophil and Treg infiltration, with no survival benefits in the combination arm. We found that R-FMT metabolically competent CD4⁺ T cells (GLUT⁺) switched to effector transcriptomic program in the combination arm but not NR-FMT counterparts. RT-treated tumor in the R-FMT context significantly expanded T effector memory clones and NKT cells, compared to NR-FMT. **SIGNIFICANCE:** This is the first study using FMT to modulate response to combination therapies in MIBC, paving the way for more personalized treatment approaches for MIBC patients who cannot undergo surgery.

Microbiology and Microbiome

Monday, November 3, 2025

Microbial Metabolite Modulation of CAR-T Therapy - Poster #406

Presenter: Amy Shupe, University of Calgary

Other Authors: Larisa Kovtonyuk, Sacha Benaoudia, Kyle Potts, Doug Mahoney, Kathy D. McCoy

ABSTRACT_ID#2806

ABSTRACT

While chimeric antigen receptor (CAR) T therapy has revolutionized the treatment of hematological malignancies, many patients still do not respond. Recent research has pointed to the microbiome, the collection of microbes that live on and in our bodies, as a key player in the response to immunotherapies. However, the underlying mechanisms by which bacteria influence the responses to cancer and cancer immunotherapies are still largely unknown. Our lab has previously demonstrated that the metabolite inosine, produced by *Bifidobacterium pseudolongum* and other bacteria, improved the efficacy of another form of immunotherapy called immune checkpoint blockade therapy (ICBT), in a context dependent manner. We hypothesized that inosine would also increase the efficacy of CAR-T therapy. Pre-treatment of human CAR-T cells with inosine was found to improve the killing of a Nalm6 human lymphoma cancer cell line in vitro. CAR-T cells treated with inosine showed higher levels of the effector molecules IFN γ , TNF, Granzyme B and Ki67, than untreated CAR-T cells measured via flow cytometry. In addition, CAR-T cells incubated with inosine in vitro also showed superior ability in vivo killing capacity when transferred to germ free mice bearing Nalm6 as compared to untreated CAR-T cells. Overall, these findings could pave the way for the use of microbial products that can increase the efficacy of CAR-T therapy.

LAY SUMMARY

Good gut bacteria may help cancer therapies work better. We studied a molecule, produced by gut bacteria, called inosine, and found it can make a certain form of cancer immunotherapy (chimeric antigen receptor or CAR T cells) more effective. Inosine boosts the ability of CAR-T cells to kill cancer cells. We tested inosine treated CAR-T cells and found that they were better at killing cancer cells and showed more signs of effector function. These findings could pave the way for the use of bacterial products (like inosine) to increase the efficacy of cancer therapy.

Impact of the gut microbiota on the response to Bacillus Calmette-Guérin immunotherapy in bladder cancer: towards a predictive molecular model - Poster #407

Presenter: Marine Boireau, CRCHUdeQuebec- Université Laval

Other Authors: Jalal Laaraj, Gabriel Lachance, Roxane Tourigny, Sandra Isabel, Yves Fradet, Karine Robitaille, Vincent Fradet

ABSTRACT_ID#3046

ABSTRACT

Objectives: Treatment of high-risk non-muscle invasive bladder cancer (NMIBC) involves transurethral tumor resection followed by intravesical immunotherapy with Bacillus Calmette-Guerin (BCG) instillation. Unfortunately, 40% of patients relapse within five years. Several studies showed that gut microbiota influence immunotherapy response and identified specific bacteria capable of improving systemic immunotherapy effectiveness. However, it is unknown whether these bacteria also play a role in BCG response. **Methods and results:** We performed shotgun metagenomic analyses on fecal DNA from 58 NMIBC patients collected before their BCG treatment to catalog gut microbiota composition. We first tested the TOPOSCORE, a predictive model for systemic immunotherapy response validated in various cancers. Although this predictive score identified 100% of BCG non-responders (NR), it showed low specificity (57%) in predicting BCG response. Furthermore, our preliminary data suggest that 50% of the NR bacteria are associated with antibiotic resistance. In parallel, we found a unique microbiota signatures linked to BCG responsiveness using whole-genome sequencing. Several BCG response-associated bacterial species (RABS) were uniquely associated with improved recurrence-free survival of NMIBC patients. **Conclusion:** Our results suggest that gut microbiota associated with non-response to systemic immunotherapy is similar to that of BCG non-responders in NMIBC, while the bacteria linked to BCG response differ. Our current research aims to develop a unique predictive molecular test based on the microbiota signature of NMIBC patients, which will predict clinical response to BCG and optimize the patient care. Moreover, RABS identification supports the development of a BCG adjuvant treatment via a probiotic supplementation to improve BCG response.

LAY SUMMARY

The gut microbiota influences the response to systemic immunotherapy in various cancers, and some bacteria improve treatment efficacy. A generic predictive test based on gut microbiota composition exist for systemic immunotherapy, but is not accurate for localized immunotherapy like BCG in bladder cancer. We identified specific bacteria, called response-associated bacterial species (RABS), linked to a better recurrence-free survival in patients, following BCG treatment. These RABS appear to harbor no antibiotic resistance genes, making them promising candidates as probiotic adjuvants to BCG therapy in bladder cancer. Identifying a bacterial signature specifically associated to BCG response or non-response could lead to a molecular test predicting individual patient response to BCG therapy in clinic.

Gut microbiome influences the antitumor response to BCG immunotherapy in bladder cancer - Poster #408

Presenter: Jalal Laaraj, Oncology Research program, CHU de Québec-Université Laval Research center and Cancer Research Center

Other Authors: Gabriel Lachance, Prisca Nadège Koné, Roxane Tourigny, Typhaine Gris, Paul Toren, Alain Bergeron, Sandra Isabel, Yves Fradet, Karine Robitaille, Vincent Fradet

ABSTRACT_ID#2685

ABSTRACT

Introduction: Bacillus Calmette-Guérin (BCG) intravesical immunotherapy is the standard treatment for non-muscle-invasive bladder cancer (NMIBC). Despite its efficacy, still 40% of patients experience recurrence. Recent evidence suggests that the gut microbiome influences clinical efficacy of systemic immunotherapies. We evaluated the impact of gut microbiota on the antitumor activity of BCG in NMIBC patients and mouse models. Methods and Results: We prospectively collected fecal samples from 58 NMIBC patients before initiating BCG therapy and performed both, shotgun metagenomic and 16S rRNA sequencing to profile the gut microbiota. Differential analysis identified three bacterial species enriched in responders (R, n=42) compared to non-responders (NR, n=16). To evaluate the causal relationship between the gut microbiome and response to BCG, we conducted fecal microbiota transplantation (FMT) from R and NR patients into antibiotic pre-treated MBT-2 tumor-bearing mice, followed by weekly intratumoral BCG. FMT from NR significantly impaired BCG efficacy compared to FMT from R. Flow cytometry and bulk RNA sequencing analysis showed that R FMT was associated with potent antitumor immunity and reduced immunosuppressive populations in tumors compared to NR FMT. Oral supplementation of NR avatar mice with the response-associated bacterial species completely restored BCG efficacy, an effect that was mediated by T cell antitumor immunity and reduced myeloid immunosuppressive populations in both tumors and spleens, supporting an immune-potentiating effect. Conclusions: Our findings demonstrate that the gut microbiome impacts BCG immunotherapy response in NMIBC patients. This study paves the way for developing microbiome-targeted strategies to enhance BCG treatment efficacy in bladder cancer.

LAY SUMMARY

The gut microbiome influences bladder cancer treatment: We discovered that the bacteria living in our intestines can influence how well patients respond to BCG immunotherapy, the standard treatment for early-stage bladder cancer. Patients who responded well to BCG had certain beneficial bacteria in their gut. When we transferred fecal samples from these responder patients into mice, the treatment was more effective compared to mice that received samples from non-responders, who showed poorer outcomes. Targeting the gut microbiome can improve BCG treatment efficacy: When we supplemented non-responder avatar mice with the beneficial bacteria found in responders, their response to BCG improved significantly. This suggests that modulating the gut microbiome using live biotherapeutic probiotics could enhance the efficacy of bladder cancer treatment.

Exploring gut microbiome-driven estrogen metabolism and its role in breast cancer - Poster #409

Presenter: Ashley Larnder, University of British Columbia

Other Authors: Thad J. Edens, Julian J. Lum, David Goodlett, Jun Han, Kathryn Isaac, Rebecca Warburton, Michelle E. Goecke, Allen H. Hayashi, Alison Ross, Jane Shearer, Kristin L. Campbell, Amee R. Manges, Rachel A. Murphy

ABSTRACT_ID#2526

ABSTRACT

Background: Growing evidence links the gut microbiome to carcinogenesis, with the estrobolome—a subset of gut microbiota with estrogen-related functions—implicated in breast cancer (BC) risk. However, estrobolome targets and mechanisms remain poorly defined. **Methods:** In this case-control study, postmenopausal women with treatment-naïve HR-positive BC (n=70) and healthy controls (n=70) provided survey data, blood and stool samples at baseline and 6-months. The gut microbiome was profiled using whole metagenomic shotgun sequencing, and 245 metabolites were quantified in plasma and stool using targeted metabolomics. Diversity measures and differential abundance analyses (MaAsLin2) were used to characterize the microbiome. Confounder-adjusted logistic regression examined estrobolome targets in relation to BC risk (FDR $q < 0.25$). ElasticNet identified additional, untargeted microbiome features, which were tested for associations with metabolites using linear regression. **Results:** Cases showed lower α -diversity, with 57 species differentially abundant. Three estrobolome taxa were modestly associated with BC ($p < 0.05$, $q > 0.25$): *B. bifidum* (OR=0.85), *C. butyricum* (OR=1.15), *E. ramosum* (OR=1.14). ElasticNet identified eight taxa associated with BC (e.g. *A. rubiinfantis*). Thirteen metabolites were associated with BC, including five phytoestrogens—matairesinol (OR=0.40), genistein (OR=1.14), daidzein (OR=1.15), pinoresinol (OR=0.58), SECO (OR=0.65)—and three hormone-related metabolites DHEA-S (OR=1.36), estriol (OR=1.10), and testosterone (OR=1.73). Microbiome–metabolite associations among significant features were observed, including phytoestrogen links to *A. rubiinfantis* and *C. butyricum*. **Conclusions:** Few estrobolome targets were linked to BC, although associations with hormone- and phytoestrogen-related features indicate broader microbial involvement in hormone homeostasis. Additional microbiome/metabolome differences support the need to investigate non-estrogenic pathways, including immune, inflammatory, and dietary influences, in the microbiome–breast cancer relationship.

LAY SUMMARY

No clear estrogen-specific microbiome signals were found in our sample: Microbes and enzymes previously proposed as part of the estrobolome were not clearly linked to breast cancer risk. While these targets may play a role, they do not appear to be strong or direct contributors to disease in this context. The estrobolome itself may also remain poorly or incompletely defined. Microbes may affect hormones more broadly: Even without strong estrobolome signals, several gut bacteria were linked to other hormone and phytoestrogen levels, pointing to a broader microbial role in hormone balance. Non-estrogen pathways matter too: Differences in gut microbes and metabolites between women with and without breast cancer suggest that immune, inflammatory, and dietary factors—beyond estrogen—may also be involved in the microbiome–breast cancer connection.

Cervicovaginal microbiome composition in women negative for pre-cancer (CIN2+) over a decade - Poster #410

Presenter: Ayesha Lalani, Women's Health Research Institute

Other Authors: Smritee Dabee, Ana Citlali Márquez, Gabrielle Angelo P. Cortez, Mruthula Narayan, Agnes Truc Nguyen, Janet E. Hill, Laurie Smith, Gina Ogilvie, Inna Sekirov

ABSTRACT_ID#2519

ABSTRACT

Background: Cervical cancer is the fourth most common cancer in women globally with HPV infection being a necessary cause for cancer progression. Infection risk and pre-cancer progression, cervical intraepithelial neoplasia [CIN] grade 2+, are associated with microbial dysbiosis. While pathogenic and inflammatory microbial links have been described, studies exploring protective microbiome patterns are limited. We aim to characterize the microbiome of women who had <CIN2 outcomes over ten years post negative HPV testing. Methods: We identified women with <CIN2 outcomes over 10 years of screening from the FOCAL randomized clinical trial dataset (N=15; data generation ongoing). Microbiome composition was determined using chaperonin protein (cpn60) barcode sequencing. Demographic characteristics at baseline were captured using a survey. Results: Participants had a mean age of 49.6 years (SD = 4.9). Majority self-identified as Caucasian and 40% (n=6) reported use of barrier birth control at the baseline visit. Lactobacillus species formed 52% of the microbiome [SE: 0.11] with *L. crispatus* being the most present (41% [SE: 0.08]). Previously described BV-associated genera *Prevotella*, *Gardnerella* and *Atopobium* were found at <5% relative abundance except *Gardnerella swidsinskii* and *Prevotella buccalis* (10.8% and 6.2% respectively, [SE: 0.06]). With sequencing ongoing (for a total sample of 100), microbiome analyses and logistic regression will be used to determine the effect sizes of the microbial species' impact on CIN2+ outcomes stratified by demographic variables. Conclusion: These findings underscore the microbiome's role in HPV susceptibility and cervical cancer outcomes, supporting its potential use in HPV-based primary screening to predict disease progression.

LAY SUMMARY

Preliminary analysis reveals Lactobacillus species comprises over half the cervicovaginal microbiome in women who remained negative for pre-cervical cancer (CIN2+) over a decade. Bacterial vaginosis (BV)-associated bacteria were present at low relative abundances, suggesting a potentially protective microbial profile against HPV infection and cervical cancer progression. Associations between microbiome composition and key demographic and clinical factors—including age, ethnicity, contraceptive use, smoking status, and pregnancy history—will be examined to explore how host factors may influence microbiome composition and related cervical cancer outcomes. Understanding the role of the genital microbiome in cervical cancer progression may help identify microbial markers that enhance HPV-based screening and risk prediction.

Microbiology and Microbiome

Sunday, November 2, 2025

Effects of Colorectal Cancer Treatment on Gut Microbiota: A Pilot Feasibility Study - Poster #411

Presenter: Gavin Head, University of Calgary

Other Authors: Sasha Sharow, Lais Cezario, Vagner Tavares, Madison Leia, Christie Farrer Rogers, Kathy McCoy, Anthony MacLean, Lin Yang, May Lynn Quan, Donald Buie, Renelle Daigle, Colleen Cuthbert

ABSTRACT_ID#3083

ABSTRACT

Background: Colorectal cancer (CRC) treatments can lead to gut dysbiosis, contributing to adverse effects (AEs). The gut microbiome may represent an integral role in AE development. Understanding dysbiosis in CRC patients may impact AE prevention and management strategies and enhance patient quality of life. Aims: Determine feasibility of collecting microbiota samples from CRC patients from diagnosis to one-year post-treatment completion, examine longitudinal changes to microbiota, and identify preliminary associations between microbiota changes with treatment completion, adverse experiences, and patient reported outcomes (PROs). Methods: CRC patients (stage I-III) were referred by surgical oncologists prior to treatment. Participants reported PRO data and collected at-home fecal samples at 4 timepoints. Fecal samples were analyzed using shotgun metagenomic sequencing to assess gut microbiome composition and diversity. Results: Eleven of 14 (78%) referred patients were consented. One (9%) participant withdrew before sample collection; 4 (36%) collected two or fewer samples, and 5 (45%) provided all samples. Sequencing revealed a reduction in microbial diversity following treatment, with diversity levels not returning to baseline after one year, suggesting a lasting impact of therapy on the gut microbiome. Participants demonstrated the highest positive gains to quality of life, anxiety, depression, fatigue, and cognitive function six-months post-treatment. Conclusion: Reliance on surgeon-initiated referrals was a key feasibility challenge due to competing clinical demands, suggesting the need for multi-pronged recruitment approaches in future studies. Although patients showed clinically meaningful improvements in several PROs, studies should investigate how and if these changes are correlated to microbiome diversity in a larger sample.

LAY SUMMARY

Understanding how colorectal cancer treatment affects the gut microbiome, and whether microbiome changes are associated with patient reported outcomes, could help us find ways to reduce treatment side effects and improve patient quality of life. Participants experienced a reduction in microbiome diversity after treatment. These levels did not return to baseline after one year following completion of treatment, which suggest colorectal cancer treatment has a long term impact on the gut microbiome. We observed meaningful improvements to patient reported outcomes such as quality of life approximately 6 months following treatment completion.

SMARCA4 loss drives dedifferentiation and the senescent associated secretory phenotype in dedifferentiated endometrial carcinoma - Poster #413

Presenter: Hannah Plummer-Doherty, Queen's University

Other Authors: Mackenzie Coatham, Bianca Dauber, Julia Vassalakis, Einav Renert, Farzaneh Afzali, Tyler Cooper, Guihua Zhang, Jiahui Liu, Lynne-Marie Postovit, Cheng-Han Lee

ABSTRACT_ID#2636

ABSTRACT

Endometrial cancer is the most common gynecological malignancy worldwide, with incidence on the rise. While the prognosis for localized endometrial cancer is excellent, certain patients develop dedifferentiated endometrial cancer (DDEC), wherein the five-year survival rate is less than 25%. We have discovered that the loss of SMARCA4, the catalytic subunit in the SWI/SNF complex, causes well differentiated endometrial cancer cells to dedifferentiate. SWI/SNF is a chromatin modifier, which functions by sliding and evicting nucleosomes, ultimately altering downstream gene transcription. Evidence from our lab suggests that SMARCA4 loss enables dedifferentiation only upon passaging in a mouse as initially cells manifest a senescent-like phenotype. This phenotype is indicated by the expression of b-galactosidase and manifestation of the Senescence Associated Secretory Phenotype (SASP). This suggests that features of the growing tumour, such as hypoxia, may enable dedifferentiation and that the SASP may characterize a transitional cell state. The mTOR pathway, which regulates metabolism, survival, and mRNA translation, has been shown to promote the SASP and is inactivated in response to cellular stresses (hypoxia). We hypothesize mRNA translation, associated with aberrant mTOR activation, mediates the senescence-like phenotype and dedifferentiation in response to stress after SMARCA4 KO. Our results have shown that KO cells are resistant to hypoxia treatment, maintaining high levels of mTOR activity and have reduced phosphorylation of eIF4E. Polysome analysis has shown KO cells maintain higher levels of mRNA translation under hypoxic conditions. Future directions will include RNA sequencing to determine changes in mRNA targeting with SMARCA4 KO under hypoxia.

LAY SUMMARY

- The loss of SMARCA4 causes endometrial cancer cells to initially become senescent-like, and only after passaging in a mouse do the cells emerge from this phenotype and dedifferentiate.
- The loss of SMARCA4 causes cells to become resistant to hypoxia, maintaining higher growth rates and mTOR signalling in 24 hours of hypoxia.

Targeting the translational machinery to overcome kinase inhibitor resistance in melanoma - Poster #414

Presenter: Anavasadat Sadr Hashemi Nejad, The Université de Montréal

Other Authors: Alejandro Schcolnik-Cabrera, Meriem Takdenti, Zaynab Nouhi, Martin Roffe, Tommy Alain, Laura Hulea

ABSTRACT_ID#2383

ABSTRACT

Cancer cells rewire their mRNA translation programs and their metabolism to support growth and resistance to anti-cancer therapies like oncogenic kinase inhibitors (KIs). Our previous studies delineated translational programs that induce metabolic plasticity, which underpinned adaptation to KIs. Importantly, cancer-specific perturbations in the translational apparatus provide a therapeutic window to selectively target the translational programs of malignant cells while sparing normal tissues. The eukaryotic translation initiator factor 4F (eIF4F) complex is a key mediator facilitating translation, leading to proliferation and survival. Therefore, we hypothesize that targeting the translational machinery via inhibition of eIF4A, a subunit of eIF4F, using an eIF4A inhibitor (eIF4Ai), attenuates resistance to oncogenic KIs by preventing the metabolic reprogramming that supports cancer cell adaptation to therapies. This has been explored in our lab with in vitro and in vivo studies in BRAF inhibitor (BRAFi)-resistant melanoma models. About 50% of malignant melanoma (MM) harbor BRAF mutations, with many developing resistance to BRAFis. We found that eIF4Ais block cell proliferation, induce apoptosis, reduce mRNA translation levels, and strongly affect metabolic processes. This is paralleled by downregulation of target proteins involved in proliferation, survival and metabolic enzymes. To characterize mRNA translational modulation upon eIF4Ai, we performed polysome profiling and subsequent RNA sequencing. Furthermore, to confirm our in vitro findings, the efficacy of eIF4Ai alone and/or plus BRAFi on tumor growth is determined in vivo using cell line-derived xenografts. We anticipate that combined treatment with eIF4Ai and KIs will establish the efficacy of translational inhibitors in overcoming KI resistance in MM cells.

LAY SUMMARY

Cancer cells change how they make proteins and process nutrients to survive treatments like kinase inhibitors; by targeting key mediators of protein-making pathways, we might be able to stop this resistance. Blocking a key subunit of a specific mediator involved in protein synthesis (eIF4A) slows down melanoma cell growth, triggers cell death, and disrupts how these cells manage energy especially in cases resistant to current therapies. Combining eIF4A inhibitors with current targeted therapies (BRAF inhibitors) showed promising results in our laboratory and animal studies, suggesting this approach could help overcome drug resistance in melanoma.

Importance and Function of the Tumor Suppressor Ikaros in Genomic Stability - Poster #415

Presenter: Ailyn de la Caridad Ramon Sanchez, Université de Montréal

Other Authors: Norreen Quansah, Stefania Bottardi, El Bachir Affar, Hugo Wurtele, Eric Milot

ABSTRACT_ID#3117

ABSTRACT

Genotoxic chemotherapy is a preeminent therapeutic option for Acute Lymphoblastic Leukemia (ALL). While response to therapy has improved, a significant fraction of ALL patients still succumbs to this disease. It is critical to further investigate mechanisms that influence treatment response in ALL. Mutation or deletions of the tumor suppressor Ikaros characterizes a significant fraction of high-risk ALL. The influence of Ikaros on nuclear functions other than transcriptional regulation remains poorly defined. We detected that the DNA double-strand break (DSB) mark γ H2AX is increased in hematopoietic cells when Ikaros is mutated, thus suggesting potential implication in genomic stability. Transcriptomic analysis did not reveal transcriptional changes explaining the genomic instability in IKko cells. Based on the above, we hypothesise that the disruption of Ikaros influences genomic stability and affects the response of ALL cells to genotoxic chemotherapy. We are using Jurkat T-ALL cells in which the endogenous Ikaros has been inactivated (IKko) as model systems. To assess the effect of IKko on genomic stability and determine whether Ikaros isoforms differentially affect genomic stability, cells are treated with zeocin followed by detection of DNA damage markers by Western blot and comet assay. Our results indicate that the absence of Ikaros increases spontaneous DNA DSB and impairs activation of DNA damage checkpoints. Furthermore, we found that the functional Ikaros isoforms IK-1 and IK-2 can rescue these phenotypes. Our work is expected to elucidate the effect of Ikaros disruption on genomic stability during leukemogenesis and may provide valuable information towards the development of therapies.

LAY SUMMARY

Leukemia treatment still faces challenges: Although chemotherapy has improved outcomes for many patients with Acute Lymphoblastic Leukemia (ALL), some patients especially those with high-risk forms, such as when Ikaros is disrupted, still do not respond well. We need a better understanding of what affects treatment success. The Ikaros protein helps keep DNA stable: We found that when the protein Ikaros is missing or mutated, leukemia cells show more DNA damage, even before treatment. This suggests that Ikaros plays a role in protecting cells' genetic material. Restoring Ikaros function improves cell stability: When we reintroduced normal versions of Ikaros into leukemia cells lacking it, the cells had less DNA damage and better response to stress. Transcriptomic analysis did not reveal transcriptional changes explaining the genomic instability in IKko cells. This points to Ikaros as a potential target for improving leukemia therapies in the future.

Ash2L: an epigenetic regulator of tumor invasion - Poster #416

Presenter: Arielle Beaudoin, Université Laval

Other Authors: Arianne Gervais, Nafiseh Sadat Mousavi Maleki, Emeline Lelong, Samer Hussein, François Bordeleau, Darren E. Richard, Josée N. Lavoie

ABSTRACT_ID#3062

ABSTRACT

The metastatic migration of tumour cells requires large cellular deformations, which are limited by the size and rigidity of the nucleus. Alterations in chromatin organization allow tumour cells to adjust nuclear mechanical resilience and increase their invasive potential. Metastatic migration also involves the formation of invadopodia, invasive structures that secrete matrix proteases. The formation of invadopodia is coordinated with nuclear changes that promote metastatic migration, through mechanisms that remain poorly understood. We discovered that the phosphorylation of the epigenetic factor Ash2L is involved in the regulation of nuclear mechanical resilience by remodeling chromatin. Moreover, Ash2L is phosphorylated by the oncogene Src, a kinase that regulates invadopodia formation. Our hypothesis is that Ash2L phosphorylation coordinates changes in nuclear organization and invadopodia formation to regulate tumour invasion. The objectives of this study are to analyze the impact of Ash2L and its phosphorylation on invadopodia formation and to investigate the regulation mechanisms by Ash2L. Using genetically modified MDA-MB-231 metastatic breast cancer cell lines, we showed that either the depletion of Ash2L or the expression of a phosphomimetic Ash2LY489E stimulated invadopodia formation. In contrast, the expression of non-phosphorylated Ash2LY489F inhibited these structures. Furthermore, Ash2L regulation of cell mechanics was impaired by epigenetic drugs or the disruption of LINC complexes, molecular connectors for nucleocytoplasmic interactions. These results indicate that Ash2L phosphorylation coordinates nuclear mechanics and invadopodia formation by a mechanism that requires nuclear and cytoskeleton connections. Future transcriptomic analyses will help define novel intermediates involved in Ash2L's role in promoting metastatic migration.

LAY SUMMARY

Deaths associated to solid cancers are primarily due to the spread of malignant cells to distant organs, where they form metastases. In order to succeed, metastatic cells acquire new properties that promote their migration through the confined spaces of surrounding tissues. This metastatic migration requires extreme cellular deformations, which are limited by the stiffness of the nucleus—the organelle that safeguards the genetic material. To navigate these narrow spaces, metastatic cancer cells must adopt various migratory strategies, including altering nuclear stiffness. They also employ specialized structures known as invadopodia, which enable them to widen tissue gaps and create migration tracks. However, the molecular mechanisms regulating these processes remain poorly understood. In our lab, we are working to define a new mechanism involved in metastatic migration. This signaling mechanism involves the modification of a protein that regulates nuclear organization. We discovered that this pathway enables metastatic cancer cells to alter nuclear stiffness and increase their ability to form invadopodia. Ultimately, we hope this study will contribute to a better understanding of metastatic migration and lead to new therapeutic avenues for treating metastatic cancers that develop resistance to commonly used treatments.

Exploring autophagy as a therapeutic vulnerability in pancreatic neuroendocrine neoplasms - Poster #418

Presenter: Arlene Gidda, Simon Fraser University

Other Authors: Suganthi Chittaranjan, Syed Zaidi, Emma Titmuss, Jing Xu, Steve Kalloger, Hugo Horlings, Daniel Renouf, David Schaeffer, Jonathan Loree, Sharon Gorski

ABSTRACT_ID#2515

ABSTRACT

Pancreatic neuroendocrine neoplasms (PNEs) are an uncommon pancreatic cancer arising from the islets of Langerhans. Unresectable or metastatic PNEs rely on systemic treatments which eventually fail due to treatment resistance. Hence there is a need to explore new therapeutic vulnerabilities and treatment resistance mechanisms. To address this need, we are exploring autophagy, an intracellular recycling pathway, implicated in treatment resistance in other cancer types. My overall objective is to determine if the autophagy pathway is a targetable vulnerability for PNEs. First, to examine autophagy-related expression, IHC staining for MAP1LC3B was performed on a tissue microarray that we developed with 89 PNE samples. High levels of punctate MAP1LC3B were associated with worse overall survival ($p=0.017$), supporting a potential role for autophagy in this disease. Next, we evaluated the combination of temozolomide, a standard PNE treatment, and the autophagy inhibitor chloroquine in a PNE cell line using a cell growth assay. We found that the combination resulted in a greater reduction in growth compared to either temozolomide or chloroquine treatment alone ($p=0.0022$ and $p=0.0047$, respectively). Our results suggest that autophagy inhibition may be an exploitable therapeutic target for PNEs. We are continuing this work using other patient-derived PNE models and drug combinations. This study has the potential to uncover new autophagy-related combination treatment options for patients with PNEs.

LAY SUMMARY

High levels of an autophagy protein were associated with worse overall patient survival, supporting a potential role for autophagy, an intracellular recycling pathway in pancreatic neuroendocrine neoplasms (PNEs). We found the combination of temozolomide, a commonly used chemotherapy, and chloroquine, an autophagy inhibitor, resulted in a greater reduction in PNE cell growth compared to either treatment alone.

Molecular and Cellular Biology

Sunday, November 2, 2025

Assessing the Biological Impact of Wildfire Smoke Using *Drosophila melanogaster* as a Model Organism - Poster #419

Presenter: Nawanjani Pathinayake, Queen's University

Other Authors: Maria Aristizabal, Benedikt Gregor

ABSTRACT_ID#2563

ABSTRACT

Wildfires are an emerging health risk and are quickly becoming one of the largest contributors to outdoor air pollution in Canada and around the world. Wildfire smoke exposure is linked to several disease states including asthma, heart disease, and lung cancer, but the exact mechanism by which wildfire smoke contributes to these health conditions remains unclear. To shed light on this process, we propose to use the fruit fly model system, which has several advantages: 1) flies are multicellular organisms that share 60% genetic homology with humans; 2) flies contain respiratory systems with key similarities to the human lung; 3) flies can be exposed to smoke via inhalation the relevant route of exposure in humans. Pilot studies underscored the utility of fruit flies as a model system to understand the molecular mechanism by which wildfire smoke disrupts biology. Results suggested sex and dose-specific changes in the longevity. Although our results lay the framework for understanding the effect of wildfire smoke on living systems and support the use of fruit flies for the systematic analysis of the effects of this emerging health threat.

LAY SUMMARY

Fruit flies are a useful model for studying wildfire smoke: Because fruit flies share many genes and similar respiratory systems with humans, they help us understand how wildfire smoke might affect people. Wildfire smoke affects lifespan differently based on sex and exposure level: Early results show that male and female flies respond differently to smoke, and the amount of smoke exposure also changes how long they live. This research helps explain how wildfire smoke harms living organisms: Studying fruit flies gives us clues about the specific ways wildfire smoke can damage health, which could lead to better ways to protect people. Future Plans for the Project: Analyze gene expression changes using RNA sequencing (RNA-seq): We will examine which genes are turned on or off in fruit flies after wildfire smoke exposure to identify key biological pathways affected. Investigate DNA modifications with DNA sequencing: By studying DNA methylation and other epigenetic changes, we aim to understand how smoke exposure can cause lasting effects on gene regulation. Use mass spectrometry to study protein changes: We will measure how wildfire smoke changes the abundance and modification of proteins in fruit flies, connecting gene-level changes to actual biological outcomes.

From Spheroids to Solid Tumours: How C/EBP β Drives Cancer - Poster #420

Presenter: Aisha Saleh, University of Ottawa

ABSTRACT_ID#3296

ABSTRACT

CCAAT/enhancer-binding protein beta (C/EBP β) is a basic leucine-zipper transcription factor implicated in increased tumour aggressiveness, therapeutic resistance, and decreased survival across numerous cancers. As a known cellular stress sensor, C/EBP β integrates environmental cues to promote survival and adaptation. We found that C/EBP β expression is strongly upregulated under stress-associated conditions, including three-dimensional spheroid culture—a model that mimics hypoxia and cell–cell contact found in solid tumours. This shift to spheroid growth induces a dormant-like state characterized by the downregulation of cell cycle programs and an accumulation of cells in the G0/G1 phase. Transcriptomic analysis identified the upregulation of autophagy-related pathways as a key mechanism in these spheroids. Critically, genetic knockout of C/EBP β in both Lewis lung carcinoma (LLC) and MC38 colon carcinoma cells significantly impaired spheroid growth and viability. This highlights C/EBP β as a crucial mediator of cancer cell viability and behaviour in 3D systems that recapitulate key features of the tumour microenvironment. In vivo, C/EBP β overexpression promoted tumour growth, while its loss inhibited expansion. Flow cytometric profiling showed that C/EBP β remodels the immune landscape by reducing NK and T-cell infiltration. Transcriptomic and histological analysis further demonstrated that C/EBP β promotes angiogenic gene signatures, resulting in increased CD31⁺ vascularization. Altogether, these findings identify C/EBP β as a key regulator of tumour cell dormancy and microenvironmental remodeling, facilitating immune evasion and angiogenesis. These coordinated responses may underlie the association between elevated CEBPB and poor prognosis across cancers and highlight C/EBP β as a potential target in tumour persistence and resistance.

LAY SUMMARY

Key Insights:

- C/EBP β helps cancer cells survive in spheroids. We use a spheroid model where cancer cells are packed together tightly in a ball of cells (like in real tumours) and oxygen is low. We found that C/EBP β became more active in these models. When we removed C/EBP β from cells, these cells could not grow as well and died.
- C/EBP β reshapes the tumour environment. In animal studies, tumours with more C/EBP β grew larger, had more blood vessels, and showed fewer cancer-fighting immune cells like T cells and natural killer cells. This suggests that C/EBP β helps tumours avoid the immune system and build the support they need to grow. Why this matters: High levels of C/EBP β are linked to worse outcomes in many cancers. Targeting this protein could help stop tumours from surviving, growing, and resisting treatment.

Molecular and Cellular Biology

Sunday, November 2, 2025

Spatio-Temporal Organization of DNA Repair Proteins at DSBs by PAR and PARG - Poster #421

Presenter: Natnael Abate, University of Alberta

Other Authors: Jean-Philippe Gagne, Guy Poirier, Jean-Yves Masson, Michael Hendzel

ABSTRACT_ID#3352

ABSTRACT

DNA double-strand breaks (DSBs) are among the most severe forms of genomic damage and require precise coordination of repair pathways to maintain genome integrity. Poly(ADP-ribose) (PAR) polymerases (PARP1/2) enzymes catalyze the addition of PAR chains at DNA DSBs, facilitating the recruitment of DNA end resection factors such as BRCA1 and CtIP. This process is tightly regulated by the enzyme poly(ADP-ribose) glycohydrolase (PARG), which removes PAR chains to restore homeostasis. Our project aims to define how PAR and PARG regulate the spatial-temporal organization of DNA repair proteins at DSBs. Using the U2OS 265 DSB reporter system and advanced imaging approaches including single-molecule localization microscopy, FRAP, and correlative light and electron microscopy, we will (1) determine PAR dynamics using PAR-binding proteins that recognize different forms of PAR (2) map the spatiotemporal distribution of PARylation within the DSB compartments under conditions of PARP and PARG inhibition. We'll use fluorescently tagged PAR-binding domains (WWE, PBZ, PBM, Macro) and ADP-ribose-detecting antibodies to better identify and analyze PAR, which can exist as single units (mono), multiple units (poly), or in branched and linear chain structures. This study will clarify how PAR coordinate DNA repair protein assembly and function and provide mechanistic insight into the altered resection observed with PARP inhibitors and PARG inhibitors. Ultimately, this work aims to inform therapeutic strategies to overcome PARP inhibitor resistance in HR-deficient cancers.

LAY SUMMARY

PAR is a signal. Enzymes called PARP1/2 quickly add chains of PAR at the site of the DNA break. These PAR chains then act like a magnet, drawing in other important repair proteins, such as BRCA1 and CtIP, to start the fix. PARG is the cleanup crew. Once the repair proteins are in place and working, PARG steps in to remove the PAR chains, resetting the system. We will use specially designed PAR-binding tools (WWE, PBZ, PBM, Macro) and antibodies that light up different forms of PAR (short chains, long chains, branched, or straight) so we can observe the evolution of PAR during DNA repair.

Investigating Kaiso and TGF β signaling crosstalk in triple-negative breast cancer (TNBC) metastasis - Poster #423

Presenter: Hanad Adan, McMaster University

Other Authors: Lindyann Lessey, Stephanie Ali Fairbairn, Rob Cowan, Juliet Daniel

ABSTRACT_ID#3116

ABSTRACT

Triple-negative breast cancer (TNBC) is an aggressive and highly metastatic breast cancer subtype. Due to the high propensity of TNBC tumors to metastasize, TNBC disproportionately accounts for almost half of all breast cancer-related deaths, despite only representing a minority of breast cancer cases. Many studies attribute TNBC's ability to undergo metastasis to epithelial-to-mesenchymal transition (EMT), a cellular process characterized by the loss of cell-to-cell adhesion, the acquisition of mesenchymal properties, and is often co-opted in cancer to facilitate migration and invasion. The transcription factor Kaiso, which is often misexpressed in TNBC, has been shown to be critical for TNBC metastasis in mouse xenograft models, as a regulator of EMT. In elucidating Kaiso's mechanism of action in this cellular process, we identified that Kaiso regulates the expression of various proteins in the Transforming Growth Factor Beta (TGF β) signaling pathway, another potent inducer of EMT in breast cancer. Interestingly, we also observed that TGF β 1 treatment increases Kaiso expression in our TNBC cell lines and that the TGF β pathway potentially regulates Kaiso as a downstream target gene, as indicated by our chromatin immunoprecipitation (ChIP)-PCR analysis of the Kaiso promoter. To further examine Kaiso's role in TNBC aggressiveness and metastasis, we aim to fully characterize this Kaiso-TGF β feedback loop and examine its expression in TNBC patient samples to determine if there is a unique Kaiso/TGF- β signature that could be used as a diagnostic/prognostic biomarker for TNBC.

LAY SUMMARY

Triple-negative breast cancer (TNBC) is a breast cancer subtype that is highly aggressive and tends to metastasize (i.e., spread to other parts of the body), resulting in poor patient survival rates. TNBC's ability to undergo metastasis has been attributed to the epithelial-to-mesenchymal transition (EMT). This cellular process enables cancer cells to invade tissues and subsequently spread to other organs. Kaiso is often highly expressed in TNBC tissues. Previous research from our lab and others has demonstrated that Kaiso regulates EMT and is crucial for the metastatic potential of TNBC tumors. Our research indicates that Kaiso interacts with TGF β , another protein implicated in breast cancer, and that these two proteins work together to promote TNBC metastasis. This research highlights the potential of developing therapies that target Kaiso to inhibit TNBC metastasis and improve patient outcomes.

Molecular and Cellular Biology

Monday, November 3, 2025

Loss of Sox10 inhibits mammary tumourigenesis in HER2-positive breast cancer - Poster #424

Presenter: Brennan Garland, University of Ottawa

Other Authors: Samuel Delisle, John Abou-Hamad, Luc Sabourin

ABSTRACT_ID#2898

ABSTRACT

The transcription factor SOX10 plays a critical role in neural crest development, but its function in epithelial tumorigenesis remains unclear. Here, we identify SOX10 as a key regulator of stem-like identity and tumour-initiating potential in HER2-driven mammary cancer. Genetic ablation of Sox10 in MMTV-Neu (NIC) mice conferred a dose-dependent delay in tumour onset, with complete loss resulting in a striking resistance to tumour formation. In vitro, CRISPR-mediated Sox10 inactivation in Neu-transformed mammary epithelial cells led to reduced 3D invasion, diminished self-renewal in serial mammosphere assays, and impaired tumorigenic capacity in both orthotopic and limiting dilution transplantation models. Clonal Sox10-null lines exhibited profound loss of tumor-initiating cell (TIC) frequency and failed to colonize lung tissue following tail vein injection. Transcriptomic profiling revealed that Sox10 directly regulates gene networks associated with stemness, neural crest identity, and epithelial plasticity. Motif enrichment analysis confirmed overrepresentation of SoxE-binding sites in promoters of downregulated genes. Collectively, these findings demonstrate that Sox10 defines a transcriptional state for HER2-mediated transformation and is required to sustain the invasive and self-renewing traits that drive tumor progression and metastasis.

LAY SUMMARY

Sox10 is required for HER2-mediated cancer cell transformation Deletion of Sox10 confers a dose dependent survival advantage in a murine model of HER2-positive mammary tumourigenesis

Molecular and Cellular Biology

Sunday, November 2, 2025

SMYD5 is a novel suppressor of breast cancer metastasis - Poster #425

Presenter: Ashley Janna, University of Ottawa

Other Authors: Nidhi Chaudhary, Sara Asif, Christopher J. Bergin, Marilyn Whelan, Martin Pelchat, Yannick Benoit, Michele Ardolino, Jean-François Couture

ABSTRACT_ID#3124

ABSTRACT

Most breast cancer-related deaths are attributed to metastasis. This process requires primary tumor cells to acquire migratory and invasive properties, among many other traits, ultimately leading to the colonization of distant tissues. Currently, our understanding of the molecular mechanisms underlying this process remains limited. Here, we examine the role of the lysine methyltransferase SMYD5 in metastatic breast cancer and whether the methylation of its major biological substrate, ribosomal protein L40 (rpL40), contributes to translational regulation. Our findings demonstrate that the metastatic potential of breast tumors negatively correlates with SMYD5 protein levels. In a non-metastatic cellular model, depletion of SMYD5 results in the acquisition of migratory properties and promotes organoid formation. Notably, SMYD5-deficient organoids also shed cells that migrate away from their origin and establish new, independent organoids, mirroring the metastatic process. SMYD5 depletion also reduces rpL40 trimethylation at lysine 22 (rpL40K22me3), diminishes de novo protein synthesis, and decreases the number of actively translating polysomes. Sustained expression of SMYD5 in these same models partially rescues cells from the translation-suppressing effects of the mTOR inhibitor Torin1. Conversely, in a highly metastatic cellular model lacking endogenous SMYD5, the overexpression of functional SMYD5 – but not of a catalytically inactive mutant (SMYD5MUT) – inhibits migration and invasion. Significantly, in mouse models of primary mammary tumors, SMYD5, but not SMYD5MUT, reduces the incidence of metastases to the lung. Altogether, our work establishes a role for SMYD5 in impeding breast cancer metastasis. More specifically, SMYD5-mediated methylation of rpL40 may serve to promote the translation of metastasis-suppressing protein networks.

LAY SUMMARY

Most breast cancer deaths are caused by metastasis, a process by which the cancerous cells spread to other parts of the body. We have found that the protein SMYD5 blocks breast cancer cells from spreading. In cell and animal models, loss of the SMYD5 protein causes cells to acquire metastatic behaviours/abilities; conversely, gain of the SMYD5 protein causes cells to lose metastatic behaviours/abilities. SMYD5 is responsible for modifying another protein, rpL40, which is a component of the ribosome (the cells' machinery for synthesizing proteins). The purpose of this research project is to investigate whether this modification of rpL40 may control the production of proteins that act to block the spreading of cancerous cells.

TET2 Mutations as Potential Modulators of Radiation-Induced Fibrosis - Poster #426

Presenter: Caitlin Hui Xarn Tan, University of Toronto

Other Authors: Pierre-Antoine Bissey, Justin Williams, Wei Shi, Madison Hossack, Kenneth Yip, Fei-Fei Liu

ABSTRACT_ID#2621

ABSTRACT

Introduction: Radiation therapy is a cornerstone of modern oncology, but up to 95% of patients experience off-target tissue damage, with fibrosis emerging as a common long-term consequence. The resulting inflammation – marked by immune cell recruitment, fibroblast activation, and transforming growth factor beta (TGF- β) release – can lead to collagen deposition and tissue remodelling, a condition known as radiation-induced fibrosis. Despite medical advances, fibrosis remains incurable, underscoring the need to understand its molecular underpinnings. Recent studies suggest that clonal haematopoiesis of indeterminate potential (CHIP), particularly TET2 mutations, may affect treatment outcomes. Therefore, this study aims to explore the role of TET2 as a modulator of fibrotic responses. **Methods:** RAW macrophages and fibroblasts derived from Balb/c wildtype and TET2 knockout mice were used. In vitro, collagen-1 expression served as the primary marker of fibrosis and was evaluated via RT-qPCR, Western blotting, and ELISA. In vivo, picrosirius staining followed by polarised microscopy was utilised to assess collagen deposition, while tibiofemoral joint angle measurements were used to determine the degree of fibrosis. **Results:** Collagen-1 expression was negligible in macrophages compared to fibroblasts, regardless of genotype. Preliminary co-culture experiments revealed that TET2 knockout (TET2 $-/-$) macrophages led to a greater reduction in collagen-1 expression in the fibroblasts than wild-type (TET2 $+/+$) macrophages. Lower collagen deposition is observed in TET2 heterozygote mice (TET2 $+/-$). **Conclusion:** These findings suggest that TET2 mutations may exert a protective effect against fibrosis. Further investigations will focus on the mechanisms by which TET2 CHIP mutations elicit this effect.

LAY SUMMARY

Macrophages expressed negligible levels of collagen-1 in comparison to Balb/c fibroblasts. Collagen-1 expression in fibroblasts was higher in the presence of TET2 wildtype (TET2 $+/+$) macrophages compared to TET2 knockout (TET2 $-/-$) macrophages. In vivo, TET2 mutations were shown to decrease the severity of fibrosis.

Molecular and Cellular Biology

Sunday, November 2, 2025

Determining the Role of Obesity in Breast Cancer - Poster #427

Presenter: Alexandra Perlman, Concordia University

Other Authors: Christina Dembegiotis, Claire Plissonneau, Sylvia Santosa, Alisa Piekny

ABSTRACT_ID#3192

ABSTRACT

Obesity is a known risk factor for breast cancer. Hormone-sensitive subtypes are strongly influenced by obesity-related metabolic and inflammatory changes, including increased adipokines and estrogen. Triple-negative breast cancers (TNBCs) are also linked to obesity, but the mechanisms remain unclear. One potential factor is extracellular vesicles (EVs) released from adipose tissue, which may alter the tumor microenvironment. Abdominal subcutaneous adipose tissue may be especially influential due to distinct molecular traits. To investigate this, we used conditioned media from cultured subcutaneous adipocytes derived from individuals with or without obesity. These were applied to MCF7 (hormone-responsive), Hs578t and HCC1143 (TNBC), and MCF10A (non-cancerous) cells. Media from adipocytes from individuals with high BMI enhanced MCF7 proliferation but had no effect on MCF10A or the TNBC lines. Hs578t cells showed higher baseline migration, and conditioned media increased migration across all cell lines. We then isolated and analyzed EVs from adipocyte media using mass spectrometry. Several adipokines and proteins involved in EV structure and biogenesis were identified that may influence cancer cell proliferation and migration. These findings suggest adipocyte-derived EVs could play a key role in breast cancer progression, with effects varying by cancer subtype. Our study highlights the importance of considering obesity in developing therapeutic strategies, particularly for hormone-sensitive and triple-negative breast cancers.

LAY SUMMARY

Adipocytes may send signals that help some breast cancers to progress: Substances released by fat cells, especially from people with obesity, can make hormone-sensitive breast cancer cell progression. Obesity-related signals can affect how cancer cells migrate: These fat cell signals also seem to increase the movement (migration) of breast cancer cells, which may play a role in how cancer spreads. Cargo transporters from fat cells could carry cancer-promoting messages: Like most cells, fat cells release small particles (called extracellular vesicles) containing proteins that might influence how breast cancer develops and spreads.

IL17A mediates lymphatic dysregulation and extracellular matrix remodeling in cancer-related lymphedema

- Poster #428

Presenter: Chong Wa To, University Health Network

Other Authors: Jeffrey C.W. To, Rosa Li, Mandi Ma, Rada Tazhitdinova, Wei Shi, Justin Williams, Pierre-Antoine Bissey, Kenneth Yip, Fei-Fei Liu, Jennifer Y.Y. Kwan

ABSTRACT_ID#2777

ABSTRACT

Introduction: Lymphedema is a chronic disease caused by lymphatic insufficiency resulting in the excess accumulation of lymph fluid and proteins in the interstitial space; it commonly affects patients following oncologic surgery or radiotherapy. However, the underlying molecular mechanisms remain poorly defined. This study identifies interleukin-17A (IL-17A) as a potential cytokine linking lymphatic endothelial dysfunction to extracellular matrix (ECM) remodeling in lymphedema. **Methods:** A C57BL/6 murine tail model of lymphedema was profiled using bulk RNA-seq, single-cell RNA-seq (scRNA-seq), single-cell mass cytometry (CyTOF), and proteomics to determine changes in gene expression, cell types and states, and protein expression in lymphedema. Findings were validated using immunohistochemistry (IHC) on murine and human skin, in vitro models of human dermal lymphatic endothelial cells (LECs), and IL17a-knockout mice. **Results:** Temporal transcriptomic analysis revealed significant upregulation of IL-17 signaling during acute and chronic stages of lymphedema, with IL-17A as a key isoform ($p < 0.0001$). IHC demonstrated an increased trend in IL-17A protein expression in human lymphedema skin ($p = 0.055$). scRNA-seq and CyTOF analyses revealed the presence of ROR γ t⁺ Th17 cells in lymph from lymphedematous mice, which co-localized with IL-17A staining on IHC ($p < 0.0001$). Proteomic analysis identified ECM remodeling as the top dysregulated pathway. Key ECM genes—including LOX, FMOD, ANGPT2, and COL2A1—were upregulated in IL-17A-treated LECs ($p < 0.01$). IL17a-knockout mice exhibited significantly reduced tail swelling ($p < 0.05$) and ECM deposition ($p < 0.01$). **Conclusions:** These findings identify IL17A as a central regulator of lymphatic dysfunction and ECM remodeling in lymphedema. Targeting IL-17A may represent a novel therapeutic strategy for cancer-related lymphedema.

LAY SUMMARY

IL-17A is a potential driver of lymphedema, identified as increased in both human lymphedema skin and a mouse tail model of the disease. IL-17A activates key extracellular matrix genes, including LOX, FMOD, ANGPT2, and COL2A1, in human lymphatic endothelial cells, suggesting a role in tissue stiffening associated with lymphedema. IL-17A-deficient mice showed significantly reduced swelling and scarring, suggesting that targeting IL-17A may help treat cancer-related lymphedema.

Exploring Kaiso's role in Androgen Receptor expression in triple-negative breast cancer - Poster #429

Presenter: Stephanie Ali Fairbairn, McMaster University

Other Authors: Kyle Kim, Robert Cowan, Akudo Eze-Onurah, Lindyann Lessey, Ryan Rattan, Salija Golamari, Juliet Daniel

ABSTRACT_ID#3138

ABSTRACT

Triple-negative breast cancer (TNBC) disproportionately affects women of African ancestry (WAA), who experience higher mortality rates compared to white women, despite lower incidence rates. Emerging evidence suggests that an ancestral genetic susceptibility may contribute to this disparity. Our lab identified the transcription factor Kaiso as a potential driver of increased metastasis and mortality in WAA TNBC patients. Notably, 60–80% of TNBC cases also exhibit low androgen receptor (AR) expression, correlating with poor response to AR inhibitors and reduced overall survival. This finding highlights a distinct TNBC subtype—quadruple-negative breast cancer (QNBC). Since *in silico* analyses revealed that high Kaiso and low AR expression are associated with worse survival outcomes in breast cancer patients, we hypothesized that this expression pattern contributes to the elevated mortality observed in WAA with TNBC. Immunohistochemistry of TNBC tissues from WAA and white patients confirmed significantly lower AR expression in WAA and a higher prevalence of AR-negative (QNBC) cases. Chromatin immunoprecipitation indicated that Kaiso associates with the AR promoter, suggesting transcriptional regulation. CRISPR-Cas9-mediated Kaiso knockout in TNBC cells increased AR mRNA and protein levels while reducing expression of the AR target gene ACSL4. Preliminary data revealed that Kaiso knockout TNBC cells are more sensitive to the AR inhibitor enzalutamide and to the chemotherapeutics, cisplatin and doxorubicin. Our findings suggest that Kaiso negatively regulates AR expression and may serve as a therapeutic target. Targeting Kaiso in combination with AR inhibitors could offer a novel strategy to improve outcomes for TNBC patients, particularly among WAA.

LAY SUMMARY

Black women are 40 % more likely to die from breast cancer and this may be due to an ancestral genetic susceptibility and a higher prevalence of the aggressive, triple-negative breast cancer subtype (TNBC). There is a high expression of the protein, Kaiso in TNBC tissues and past research from our lab and others showed that this correlates with a higher likelihood of a poor outcome. Our research shows that Kaiso may reduce androgen receptor (AR) expression in TNBC and make it less responsive to drugs such as enzalutamide that target AR. Clinical trials showed that enzalutamide improves the survival rates in patients who express AR. We used a technique called CRISPR-Cas9 to decrease Kaiso expression in TNBC cells and this resulted in the cells being more responsive to the AR inhibitor enzalutamide and to the chemotherapeutics, cisplatin and doxorubicin. Thus, highlighting the potential to target Kaiso in combination with AR to improve outcomes for TNBC patients.

Synergistic effect of BET/BRD4 bromodomain and ErbB receptor kinase inhibition reduces the tumor burden of HER2+ breast cancer brain metastases - Poster #430

Presenter: Danyyl Ippolitov, University of Manitoba

Other Authors: Yi-Han Lin, Abhijeet Kapoor, Raul Calvo, Victor Spicer, Rene Zahedi, Juan Marugan, Mark Henderson, Thomas Klonisch, Sabine Hombach-Klonisch

ABSTRACT_ID#2966

ABSTRACT

Background: Brain metastases remain a serious complication of advanced HER2-positive breast cancer (BC). Although some receptor tyrosine kinase inhibitors (RTKis) cross the blood–brain barrier (BBB), their clinical benefit is limited and associated with significant systemic toxicity. This study aimed to identify synergistic partners to enhance the tolerability and efficacy of RTKis. **Methods:** High-density assays were utilized to identify drugs synergizing with Pozitotinib (covalent pan-ErbB RTKi) in two brain metastatic BC cell lines. BBB-penetrability of the top compounds was predicted with in silico tools and validated via pharmacokinetics. Additional proteomic, metabolic, and colony formation assays were employed to verify the high-throughput findings. Target proteins were analyzed by WB. For in vivo studies, Luminal B BC cells were grafted into a mouse brain, and treated with a mono- or combination therapy. Tumor burden was assessed using MRI. IHC was used to analyze tumor cell proliferation markers. **Results:** BET/BRD4 inhibitor AZD5153 emerged as a potent synergistic partner to Pozitotinib. BRD4 expression and associated histone acetylation were confirmed in the tested BC models. In vitro, the combination treatment reduced cell viability and enhanced apoptosis when compared to Pozitotinib or AZD5153 alone. These changes were associated with a complete suppression of c-Myc protein by AZD5153. In mice, the addition of AZD5153 allowed a reduction of the effective Pozitotinib dose. Dual therapy decreased brain tumor volume and proliferation in comparison with monotherapies. **Conclusion:** BET/RTK inhibitor synergy allows for dose reduction and decreased toxicity while enhancing the therapeutic efficacy of RTKis in HER2+ BC brain metastases.

LAY SUMMARY

Some breast cancers (specifically HER2-positive) can spread to the brain, becoming very difficult to treat. While certain drugs can reach the brain, they often don't work well or cause serious side effects. We focused our search on drugs that show synergy, the phenomenon where combining two or more treatments results in a therapeutic effect that is greater than the sum of the individual effects. We tested hundreds of drug combinations in lab-grown breast cancer cells taken from brain tumors. We then used computer tools and mouse experiments to check whether the drugs could reach the brain and shrink tumors. We identified a drug called AZD5153 (a BET/BRD4 inhibitor) that synergized well with Pozitotinib (an existing cancer drug). The combination therapy helped to shrink brain tumors in mice more effectively than either drug alone. The synergistic action of these drugs also helped to lower the effective dose of Pozitotinib, allowing for a reduction of the dose-dependent side effects.

CRC Unplugged: Tracing NFE2L3 from gut microbiota to colitis and cancer - Poster #431

Presenter: Anantpreet Kaur Sood, McGill University

Other Authors: James Saliba, Linda Yaker, Julia Osborne, Volker Blank

ABSTRACT_ID#2816

ABSTRACT

Prevalence of early-onset colorectal cancer (CRC) (18-49yo), contrary to late-onset CRC (>50yo), is increasing alarmingly and primarily present in the distal/rectal colon. Our lab found that loss of NFE2L3 transcription factor in a colitis-induced CRC mouse model preferentially reduced number of distal colon tumors. NFE2L3 promotes growth of colorectal cancer cells and colon inflammation, both in humans and mice, suggesting that NFE2L3 is a driver of early onset CRC in the context of an underlying inflammatory condition. Our recent studies also revealed a function for NFE2L3 in oxidative stress signaling in the colon. RNA-seq analyses revealed significant gene expression changes following NFE2L3 knockdown, and integration with ChIP-seq data enabled us to identify candidate target genes for functional validation. Additionally, we found that NFE2L3 loss reduces inflammation in mice treated with probiotics, highlighting a potential link to the gut microbiome. To further explore this connection, we performed a large-scale mouse study to determine whether treatment with probiotics alters inflammation and bacterial strain composition. Thus, the obtained samples underwent full-length 16S rRNA sequencing coupled with database analyses and molecular approaches to identify changes in the microbiome composition and examine the effect of microbiota on inflammation. By investigating these target genes of NFE2L3 in human cellular models and assessing the link between NFE2L3 and the gut microbiota in mice, we will determine how NFE2L3 transcription factor activity may uncover the molecular mechanisms leading to inflammation-induced, early onset CRC. These studies may lead to the design of novel therapeutic avenues for colon cancer patients.

LAY SUMMARY

NFE2L3 functions possibly as a key driver gene in early-onset colorectal cancer through inflammation: Our studies using a colitis-associated colorectal cancer mouse model revealed that loss of the transcription factor NFE2L3 significantly reduces tumor burden in the distal colon, a common site of early-onset CRC. This suggests that NFE2L3 promotes cancer progression under inflammatory conditions. NFE2L3 regulates oxidative stress and proinflammatory gene expression: Knockdown of NFE2L3 alters key pathways related to inflammation and oxidative stress, based on our studies on both human cells and mice. We identified candidate NFE2L3 target genes that may mediate its tumor-promoting effects and are currently validating their functional roles in human colorectal cancer cell models. NFE2L3 potentially modulates inflammation via the gut microbiome: Probiotic treatment in NFE2L3-deficient mice led to reduced colonic inflammation, indicating a potential interaction between NFE2L3 and gut microbial dynamics. To explore this, we are analyzing the microbiome changes to identify microbial shifts and their associations with inflammatory responses.

FANCI L604F sensitizes cells to low-grade replicative stress without affecting HDR - Poster #432

Presenter: Dardan Konjusha, McGill University

Other Authors: Yojiro Yamanaka

ABSTRACT_ID#2771

ABSTRACT

Primary risk factors for high-grade serous ovarian carcinoma are mutations in BRCA1/2 – members of the Fanconi Anemia (FA) pathway – with key roles in homology-directed repair (HDR) of interstrand crosslinks (ICLs). Loss of FA Complementmentation group I, FANCI, results in failure to activate the FA pathway, with ensuing hypersensitivity to ICLs. The FANCI L605F variant (Mouse: L604F) was identified in a family with multiple ovarian cancer patients lacking BRCA1/2 mutations, implicating FANCI as a new ovarian cancer-predisposing gene. Unlike BRCA1/2 mutations, FANCI mutations are insensitive to PARP inhibition, suggesting functional points apart from HDR. Interestingly, FANCI, independently of the FA pathway, plays a role in resolution of transcription-replication conflicts as well as modulation of dormant origin firing. We have established mice carrying a L604F variant or a KO allele. KO mice are infertile in both sexes, while L604F females have a reduced number of oocytes. L604F females present with premature age-associated vacuolization of oviduct epithelial cells, which is linked to cellular stress. MEFs derived from L604F embryos and treated with low concentrations of hydroxyurea have increased expression of γ H2AX and Cleaved-Caspase-3, while the same concentration elicits no effect on WT or KO MEFs. Conversely, ICL induction by Mitomycin C induces strong expression of γ H2AX in WT and L604F MEFs, but to a lesser extent in KO MEFs, indicating that HDR/ICL-detection is unaffected by the L604F mutation. Our findings suggest that the L604F point mutation creates a unique cellular environment more susceptible to replicative stress without affecting FANCI's canonical role in ICL-detection.

LAY SUMMARY

FANCI L604F functionally resides elsewhere than homology-directed repair - the canonical role of Fanconi Anemia pathway proteins such as BRCA1/2. The L604F mutation increases the cells susceptibility to replicative stress. Our findings further support the classification of FANCI as a novel ovarian cancer-predisposing gene.

Determining the impact reduced FBXO44 expression has on chromosome instability in colorectal cancer - Poster #433

Presenter: Demi Andromidas, Paul Albrechtsen Research Institute CancerCare Manitoba

Other Authors: Zelda Lichtensztejn, Nicole Neudorf, Rubi Campos Gudiño, Pamela Hebbard, Kirk McManus

ABSTRACT_ID#3161

ABSTRACT

Introduction: Colorectal cancer (CRC) is the fourth most diagnosed cancer and the second leading cause of cancer-related mortality in Canada. Chromosome instability (CIN), defined as ongoing gains or losses in whole chromosomes or chromosome fragments, occurs in ~70% of CRC cases and is implicated in cellular transformation, tumour progression, therapeutic resistance, metastasis, and unfavourable clinical outcomes. However, the aberrant genes underlying CIN remain elusive. The SCF (SKP1-CUL1-F-box protein) E3 ubiquitin ligase complex, and specifically FBXO44 within this complex, is hypothesized to play a critical role in maintaining chromosomal stability. FBXO44, a ubiquitylation gene responsible for targeting substrates for degradation, is thought to induce CIN when its expression is reduced. Methods: In this study, single-cell quantitative imaging microscopy was employed to characterize CIN-associated phenotypes following reduction of FBXO44 expression in karyotypically stable non-malignant and malignant colonic epithelial cell lines. FBXO44 expression was transiently reduced through siRNA-based silencing. Briefly, CIN phenotypes include changes in nuclear area distribution, increases in micronuclei formation (i.e., extranuclear DNA-containing bodies), and aberrant chromosome numbers. Results: Quantitative analyses indicate that siRNA-silenced FBXO44 results in statistically significant alterations in CIN phenotypes relative to controls, supporting its potential role in the regulation of chromosomal stability. Conclusions: These findings suggest that FBXO44 may serve as a key modulator of chromosomal stability in CRC, and its dysregulation may contribute to disease progression and poor patient outcomes. Further investigation into the role of FBXO44 in CIN may provide novel insights into CRC pathogenesis and identify potential therapeutic targets.

LAY SUMMARY

Chromosome instability (CIN) occurs in ~70% of colorectal cancers (CRCs) and is proposed to be a major driver of early disease development, yet the underlying causes promoting CIN remain largely unknown. Genomic loss of FBXO44 occurs in ~34% of CRCs, and is associated with reduced expression, genome instability and worse CRC patient outcomes. FBXO44 silencing corresponds with increases in CIN phenotypes, including nuclear areas, micronucleus formation and aberrant chromosome numbers, suggesting it is a novel CIN gene with pathogenic implications for CRC.

FLT4, a novel regulator of Renal Cell Carcinoma progression - Poster #434

Presenter: Djazia Haferssas, Université de Montréal

Other Authors: Jonatan Barrera Chimal, Nathalie Henley, Casimiro Gerarduzzi

ABSTRACT_ID#3255

ABSTRACT

Background: Renal Cell Carcinoma (RCC) is among Canada's 10 most frequent cancers. Disruption of p53 function, a critical tumor suppressor, plays a pivotal role in cancer development. While p53 mutations are frequent, these occur at low incidence in RCC. Reactivating this pathway presents a potential therapeutic strategy as MDM2 and MDMX, the major negative regulators of p53, are frequently elevated in RCC patients. However, the molecular mechanisms underlying MDM2/MDMX-mediated p53 regulation in RCC are unclear. Our study identified FLT4 as a novel regulator of MDM2/MDMX and p53 activity. Notably, both FLT4 and its ligand, VEGF-C, are overexpressed in RCC patients. We propose that FLT4 activation enhances the stability of the MDM2/MDMX complex, resulting in p53 inactivation and promoting RCC tumorigenesis. Objective: Determine the molecular mechanisms of FLT4-mediated p53 inactivation in RCC. Methods: 1) Bioinformatic analysis of RNAseq data of FLT4 and VEGFC expression in RCC patients. 2) RCC cell lines (ACHN and Caki-2) treatment with VEGFC and/or FLT4 inhibitor for functional assays, as well as p53 analysis. 3) Transfection with combination of FLT4, MDM2 and MDMX plasmids for p53 axis analysis. 4) RCC cells transduction with Luciferase and FLT4 and injection in immunodeficient mice to study the tumor development and metastatic potential. Results: FLT4 activation stabilizes MDM2/MDMX and degrades p53. This p53 degradation lowers the sensitivity to genotoxic treatments and promotes RCC cell proliferation and migration in vitro and in vivo. Conclusion: Aberrant FLT4 signaling contributes to p53 suppression through the modulation of MDM2/MDMX promoting RCC proliferation and survival.

LAY SUMMARY

- Our study has identified FLT4, a Receptor Tyrosine Kinase frequently deregulated in Renal Cell Carcinoma, as a key player in cancer progression.
- FLT4 and its ligand, VEGF-C, are both overexpressed in Renal Cell Carcinoma, leading to aberrant activation of FLT4.
- The aberrant activation of FLT4 in Renal Cell Carcinoma is observed in tumor epithelial cells and plays an oncogenic role. Specifically, FLT4 activation increases the levels of MDM2 and MDMX, which are negative regulators of p53. This results in enhanced degradation of p53. Since p53 is a crucial tumor suppressor that regulates cell proliferation and survival, its degradation leads to increased proliferation and survival of tumor cells.
- FLT4 was previously known for its role in controlling vascularization. Our study reveals an additional role in promoting cancer progression, specifically in Renal Cell Carcinoma, through the modulation of the MDM2/MDMX/p53 signaling axis.

Challenges of galectins in the context of cancer cell biology - Poster #435

Presenter: Alexander Timoshenko, Western University

ABSTRACT_ID#3246

ABSTRACT

Galectins represent a family of soluble glycan-binding proteins, which are proposed as diagnostic/prognostic biomarkers and targets for cancer therapy. However, although many galectins are reported to change their expression in cancer cells and tissues, the clinical success of galectin-related drugs including galectin inhibitors is still inconclusive. One conceptual idea, which is often overlooked, is the difference in the expression patterns and functions of common and tissue-specific galectins. While the elevated expression of some galectins is frequently associated with tumor progression and metastases, many galectins (e.g. 4, 7, 8, 9, and 12) are claimed as tumor suppressors. This report will review original findings with two highly tissue-specific galectins encoded by LGALS12 and LGALS16 genes in support of the concept of tissue-specific galectins as tumor suppressors marked by their upregulation upon differentiation of cancer cell lines. Our findings revealed that the expression of LGALS12 was upregulated by all-trans retinoic acid in cells representing leukemia (HL-60) and breast cancer (MDA-MB-231), while LGALS16 was upregulated through cAMP-mediated signaling in cell lines originated from choriocarcinoma (BeWo and JEG-3) and glioblastoma (U-87MG). These changes were associated with specific features of differentiated cells representing neutrophilic, epithelial, trophoblastic, and neuronal cell lineages based on the cell morphology and expression of differentiation markers. In addition, the tissue expression of LGALS12 was downregulated in all molecular subtypes of breast cancer, suggesting its potential role as a tumor suppressor gene. Thus, the role of tissue-specific galectins might require more focused attention considering their tumor suppressor functions and differentiation therapy of cancer.

LAY SUMMARY

Galectins are glycan-binding proteins, which may change their expression in cancer cells and tissues. Depending on the type of galectins, they are proposed to play different roles in tumor development, progression, and metastasis. Our research supports the hypothesis that tissue-specific galectins may serve as tumor suppressors.

Identification of Molecular Targets Regulating the Entry of Platelet Derived Microparticles - Poster #436

Presenter: Félix Després, Université de Moncton

Other Authors: Gilles A. Robichaud

ABSTRACT_ID#2435

ABSTRACT

Cancer metastasis is responsible for 90% of deaths associated to the disease. Understanding the mechanisms driving metastasis is vital to mitigate mortality rates and improve patient outcome. Our research team has recently reported that cancer cells have the ability to internalize platelet-derived microparticles (PMPs) and gain bioactive content transferred by PMP cargo. As a result, cancer cells gain malignant features (ex: migration and invasion) and impart drug resistance thus contributing to disease progression. Given that the mechanisms enabling cancer cells to internalize PMPs are unfortunately not well understood, we set to identify key surface receptors mediating PMP entry into target cells. Using a combined approach of bioinformatics (artificial intelligence) and mass spectrometry on a series on PMP-permissive and non-permissive target cells, we generated a list of surface receptors as potential PMP internalization mediators. Current validation studies are under way using RNAi silencing approaches on selected receptors to further identify specific mediators of PMP cancer cell entry. Our ultimate goal is to characterize PMP internalization mechanisms to develop new strategic inhibitors against PMP cancer cell internalization processes. As a result, extracellular vesicle (EV) blockade would amputate the cancer cells' ability to benefit from EV transfer of bioactive cargo and their functions driving cancer malignancy.

LAY SUMMARY

Cancer cells have the ability to internalize platelet-derived microparticles (PMPs) and gain bioactive content transferred by PMP cargo. The mechanisms enabling cancer cells to internalize PMPs are unfortunately not well understood. Using a combined approach of bioinformatics (artificial intelligence) and mass spectrometry on a series on PMP-permissive and non-permissive target cells, we generated a list of surface receptors as potential PMP internalization mediators. Our ultimate goal is to characterize PMP internalization mechanisms to develop new strategic inhibitors against PMP cancer cell internalization processes. As a result, extracellular vesicle (EV) blockade would amputate the cancer cells' ability to benefit from EV transfer of bioactive cargo and their functions driving cancer malignancy.

Investigation of IDH2 inhibition-associated vulnerabilities in triple negative breast cancer cells - Poster #437

Presenter: Bahareh Heidari, Institute for Research in Immunology and Cancer, Université de Montréal

Other Authors: Nariman Gharari, Roberto Piva, Sylvie Mader

ABSTRACT_ID#3279

ABSTRACT

Introduction: Isocitrate dehydrogenase 2 (IDH2) is a key mitochondrial enzyme in the tricarboxylic acid cycle, catalyzing the oxidative conversion of isocitrate to alpha-ketoglutarate (α -KG) in a reversible manner. WT-IDH2 inhibition was shown to suppress the migration and invasion of mesenchymal triple negative breast cancer (TNBC) cells through accumulation of α -KG, which decreases ATP synthesis and HIF1 α levels, in turn inhibiting glycolysis. We hypothesize that targeting WT-IDH2 can be part of a therapeutic regimen for mesenchymal TNBC tumors. **Methods/Results:** We used a genome-wide CRISPR knockout lentiviral library to decipher the mechanism of mesenchymal TNBC MDA-MB-231 cell killing by the IDH2 inhibitor, AGI6780. Gene set enrichment analysis revealed that deletion of genes related to the oxidative phosphorylation (OXPHOS) pathway, particularly complex I and V, were synthetic lethal with AGI6780 treatment. Validation experiments confirmed that genetically knocking out of the NDUFS1 subunit of complex I or the ATP5O subunit of complex V decreased proliferation of cells in the presence of AGI6780. In addition, pharmacological inhibition of complex V by Bedaquiline increased sensitivity to AGI6780 treatment, evidenced by a drastic reduction in ATP level and increased cell death. Interestingly, although TNBC tumors tend to favor glycolysis, viability of MDA-MB-468 cells, another TNBC model, was also strongly reduced when OXPHOS was disrupted by combination of IDH2 and complex V inhibitors. **Impact:** Our study contributes to the identification of targets that can synergize for the killing of TNBC breast cancer cells and may lead to novel therapeutic approaches for suppressing metastatic progression of TNBC tumors.

LAY SUMMARY

Targeting TNBC (triple negative breast cancer) energy metabolism: We studied TNBC as an aggressive form of breast cancer that lacks hormone receptors and HER2 expression, limiting the availability of targeted therapeutic options. We focused on a mitochondrial enzyme called IDH2 (isocitrate dehydrogenase 2), which helps cells produce energy. Inhibition of this enzyme reduced the energy supply, and increased cell death. **Genome-wide screening identified weak points:** Using a genome-wide CRISPR knock out screening in MDA-MB-231 cells in the presence of IDH2 inhibitor, AGI6780, we found that cells became especially vulnerable when we inhibited both IDH2 and parts of the electron transport chain, Complex I and/or Complex V. **Therapeutic potential beyond one cell type:** This combination approach worked not just in one, but in two different TNBC cell models—suggesting that many TNBC tumors which are typically relying on glycolysis, may still be sensitive to therapies that disrupt mitochondrial function.

The functional role of PGCKA1 within recurrent chromosome 4p deletion in triple negative breast cancer - Poster #438

Presenter: Elena Kuzmin, Concordia University

ABSTRACT_ID#2703

ABSTRACT

Basal breast cancer subtype is enriched for triple-negative breast cancer (TNBC) and displays consistent large chromosomal deletions. We previously showed chr4p is a recurrent deletion in basal breast cancer, which is associated with a poor outcome and evolves early in tumorigenesis. Mechanistically we showed that chr4p loss is associated with enhanced proliferation. Gene function studies identified a novel tumor suppressor gene, C4orf19 (PGCKA1), which is a member of the PDCD10-GCKIII kinase module. Here, we characterize the biochemical interaction between PGCKA1 and PDCD10 to gain insight into its functional role in TNBC. PGCKA1 lacks a crystal structure but InterPro and AlphaFold predict it to be disordered. Modeling the interaction between PGCKA1 and PDCD10 using AlphaFold-Multimer indicate that PGCKA1 directly binds through its C-terminus to the FAT domain of PDCD10. To validate these predictions, we purified PGCKA1 and PDCD10 proteins and conducted GST-pull down assays. Our findings indicate the presence of a band at an expected molecular weight of PGCKA1 when incubated with purified full length PDCD10, suggesting that these proteins interact directly. The GST pull-down assays with truncation mutants of PDCD10 indicate that the C-terminal FAT domain and not ND domain is required for the interaction with PGCKA1. Further, biochemical and cell biology analysis with mutants of both proteins will be important to confirm the domains and specific residues that mediate the interaction and regulate proliferation. Ultimately, the findings from this study will provide knowledge of a novel tumor suppressor within TNBC specific recurrent large chromosomal deletion.

LAY SUMMARY

Cancer is associated with an accumulation of alterations in genes that confer the ability of cancer cells to grow uncontrollably. These alterations can contribute to increased deleterious function or loss of function of genes that normally regulate cell growth. We characterize a poorly known gene PGCKA1 which resides within a frequently deleted region on chromosome 4 in triple negative breast cancer, an aggressive form of breast cancer.

Regulation of glioblastoma metabolism by fatty acid-binding protein 7 and its omega-3 ligand docosahexaenoic acid (DHA) - Poster #439

Presenter: Cécilia Bernier, University of Alberta

Other Authors: Seth Peyton, Clément Chevret, Maryam Lo, Hélène Lemieux, Roseline Godbout

ABSTRACT_ID#2835

ABSTRACT

Glioblastoma (GBM) is a highly aggressive brain cancer with poor prognosis. Tumour resistance and recurrence have been attributed to GBM stem-like cells (GSCs), which express high levels of fatty acid-binding protein 7 (FABP7). FABP7 facilitates the uptake and intracellular transport of dietary omega-3 and omega-6 polyunsaturated fatty acids. We previously showed that the omega-3 docosahexaenoic acid (DHA) inhibits cell migration in FABP7-expressing GSCs. In contrast to differentiated GBM cells, GSCs preferentially utilize fatty acid oxidation for energy production. As DHA treatment promotes FABP7 co-localization with mitochondria in GSCs, we hypothesize that FABP7 contributes to metabolic reprogramming in GBM and that DHA's anti-migratory effects may involve changes in mitochondrial function. Two approaches were used for this study: (i) mass spectrometry to evaluate the effects of DHA and FABP7 on metabolite levels in GSCs, and (ii) high-resolution respirometry to assess the effects of FABP7 and DHA treatment on oxidative phosphorylation (OXPHOS). Metabolomics analyses showed increased levels of DHA in GSCs cultured in DHA-enriched medium, an effect that was dependent on FABP7. We also found that DHA treatment led to changes in glutathione and amino acid metabolism, indicating a rerouting of antioxidant pathways. Respirometry experiments revealed a role for DHA in mitochondrial coupling efficiency, suggesting altered membrane properties and/or OXPHOS pathway use. Together, these results indicate that FABP7 influences how GBM cells adapt their metabolism to DHA. Next, we will examine how FABP7 and DHA influence specific OXPHOS pathways, including amino acid-linked pathways, with the aim of identifying new therapeutic targets for GBM.

LAY SUMMARY

Three key points:

1. Fatty acid-binding protein 7 (FABP7) is associated with decreased survival in glioblastoma patients and is highly expressed in glioblastoma stem-like cells believed to be the underlying cause for treatment resistance and recurrence. A preferred ligand of FABP7 is the omega-3 fatty acid docosahexaenoic acid (DHA) well-recognized for its health benefits.
2. Treatment of FABP7-expressing glioblastoma cells with DHA results in co-localization of FABP7 with mitochondria, the powerhouse of the cell, as well as changes in metabolism, including a reduced efficiency in mitochondrial oxidative phosphorylation and a decrease in several amino acids and their metabolites.
3. We are investigating whether targeting FABP7 function and/or DHA supplementation in glioblastoma cells can limit or reverse their aggressive behaviour using different approaches, including measurement of energy production and mitochondrial properties.

Molecular and Cellular Biology

Monday, November 3, 2025

Nck adaptor proteins regulate breast cancer cell invasion and metastasis - Poster #440

Presenter: Erka Shata, University of Guelph

Other Authors: Adam Golding, Sarah Wismer, Una Adamcic-Bistrivoda, Claire Martin, Laura New, Peihua Lu, Manali Tilak, Megan Brasher, Ji Shin, Hongmei Zhao, Caihong Ma, Marc Coppolino, Josie Ursini-Siegel, Nina Jones

ABSTRACT_ID#3270

ABSTRACT

Nck1 and Nck2 are well established adaptor proteins in pathways regulating actin cytoskeleton remodeling. Work from multiple laboratories has shown a role for these proteins in cancer progression and metastasis. We have now determined that Nck1 and Nck2 are central regulators of breast cancer progression. Systematically profiling Nck across TCGA-BRCA and related datasets, we found that upregulation of Nck in breast cancer correlates with negative outcomes. Nck is shown to be mostly upregulated in triple negative breast cancer (TNBC) and cancers expressing the HER2 receptor. Using an in vivo loss of function strategy in mice which allows simultaneous expression of activated oncogene HER2/ErbB2 and Cre recombinase in mammary epithelial cells, we have shown that deletion of both Nck1 and Nck2 (NckDKO) significantly extends survival by delaying tumour onset and also reduces incidence of lung metastasis. Building upon these findings, we have characterized Nck knockout HER2+ cancer cell lines and shown a role of these proteins in cancer migration and invasion. We also compared whole proteome of NckNT (non-target/control) and NckDKO (double knockout) spheroids using mass spectrometry. From this screen, 6560 differently expressed proteins were identified in the DKO spheroids compared to the NT ones. Complementary, we have shown that Nck overexpression in TNBC cells results in enhanced invasion, gelatin degradation and invadopodia formation. Our findings provide new physiological insights verifying the role of Nck as an oncogene. This research is important as understanding signaling pathways involved in breast cancer is crucial in developing therapies to prevent and treat it.

LAY SUMMARY

The Nck adaptor proteins have been linked to cancer progression. Using patient data, a mouse model of breast cancer and cultured breast cancer cell lines, we have found that increased levels of Nck correlate with worse cancer phenotypes in two types of metastatic breast cancer. Additionally, our ongoing experiments implicate Nck in driving breast cancer invasion, which is required for metastasis, thereby suggesting it could be a novel target for anti-cancer therapeutics.

Characterizing the role of sFRP1 in melanoma: phenotypic modulation and Wnt signalling - Poster #441

Presenter: Cierra Perron, Queen's University

Other Authors: Douglas Quilty, Farzaneh Afzali, Bianca Dauber, Lynne Postovit

ABSTRACT_ID#3332

ABSTRACT

Melanoma is a highly aggressive cancer characterised by early metastatic potential and resistance to conventional therapies, with limited options for advanced-stage patients. The Wnt signalling pathway plays a critical role in tumour development and progression and is modulated extracellularly by Secreted Frizzled-Related Proteins (sFRPs). Among this family, sFRP1 is one of the most extensively studied members and has demonstrated context-dependent roles across malignancies, functioning as a tumour suppressor in some settings while promoting oncogenic activity in others. Despite its relevance in other cancers, the precise role of sFRP1 in melanoma remains poorly defined. Our group identified elevated sFRP1 expression in melanoma metastases, with high levels correlating with decreased overall and distant metastasis-free survival in patient datasets. We hypothesised that sFRP1 influences melanoma progression through modulation of Wnt signalling and downstream cellular behaviours. Comparative profiling of melanoma cell lines revealed marked heterogeneity in the expression of sFRP1, Wnt ligands, and receptors, indicative of distinct signalling microenvironments that may condition sFRP1 activity. Functional assays demonstrated that sFRP1 exerts divergent, cell-line-specific effects on oncogenic behaviours, including proliferation, migration, and invasion, underscoring the context-dependent, multifactorial nature of its activity. Preliminary data also suggest that sFRP1 may modulate aspects of the tumour microenvironment relevant to immune regulation, unveiling a previously unrecognized dimension of its function in melanoma. These findings support a multifaceted role for sFRP1 in melanoma progression, shaped by the local signalling milieu, with potential implications for both tumour behaviour and therapeutic responsiveness. Ongoing studies aim to delineate the molecular mechanisms underlying these effects.

LAY SUMMARY

sFRP1 decreased proliferation of cells cultured in non-adherent conditions, and decreased invasion, and migration. sFRP1 demonstrated cell-line-specific effects over Wnt signalling mediators that further manifested as cell-line specific impacts over melanoma cell behaviours.

Aurora kinase A promotes a leader cell phenotype and is required for EPLIN-mediated cohesion with follower cells during collective cell migration - Poster #442

Presenter: Chelsey Gough, University of British Columbia

Other Authors: Bo Peng Zhou, Chelsey Gough, Christopher Maxwell, Tariq Bhat, C. James Lim

ABSTRACT_ID#3040

ABSTRACT

The metastatic process is believed to initiate with collective cell invasion into surrounding tissues and axillary nodes, and subsequent colonization at a distant site. Previously, we found collective invasion is augmented during the G2 cell cycle phase, facilitated through Aurora kinase A (AURKA)-mediated centrosome polarization in the leader cell. Here, we identify cell cycle-associated gene signatures as overrepresented in axillar and liver metastatic sites, with AURKA expression strongly correlated with breast cancer metastasis signatures, and pan-cancer patient survival. Then, we show GFP-AURKA expression endows breast epithelia cells with the ability to locally invade in organoids and, separately, form metastatic outgrowths when transplanted intra-amniotically within immune-incompetent chicken embryos. Multi-parametric imaging of wound closure assays reveals phenotypes enabled by, and dependent upon AURKA expression. We discover leader cells express AURKA and acquire front-polarized centrosomes, which differentiates them from other cells in the migrating group. Ectopic expression of GFP-AURKA induces a leader cell phenotype. Conversely, inhibition of AURKA activity alters actin dynamics, promotes proteasome-mediated turnover of cell contacts, and reduces coordination within migrating groups. Specifically, AURKA inhibition mislocalizes the actin regulator EPLIN to lamellipodia at the leading edge and away from E-cadherin-positive contacts. These necessary roles for AURKA make its inhibition a potential barrier to the metastatic spread of human breast carcinoma cells.

LAY SUMMARY

Movement of cellular collectives, such as during cancer metastasis, requires the formation of a unique cell phenotype, called leader cells, and connection with following cells. To form a leader cell requires changes to the cell's cytoskeleton, which can be induced by the abnormal expression of a mitotic kinase, termed Aurora kinase A. Aurora kinase A activity is also required to maintain connections with follower cells in the migrating cluster.

Identification of the Artemis-MRN interaction interface for regulation of DNA double strand break repair - Poster #443

Presenter: Danny Laurent, University of Calgary

Other Authors: Alexander Cobban, Chi-Lin Tsai, Stephanie Hiebert, Yaping Yu, Justin La, Joshua Heyza, Binhang Zhu, Miaw-Sheue Tsai, Jens Schmidt, John A. Tainer, Susan P. Lees-Miller

ABSTRACT_ID#2820

ABSTRACT

Artemis (DCLRE1C) endonuclease is canonically required for resolving closed DNA hairpins during V(D)J recombination in the adaptive immune system. Yet, it also plays an enigmatic non-canonical role in repair of ionizing radiation (IR)-induced DNA damage. Artemis loss or inactivation results in Severe Combined Immuno-Deficiency with Radiation Sensitivity (RS-SCID). Moreover, Artemis over-expression in a subset of breast, urinary tract, stomach and ovarian cancers makes it an attractive but underappreciated cancer therapy target. Importantly, the intrinsically disordered (IDR) C-terminal tail of Artemis interacts with several DNA double strand break (DSB) repair proteins including DNA-dependent protein kinase catalytic subunit (DNA-PKcs), DNA ligase IV (LIG4) and the MRE11, RAD50, NBS1 (MRN) complex. Here, we identify the critical region in the Artemis C-terminal tail for interaction with MRN. Furthermore, we show that phosphorylation of three conserved serines within this region is required for the interaction. The discovered interaction is DNA damage induced, requires the activity of ATM and/or DNA-PKcs, and is mediated by the N-terminal FHA-BRCT1-BRCT2 domain of NBS1. Computational modeling revealed a high-reliability AlphaFold3 interface for the phospho-Artemis-NBS1 complex that is being tested experimentally. Our collective data suggest that IR-induced activation of ATM and/or DNA-PKcs promotes Artemis IDR phosphorylation that triggers interaction of its CTR with NBS1 FHA-BRCT1/2 domain of the MRN complex. These phosphorylation-dependent interactions appear suitable to regulate multiple DSB repair pathway and signaling pathways. Notably, targeted disruption of this interface could have therapeutic potential for regulation of the DNA damage response in normal and cancer cells.

LAY SUMMARY

We biochemically characterized interactions of DNA repair factors Artemis with the MRN complex. This interaction is induced in the presence of DNA damage including by ionizing radiation and some chemotherapeutic agents. Understanding of Artemis-MRN interaction interface would allow us to disrupt the interaction, see its effects on DNA repair capacity of non-neoplastic and cancer cells, and potentially be used to sensitize cells to radiation therapy.

MAEA Prevents Harmful Resection at Stalled Replication Forks by Promoting Homologous Recombination Repair - Poster #444

Presenter: Elham Zeinali, University of Alberta

Other Authors: Fatemeh Mashayekhi, Rabih Abou Farraj, Talah Hasanni, Marie-Christine Caron, Yan Coulombe, Amira Fitieh, Mark Glover, Jean-Yves Masson, Ismail Ismail

ABSTRACT_ID#3107

ABSTRACT

Targeting DNA repair pathways to enhance cancer cell sensitivity to chemotherapy and radiotherapy has a proven track record of success in the clinic. Cancer therapies that deliberately exacerbate replication stress (RS), exploit the fragile dependence of tumour cells on accurate replication-fork (RF) restart. Homologous recombination (HR) is the primary pathway that facilitates the restart and repair of stalled or collapsed RFs. However, the precise mechanisms governing HR at stressed forks remain insufficiently defined. Through an E3 ubiquitin ligase gene siRNA screen, we discovered a novel function for MAEA (Macrophage Erythroblast Attacher), a core member of the human C-terminal to LisH (CTLH) E3 ubiquitin ligase complex, in HR pathway. MAEA promotes RAD51 recruitment at stalled RFs and protects fork integrity. MAEA depletion impairs HR efficiency and increases cellular sensitivity to agents that induce RS (CPT, HU, and PARPi). Additionally, loss of MAEA disrupts replication fork restart and leads to uncontrolled degradation of nascent DNA under RS, highlighting its essential role in fork recovery and genome stability. Mechanistically, our data show that MAEA associates with Ku heterodimer and mediates its ubiquitylation, a critical step for Ku removal from stressed RFs. In MAEA-deficient cells, Ku persistently accumulates at fork ends, interfering with RAD51 filament formation. Strikingly in silico analysis revealed that ovarian cancer exhibits the highest MAEA amplification frequency across all cancers. We will explore if MAEA loss by disrupting HR repair, resensitizing PARPi-resistant ovarian cancer to treatment while forcing reliance on alternative DSB repair pathways.

LAY SUMMARY

We discovered a new role for a protein called MAEA, which helps protect DNA when cells are under stress during replication, a process essential for preventing genome damage and cancer development. MAEA promotes a key DNA repair pathway called homologous recombination (HR) by helping remove the Ku protein from stalled replication forks. This allows RAD51 to bind the DNA and restart repair. Without MAEA, Ku builds up, blocking repair and causing harmful DNA breakdown. Without MAEA, cells become highly sensitive to drugs that damage DNA, and they can't restart replication properly. This leads to uncontrolled DNA breakdown and contributes to genomic instability, a hallmark of cancer.

RBPscan: A Quantitative, In Vivo Tool for Profiling RNA-Binding Protein Interactions - Poster #445

Presenter: Dmitry Kretov, Centre de recherche du CHU de Québec – Université Laval

Other Authors: Marianne Régis, Paola Pulcina, Louis-Mathieu Harvey, Daniel Cifuentes

ABSTRACT_ID#3096

ABSTRACT

RNA-binding proteins (RBPs) are key regulators of post-transcriptional gene expression which are often dysregulated and mutated in cancer. However, profiling different RNA-protein interactions in vivo in normal or pathological context remains challenging primarily due to technical differences of existing approaches. We developed RBPscan, a massively parallel reporter assay that leverages RNA editing to quantitatively measure RNA-protein interactions in vivo at scale. RBPscan is independent of UV-crosslinking, protein purification, and antibodies, enabling high-throughput analysis of RNA-protein interactions in a native cellular environment. RBPscan consists of two main components: a writer and a recorder. The writer is a fusion protein combining the RBP of interest with the catalytic domain of the RNA editing enzyme ADAR, which converts adenosines into inosines. A recorder is a synthetic mRNA containing both the RBP target site and multiple adenosines optimally positioned for ADAR activity. When co-expressed, the extent of A-to-I editing in the recorder mRNA provides a quantitative measure of RNA-protein interaction strength. We will showcase the quantitative capabilities of RBPscan, present de novo motif identification for several RBPs, and illustrate its ability to detect microRNA activity. Additionally, we optimized the design of recorder mRNAs to have a fluorescent readout, enabling real-time visualization of RBP binding in vivo. Overall, RBPscan is a new universal approach to study RNA-protein interactions that complements existing techniques and has the potential to accelerate progress in our understanding of post-transcriptional regulation, especially in the context of cancer-related RBPs.

LAY SUMMARY

1. We developed a new method called RBPscan to study how proteins interact with RNA inside living cells. These interactions are important because they regulate all aspects of gene expression and are often disrupted in diseases such as cancer.
2. Compared to other methods, RBPscan is a relatively simple approach that does not require protein purification, special antibodies, or complex bioinformatic analysis.
3. RBPscan provides not only information about the specificity of RNA-protein interactions, but also quantitative insights into their strength.

APC and TP53 Co-Mutation Enables Fetal-like Stem Cell Persistence in Colorectal Cancer - Poster #446

Presenter: Fynn Mitchell, University of Calgary

Other Authors: Arshad Ayyaz

ABSTRACT_ID#3226

ABSTRACT

Colorectal cancer (CRC) remains one of the most prevalent and deadly cancers globally, with rising incidence and poor prognosis often associated with TP53 mutations (Sawicki et. al., 2021). CRC typically develops through sequential mutations, beginning with Adenomatous polyposis coli (APC) inactivation and followed by TP53 loss (Sawicki et. al., 2021). While TP53-mutant tumors frequently resist chemotherapy, the mechanisms enabling survival and expansion of therapy-resistant cells remain unclear (Sole et. al., 2022). Recent studies have highlighted the role of LGR5⁺ cancer stem cells (CSCs) in driving CRC progression in TP53-deficient tumors (Shimokawa et. al., 2017; Sousa e Melo et. al., 2017). However, TP53 loss in the normal intestine impairs epithelial regeneration, suppressing both **LGR5⁺ intestinal stem cells and fetal-like CLU⁺** revival stem cells which regenerate intestinal epithelia following damage (Morral et. al., 2023; Ayyaz et. al., 2019). This suggests that APC loss may act as a dysregulating event, enabling TP53-mutant cells to bypass regenerative checkpoints, survive therapeutic treatment, and initiate tumor regrowth. I hypothesize that TP53 loss alone is insufficient to promote CLU⁺ stem cell persistence and that co-mutation with APC is required to unlock a regenerative, tumor-permissive cell state. I am testing this hypothesis by integrating single-cell multi-omic transcriptomics, genetically engineered intestinal organoids and tumor spheroids, CRISPR/Cas9-based editing, and smFISH aiming to dissect stem cell behavior in defined genetic contexts (Gunti et. al., 2021). This work aims to uncover cooperative dynamics between APC and TP53 that govern cancer progression and therapeutic resistance, offering novel insights into CRC treatment.

LAY SUMMARY

Two mutations work together to fuel aggressive cancer: Mutations in the TP53 gene alone aren't enough to help colorectal cancer cells survive treatment. But when TP53 loss is combined with APC mutations, it may unlock a powerful regenerative state that helps cancer stem cells persist and regrow tumors after therapy. Cancer stem cells may hijack normal repair systems: In healthy intestines, TP53 is crucial for controlling regeneration. Its loss disrupts normal stem cell activity, but in cancer, APC mutations may override this block allowing mutated cells to activate damage-repair programs inappropriately, promoting tumor growth. New tools reveal how mutations reshape cancer cell behavior: By combining gene editing, advanced organoid models, single-cell analysis, and imaging, this research aims to pinpoint exactly how these mutations change stem cell dynamics potentially uncovering new strategies to block tumor resistance.

Exploring translational mechanism vulnerabilities to target cellular senescence following cancer treatments

- Poster #447

Presenter: Emilie Hingray, Université de Montréal

Other Authors: Karine Boulay, Paul Minaya, Krzysztof Szkop, Ola Larsson, Ivan Topisirovic, Frédérick-Antoine Mallette

ABSTRACT_ID#3243

ABSTRACT

Cellular senescence is a biological response to stress during which cells stop replicating while remaining metabolically active. It can be triggered by oncogene activation, telomere shortening, oxidative stress and DNA-damaging treatments like chemotherapy and radiation. Physiologically, this response can serve as a tumour-suppressive mechanism but the aberrant accumulation of senescent cells creates a pro-inflammatory environment through their senescence-associated secretory phenotype. This can contribute to age-related diseases and cancer progression. Targeted elimination of senescent cells can improve age-related conditions, prolong cancer suppression, limit relapse and reduce adverse effects of cancer treatments. Despite showing promise, current senolytics face important limitations that prevent their use in clinical settings. The Mallette lab recently identified a translational regulator necessary for the viability of senescent cells but not proliferative cells, revealing a potential therapeutic avenue. We have also identified differentially translated mRNAs in chemotherapy-induced senescence using polysome profiling. We hypothesize that regulatory mechanisms of mRNA translation enable the expression of genes essential for the induction and maintenance of cellular senescence. By repeating polysome profiling experiments and conducting CRISPR screens, we aim to study different types of cellular senescence at the translational level. In addition to chemotherapy-induced senescence, we will investigate oncogene-induced senescence and replicative-exhaustion induced senescence, employing RT-qPCR to validate top candidates in each type. Comparing the outcomes of the CRISPR screens will allow us to identify differentially translated mRNAs that sustain cellular senescence, regardless of the inducer. These findings could identify new vulnerabilities specific to senescent cells, providing potential targets to improve cancer therapies.

LAY SUMMARY

Cancer treatments like chemotherapy and radiation aim to eliminate cancer cells, but they can trigger a response known as cellular senescence both in cancer cells and in surrounding healthy cells. Cellular senescence occurs when cells stop replicating but still secrete molecules that can negatively impact the environment. Elimination of senescent cells following cancer treatments can prolong cancer suppression, limit tumour relapse and reduce adverse side effects of cancer treatments, suggesting that such interventions can improve oncology treatment outcomes. Current drugs to eliminate senescent cells have not been approved for clinical use. We have identified differences in the regulation of protein synthesis in the context of cellular senescence that we have decided to investigate further to hopefully find new vulnerabilities that can be used to eliminate senescent cells after cancer treatments.

Novel Insights into the Clinical Relevance of Perineural Invasion - Poster #448

Presenter: Nickson Joseph, University of Saskatchewan

Other Authors: Paulos Chumala, George Katselis, Anand Krishnan

ABSTRACT_ID#2903

ABSTRACT

Introduction The invasion of cancer cells into peripheral nerves, particularly into the perineural space is called perineural invasion (PNI). Although identified as a major risk factor for cancer progression, the molecular drivers of PNI and its direct clinical relevance are poorly understood. Using our in-vitro PNI model, we characterised the molecular profile of PNI competent breast cancer (BC) cells to reveal the insights into its clinical relevance. **Methods** Dorsal Root Ganglion (DRG)-nerve explants were embedded in Cultrex into which eGFP+ MDA MB 231 cells were seeded to create a 3D co-culture. After 14 days, the explants were isolated and used either for proteomic profiling experiment or immunostaining. Single cell suspension was made from enzymatic digestion of the 14-day old explants. The eGFP+ PNI BC cells were then sorted from the suspension using a cell sorter and proteins isolated from these purified cells were used for LC-MS/MS analysis. **Results** The 2-fold change list of proteins revealed downregulation of major metabolic pathway proteins from glycolysis, pentose phosphate pathway etc. The PNI cells appeared to have unique morphology and did not give positive staining for proliferation marker Ki67. The upregulated proteins like CPLANE-1 and hemopexin may be novel targets for theranostics. **Conclusion** Our findings indicate that BC PNI cells reach a state similar to dormancy where these cells shut down metabolic pathways suggesting the clinical relevance of PNI. Upregulated proteins - CPLANE-1 and hemopexin are potential candidates to target dormant PNI cells in in-vivo experiments or even clinically.

LAY SUMMARY

1. We used an in-vitro model which can better recapitulate perineural invasion (PNI) to study its biology. Previous in-vitro models in the literature lacks intact nerve which the cancer cells could use as a scaffold for PNI.
2. A novel understanding of the underlying cellular and molecular interplay was observed. An insight into the dormancy-like state of breast cancer PNI cells was observed which is novel.
3. We also showed the capacity of the model to screen potential targets of PNI for theranostics.

Patterns of expression and activity of Estrogen Receptor, FOXA1 and FOXC1 in the normal mammary gland mirror those in breast cancer subtypes - Poster #449

Presenter: Faustine Gorse, Institute for Research in Immunology and Cancer

Other Authors: Lucas Porras, Sylvie Mader

ABSTRACT_ID#3250

ABSTRACT

Breast cancer includes three clinical subtypes distinguished by expression of two tumorigenesis drivers, the hormone-dependent transcription factor (TF) estrogen receptor alpha (ER) and the membrane receptor HER2/ERBB2. Triple negative breast cancer (TNBC) lacks both proteins and the progesterone receptor (PR), an estrogen target gene. Molecular subtypes identified by unsupervised classification of whole tumor transcriptomes partially overlap with clinical subtypes and can be recapitulated by the differential RNA levels of specific TFs. We hypothesize that the TFs ER, FOXA1, and FOXC1, which are differentially expressed in tumors, regulate mammary epithelial cell differentiation and drive tumor subtype specification. Our aims are to characterize their expression in normal and tumor tissues and their contributions to gene regulation in normal and tumoral models. Spatial RNA profiling of normal human mammary glands using the GeoMx technology highlighted the differential expression of specific TFs and keratins in myoepithelial and luminal epithelial cells, and the co-expression of genes enriched in luminal tumors in ER+ vs ER- luminal cells. Co-immunofluorescence on mammary gland tissues validated the co-expression of FOXA1 and ER and revealed a mutually exclusive expression of FOXA1 and FOXC1. In tumors, the FOXC1 protein is expressed in TNBC, whereas FOXA1 is detected in ER+, HER2+, and some TNBC tumors. TF suppression in relevant cancer cell models indicated roles in suppression of myoepithelial-enriched genes, thus contributing to epithelial cell differentiation. These results suggest that cancer subtypes retain most of the gene expression programs of normal cells of origin, which may help identify subtype specific vulnerabilities.

LAY SUMMARY

My research will enhance our knowledge of the mechanisms of normal mammary cell differentiation. Two transcription factors of the same family, FOXA1 and FOXC1, are expressed in different populations of normal and tumor mammary cells. We developed a clinically relevant assay to identify breast cancer subtypes with different biology and therapeutic treatments.

Molecular and Cellular Biology

Monday, November 3, 2025

Investigating the role of SLK in melanoma progression - Poster #450

Presenter: Hong Meng Yew, Ottawa Hospital Research Institute

Other Authors: Luc Sabourin

ABSTRACT_ID#2888

ABSTRACT

Transcriptional reprogramming and phenotype switching transforms melanoma cells from a proliferative, drug-sensitive phenotype into an invasive, drug-resistant phenotype, respectively characterized by increased expression of SRY-box transcription factors Sox10 and Sox9. In melanoma, we observed that high-Sox9 invasive cells display elevated Ste20-like kinase (SLK) expression compared to high Sox10 proliferative cells. Our goal is to demonstrate that during phenotype switching in melanoma progression, SLK activity is required for increased invasiveness and stem-like properties through regulation of cytoskeletal dynamics and EMT proteins. We performed CRISPR/Cas9 targeted gene knockout on SLK in Yale University Mouse Melanoma (YUMM) cells and isolated single clones. The high Sox10 YUMM 1.1 cells and high Sox9 YUMM 1.7 cells represent the proliferative and invasive melanoma phenotypes respectively. In YUMM 1.7 cells, SLK knockout clonal lines displayed reduced proliferation, increased invasiveness, reduced vemurafenib resistance, and increased proportion of mesenchymal-like cell populations. RNA-Seq transcriptome analysis of YUMM 1.7 SLK knockouts revealed increased expression of genes related to Notch, TGFb, and canonical Wnt signaling pathways. In vivo subcutaneous injection demonstrated that YUMM 1.7 SLK knockout displayed reduced tumorigenesis and reduced tumor initiation capacity, whereas YUMM 1.1 SLK knockouts displayed increased tumorigenesis and increased tumor initiation capacity. SLK appeared to play divergent roles in proliferative and invasive melanoma cell types. Downstream pathways revealed by RNA-Seq analysis of SLK knockouts provided future study directions. Reduction in tumorigenic properties in YUMM 1.7 SLK knockouts both in vitro and in vivo suggests SLK as a valuable potential therapeutic target for treatment of advanced melanoma.

LAY SUMMARY

SLK inhibition greatly reduces tumor size and tumor forming capacity of invasive YUMM 1.7 melanoma cells in mouse models. This makes SLK an enticing potential target for the treatment of advanced melanoma in patients. SLK inhibition affects the Notch, TGFb, and canonical Wnt signaling pathways. This suggests that SLK acts as a “master switch” for multiple mechanisms involved in cancer cell growth, survival, and progression. SLK inhibition has opposite effects on proliferative YUMM 1.1 melanoma cells in mouse models compared to invasive YUMM 1.7 cells. This implies that SLK plays very different roles depending on the state of the cancer cell in question and can act as both oncogene and tumor suppressor.

Impacts of fatty acid β -oxidation enzyme ECI1 on aggressive prostate cancer phenotype - Poster #452

Presenter: Jacques Lapointe, McGill University

Other Authors: Karl-Philippe Guérard

ABSTRACT_ID#3327

ABSTRACT

Deregulated mitochondrial fatty acid β -oxidation is known to drive prostate cancer (PCa) pathogenesis. ECI1 ($\Delta 3$, $\Delta 2$ -Enoyl-CoA Delta Isomerase 1) is a key mitochondrial fatty acid β -oxidation enzyme whose role in PCa remains to be explored. We previously showed that high levels of ECI1 detected in prostate tumors were associated with poor clinical outcome. ECI1 overexpression increased in vitro PCa cell growth, colony formation, cell motility, and tumor growth and metastasis in vivo. While ECI1 enhanced the maximal mitochondrial respiratory capacity of PCa cells, the exact mechanisms by which ECI1 promotes tumorigenesis remain unknown. We hypothesize that ECI1, beyond its contribution to energy production, reprograms PCa cells leading to a pro-metastatic phenotype. Examining the morphology of ECI1 stably overexpressing PCa cells (PC-3 ECI1) in standard growing conditions, we noticed a large proportion of round cells with blebbing (amoeboid) compared to control expressing low levels of ECI1 (PC-3 mock), which display an elongated morphology with filopodia and lamellipodia (mesenchymal). Amoeboid phenotype is promoted by the Rho/ROCK signaling pathway and characterized by weak adhesion, no degradation of the ECM, but moving through it with high velocity. Western blots showed an increased phosphorylation of MLC2 in PC-3 ECI1 cells compatible with ROCK activation. So far, our results suggest that ECI1 would promote the reprogramming of PCa cells to an amoeboid phenotype that may explain, at least in part, the enhanced motility and metastasis potential. These results support the functional capacity of ECI1 in PCa progression and its potential as therapeutic target for clinical management.

LAY SUMMARY

ECI1, a fatty acid degrading enzyme involved in energy production, is found in large quantities in a subset of prostate tumors. High levels of ECI1 in prostate tumors are associated with an increased risk of progression and metastasis. Exploring the mechanism by which ECI1 promotes metastasis, this study showed that ECI1 changes the shape of cancer cells and the way they move, which may help them escape the primary tumor to form metastasis. These results suggest that ECI1 is involved in prostate cancer progression.

Investigating the Role of Secreted Plasminogen Receptors in the Establishment of the Pre-Metastatic Breast Cancer Niche - Poster #453

Presenter: Gillian Okura, Dalhousie University

Other Authors: Alamelu Bharadwaj, Christopher Hughes, David Waisman

ABSTRACT_ID#2379

ABSTRACT

Introduction: Breast cancer, the most common cancer in women, often presents with secondary tumors at diagnosis in 25% of cases. Metastasis, responsible for 90% of cancer deaths, involves cancer cells colonizing distant organs through premetastatic niches. These niches are prepared by cancer cell-secreted factors, including the protease plasmin, which degrades tissue barriers. Cancer cells use plasminogen binding proteins to convert the inactive zymogen plasminogen to plasmin. Our research shows that malignant breast cancer cells secrete more plasminogen-binding proteins than non-malignant cells. Several candidates for novel plasminogen-binding proteins have been identified, including phosphoglycerate kinase-1 (PGK1), which is upregulated in breast and metastatic tissue. Methods: Conditioned media was generated to study the breast cancer cell secretome and investigate novel plasminogen-binding proteins. Conditioned media was collected from paired human and mouse metastatic and non-metastatic breast cancer cell lines. Soluble proteins were separated by column chromatography, and plasmin generation was measured. Mass spectrometry identified several novel candidates for plasminogen-binding proteins, and recombinant proteins were evaluated for activity. Results: Malignant breast cancer cell lines' conditioned media (i.e. MDA MB 231 & 4T1) had greater plasmin-generating ability than non-malignant cells (i.e. MCF7 & 67NR respectively). Chromatography resolved several plasmin-generating peaks, and several glycolytic enzymes were identified in the peaks, including PGK1 and phosphoglycerate mutase-1 (PGAM1). Both proteins had plasmin-generating ability. Conclusions: Malignant breast cancer cells can secrete plasminogen-binding proteins, including glycolytic enzymes. Future experiments will focus on validating PGK1 and other novel proteins as plasminogen-binding proteins and elucidate their role in metastasis and establishment of the premetastatic niche.

LAY SUMMARY

Malignant breast cancer cells secrete more proteins that can bind to plasminogen, a molecule involved in breaking down tissue barriers, than non malignant breast cancer cells. This secretion may help cancer spread by preparing distant sites in the body for new tumours to grow. Proteins involved in glycolysis, including glycolytic enzyme phosphoglycerate kinase-1 (PGK1) and phosphoglycerate mutase-1 (PGAM1), can bind plasminogen and generate plasmin. Proteins were found in higher amounts in aggressive breast cancer cell lines. Secreted glycolytic enzymes might play a role in helping cancer cells establish the pre-metastatic niche, which are environments in distant organs that make it easier for cancer to spread. Future work will focus on confirming the roles of PGK1 and other proteins in this process.

Fructose transporter (SLC2A5)-dependent regulation of STAT3 expression in cancer cell metastasis - Poster #454

Presenter: Jody Groenendyk, University of Alberta

Other Authors: Myriam Pujol, Izabela Quest, Ning Dai, Ayshna Diya, Xing-Zhen Chen, Jingfeng Tang, Luis B. Agellon, Marek Michalak

ABSTRACT_ID#2294

ABSTRACT

Endoplasmic reticulum (ER) stress plays a crucial role in cancer progression, helping cells adapt to hostile microenvironments marked by low oxygen, nutrients, and pH, alongside high metabolic demands. Using an ER stress reporter assay, we found that silencing the fructose transporter gene SLC2A5 (GLUT5) reduced fructose uptake and inhibited the IRE1 α /spXBP1 arm of the ER stress response. This loss also impaired cancer cell migration and proliferation in MIA-PaCa-2 pancreatic cancer cells (Groenendyk et al., 2022). Reverse-phase protein array (RPPA) analysis revealed that STAT3 is a key downstream effector in response to reduced fructose uptake. In SLC2A5-deficient cells, STAT3 mRNA and protein levels, along with its transcriptional activity, were significantly decreased. Further analysis demonstrated that STAT3 is regulated by ER stress through the IRE1 α /spXBP1 pathway, linking SLC2A5 deficiency to impaired STAT3 signaling. Additionally, mitochondrial association of STAT3 was diminished in the absence of SLC2A5. Using SPLICS analysis, we observed a reduction in mitochondrial-ER contact sites, and flow cytometry revealed disrupted calcium (Ca²⁺) transfer from the ER to mitochondria. This disruption was accompanied by increased mitochondrial reactive oxygen species (ROS) levels. Together, these findings suggest that SLC2A5 deficiency compromises ER-mitochondrial communication, downregulates STAT3, impairs Ca²⁺ signaling, and increases oxidative stress, ultimately disrupting mitochondrial function in pancreatic cancer cells. Groenendyk J, et al. Loss of the fructose transporter SLC2A5 inhibits cancer cell migration. *Front Cell Dev Biol* 2022 Sep 30;10:896297. doi: 10.3389/fcell.2022.896297. eCollection 2022.

LAY SUMMARY

SLC2A5 (GLUT5) knockdown in pancreatic cancer cells reduces fructose uptake and attenuates the IRE1 α /spXBP1-mediated ER stress response, leading to decreased tumor cell proliferation and migration. STAT3 expression and function are diminished in the absence of SLC2A5, with reduced mRNA and protein levels, impaired transcriptional activity, and decreased mitochondrial localization—linking fructose metabolism to key oncogenic signaling. Loss of SLC2A5 disrupts ER-mitochondrial crosstalk, resulting in impaired calcium transfer, increased mitochondrial ROS, and compromised mitochondrial integrity—highlighting a metabolic vulnerability that may be therapeutically exploitable.

Investigating the role of hypoxia in bladder cancer phenotypic plasticity - Poster #456

Presenter: Julia Vassalakis, Queen's University

Other Authors: Andrew Garven, Einav Renert, Hamid Ghaedi, David Berman, Lynne Postovit

ABSTRACT_ID#3222

ABSTRACT

Bladder cancer is a global health concern, particularly as the population ages. Patients with non-muscle-invasive bladder cancer (NMIBC) have a 90% ten-year survival rate, while 10%–30% of cases progress to muscle-invasive disease (MIBC), which has a 70% five-year mortality rate. Bladder cancers are classified into luminal and basal molecular subtypes, with luminal tumours associated with a better prognosis. The transition from NMIBC to MIBC often involves the acquisition of basal-like characteristics. Hypoxia, through mechanisms mediated by the Integrated Stress Response (ISR) and Hypoxia Inducible Factors (HIFs), induces cellular plasticity. Preliminary single-cell RNA sequencing data indicate that luminal precursors give rise to the basal phenotype in bladder cancer, suggesting that phenotypic plasticity is responsible for this change. Analyses of luminal and basal markers (GATA3 and KRT5, respectively) in bladder cancer tissues revealed luminal-to-basal transitions in 19.2% of MIBC samples. These transitions frequently co-localized with the hypoxia marker carbonic anhydrase 9 (CA9), suggesting a connection between dedifferentiation and hypoxia. Subsequent analysis supported the theory that the luminal-to-basal shift is driven by hypoxia-induced phenotypic plasticity, as it demonstrated increased activation of hypoxia response genes in basal cells. We hypothesize that hypoxia-induced ISR facilitates the luminal-to-basal transition in bladder cancer. To test this, we will use SW780 cell line and primary human bladder cancer organoids to analyze for luminal and basal markers. Initial data with SW780 cells in hypoxia showed reduced expression of the luminal markers and a slight increase in the number of spheroids formed under hypoxia.

LAY SUMMARY

Bladder cancer progression often involves the tumour progression from a luminal to a basal subtype. Data from our group suggest that in patients this transition happens and it is colocalized with hypoxic markers. Hypoxia activates the Integrated Stress Response (ISR) inducing mechanisms of plasticity in cancer. We are investigating whether hypoxia creates an environment that facilitates the transition to a more aggressive phenotype in bladder cancer. Our preliminary data in spheroids suggest that hypoxia affects the expression of luminal markers and basal markers. Moreover, the hypoxic response is confirmed to induce the ISR in the SW780 bladder cancer cell line.

The role of DDX41 in stress granules, P-bodies, and myeloid malignancies - Poster #457

Presenter: Harmony Grainger, University of Saskatchewan

ABSTRACT_ID#2369

ABSTRACT

DDX41 is a DEAD-box helicase recurrently mutated in myelodysplastic syndrome (MDS) and acute myeloid leukemia (AML). Stress granules (SGs) and processing bodies (PBs) are membraneless organelles involved in mRNA storage, translation, and decay; their dysfunction has also been linked to MDS and AML. Preliminary data suggest that DDX41 is essential for proper PB formation during cellular stress; however, its role in SG dynamics remains unclear. Given the dynamic interactions between PBs and SGs, we hypothesize that DDX41 is also essential for SG dynamics under stress, and that its dysfunction contributes to the development of MDS and AML. To test this, we will use various cell types: HeLa and HT1080 (WT and DDX41 KO), bone marrow-derived progenitor cells differentiated into macrophages and dendritic cells (WT and DDX41 KO), and AML patient-derived samples harboring the common DDX41 mutation (R525H). SG foci induction by sodium arsenite treatment will be visualized using the G3BP1 marker via immunofluorescence microscopy. Preliminary results show a reduced SG response in DDX41-deficient HeLa cells compared to WT. Future findings will clarify DDX41's role in RNP formation under stress, deepen our understanding of the molecular mechanisms driving MDS and AML progression, and potentially reveal new therapeutic targets for modulating the stress response in cancer.

LAY SUMMARY

DDX41 is a necessary gene whose mutation often leads to cancers such as MDS and AML. We do not yet know the direct molecular pathogenesis between the mutation and disease. Cells form special stress "droplets" to protect themselves from stress in the cells environment. We are studying how DDX41's absence affects the formation/disassembly of these structures. Problems in droplet dynamics is linked to cancer By understanding how DDX41 modulates the cell's stress response, we hope to uncover new ways to treat or prevent blood cancers like MDS and AML. This could lead to therapies that rescue the stress response in cancer cells.

ShcD adaptor protein drives invasion of triple-negative breast cancer cells by aberrant activation of EGFR signaling - Poster #459

Presenter: Hayley Smith, University of Guelph

Other Authors: Hayley Lau, Begüm Alural, Claire Martin, Laura New, Manali Tilak, Sara Banerjee, Hannah Robeson, Nicolas Bisson, Anne-Claude Gingras, Jasmin Lalonde, Nina Jones

ABSTRACT_ID#3228

ABSTRACT

Triple-negative breast cancer (TNBC) is highly metastatic and presents clinical challenges given the lack of targeted therapies. Here we report that the ShcD phosphotyrosine adaptor protein is upregulated in TNBC, and its expression correlates with overall reduced patient survival and decreased response to chemotherapy. In human breast cancer cells, we demonstrate that ShcD expression promotes cell invasion and reduces adhesion, and that these effects are abrogated by mutating the ShcD phosphotyrosine-binding (PTB/SH2) domains. Similarly, in a three-dimensional assembloid model, ShcD-expressing spheroids derived from brain-metastatic TNBC cells show enhanced infiltration into cerebral organoids. Using a proteomic screen for ShcD binding partners in TNBC cells, we identify multiple components of epidermal growth factor receptor (EGFR) signaling and confirm these interactions with wildtype ShcD but not the PTB/SH2 mutant protein. Interestingly, the ShcD interactome correlates with EGFR tyrosine kinase inhibitor resistance, in line with our findings that ShcD overexpression results in hyperphosphorylation of EGFR while ShcD knockout or PTB/SH2 mutation reverts this response. Lastly, pharmacological inhibition of the ShcD PTB domain using indomethacin in TNBC cells decreases EGFR binding and hyperphosphorylation and reduces cell invasion. Altogether, our results identify ShcD as a potential contributor to metastasis in TNBC and provide a molecular basis for clinical targeting of adaptor proteins.

LAY SUMMARY

- ShcD adaptor protein is frequently overexpressed in triple-negative breast cancer (TNBC), a highly metastatic subtype with limited therapeutic options. We show that ShcD expression increases TNBC cell invasiveness in both 2D and 3D contexts, suggesting it may contribute to TNBC metastasis.
- We demonstrate that ShcD binds to and promotes activation of epidermal growth factor receptor (EGFR), a signaling receptor often implicated in cancer progression and development. When this interaction is disrupted by ShcD mutation or pharmacological inhibition, TNBC cells no longer show increased invasion.
- Altogether, our results identify ShcD as a potential contributor to metastasis in TNBC and a prospective therapeutic target.

ULK1-mediated regulation of mitochondrial homeostasis in ovarian cancer spheroids - Poster #460

Presenter: Jack Webb, Western University

Other Authors: Matthew Borrelli, Adrian Buensuceso, Owen Hovey, Shawn S-C Li, Trevor Shepherd

ABSTRACT_ID#2718

ABSTRACT

ULK1 is a serine/threonine kinase best known for initiating autophagy; however, emerging evidence suggests it also plays a broader role in regulating cellular metabolism and mitochondrial quality control. Given that metabolic adaptation is critical for cancer cell survival, particularly in epithelial ovarian cancer (EOC) spheroids that mediate metastatic progression, we aimed to define ULK1's contribution to mitochondrial homeostasis in this context. We generated ULK1 knockout (ULK1KO) cell lines and engineered them to express a mitophagy reporter (mCherry-eGFP-FIS1) to investigate mitochondrial degradation. ULK1 loss enhanced mitophagy in OVCAR8 spheroids but impaired it in HEYA8 and ES2 cells. Notably, mitophagy status correlated with mitochondrial ROS levels—OVCAR8 ULK1KO spheroids exhibited reduced ROS, whereas HEYA8 ULK1KO spheroids showed elevated ROS. Knockdown of ATG5 and ATG7 either promoted degradation (HEYA8, ES2) or had no effect (OVCAR8), suggesting a non-canonical, autophagy-independent role for ULK1 in mitochondrial quality control. To further explore the functional impact of ULK1 loss, we conducted a proteomic analysis, which revealed significant downregulation of oxidative phosphorylation (OXPHOS) and alterations in glycolysis pathways in ULK1KO spheroids. These findings were validated by immunoblotting, confirming decreased expression of OXPHOS complexes and hexokinase 2. Despite these alterations, ATP levels remained stable, indicating possible metabolic compensation. Additionally, proteomic data revealed changes in membrane trafficking and Rho GTPase signaling, suggesting potential alternative routes for mitochondrial degradation. Together, these findings highlight a novel, context-dependent role for ULK1 in regulating mitochondrial homeostasis and metabolic adaptation in ovarian cancer.

LAY SUMMARY

ULK1 helps ovarian cancer cells manage energy and stress: We discovered that ULK1, a protein known for starting the recycling process called autophagy, also plays a key role in how ovarian cancer cells control their energy production and maintain healthy mitochondria—the "powerhouse" of the cell. Different ovarian cancer cells use ULK1 in different ways: In one ovarian cancer cell type, removing ULK1 caused more mitochondria to be broken down, while in others, it reduced this process. This suggests ULK1 may act as either a protector or promoter of mitochondrial breakdown depending on the cell type. Cancer cells adapt to loss of ULK1: Even though removing ULK1 reduced energy-related proteins and pathways, total energy levels stayed the same. This means ovarian cancer cells may rewire their metabolism to survive in the absence of ULK1, possibly using backup energy routes. These findings open up new ways to target cancer cells that rely on ULK1 to manage stress and energy.

NCBP2 as an Oncogenic Driver of Oral Squamous Cell Carcinoma - Poster #461

Presenter: Jackson McLean, University of Calgary

Other Authors: Ayan Chanda, Artee Luchman, Pinaki Bose

ABSTRACT_ID#2873

ABSTRACT

Oral squamous cell carcinoma (OSCC) is the most common head and neck malignancy, accounting for nearly 390,000 cases and 188,000 deaths annually. Patient outcomes remain bleak due to the lack of targeted therapies and a concerning 5-year survival rate of only 50%. The nuclear cap-binding protein subunit 2 (NCBP2) is responsible for mRNA export, transcript stabilization, and other downstream functions. We have identified NCBP2 as an oncogenic driver and prognostic biomarker in OSCC. High NCBP2 expression is associated with worse patient outcomes. Compared to normal tissue, NCBP2 is overexpressed in cancer, highlighting its potential as a therapeutic target. Depletion of NCBP2 decreases OSCC cell migration, proliferation, and invasion. We have also generated the first reported NCBP2 knockout in mammalian cells and established a robust orthotopic oral cancer xenograft model. Based on the results of RNAseq experiments after NCBP2 depletion, we are currently exploring the role of NCBP2 in glycolytic metabolism. Further, we are investigating novel small-molecule inhibitors of NCBP2. Collectively, our in vitro and site-specific in vivo studies have facilitated the characterization of NCBP2 as an oncogenic driver in OSCC and provide a robust foundation for the development of NCBP2-targeted therapies that aim to improve patient outcomes.

LAY SUMMARY

NCBP2 as an Oncogenic Driver in OSCC: Our studies have identified NCBP2 as a key driver of OSCC, high expression promotes tumour growth, progression, and worse outcomes in patients. **NCBP2 Depletion in OSCC:** We have demonstrated that depletion of NCBP2 in OSCC cells reduces cell migration, proliferation, and invasion. These findings highlight NCBP2's critical role in cancer aggressiveness and potential as a therapeutic target. **Establishment of Experimental Models and Future Therapeutic Strategies:** We have established an oral cancer mouse model that allows our research to best recapitulate OSCC, alongside our first-reported mammalian knockout of NCBP2, these models have allowed us to better describe NCBP2's role in OSCC and potential as a therapeutic target. Subsequently, we are also investigating small-molecule inhibitors of NCBP2, with the aim of targeting NCBP2 to improve treatment outcomes in OSCC.

Molecular and Cellular Biology

Sunday, November 2, 2025

Deciphering the Molecular Mechanisms Underscoring Cooperative PI3K and WNT Signaling Pathways in Triple Negative Breast Cancer - Poster #463

Presenter: Junye Che, Lunenfeld-Tanenbaum Research Institute

Other Authors: Elizabeth Rubie, Benjamin Haibe-Kains, Jim Woodgett

ABSTRACT_ID#2454

ABSTRACT

Triple-negative breast cancer (TNBC) is an aggressive subtype characterized by early recurrence and high metastatic potential that is often exacerbated by loss of PTEN. PTEN inactivation activates both the phosphoinositide 3-kinase (PI3K) and Wnt signaling pathways. While PI3K dysregulation is common in breast cancer and Wnt signaling is crucial for mammary stem cell maintenance, the interplay between these two pathways in TNBC progression remains poorly understood. Preliminary work with genetically engineered mouse models demonstrated a synergistic effect of co-activated PI3K and Wnt signaling in mammary tumor development. Clinically, TNBC patients exhibiting co-activation of these pathways have significantly worse survival outcomes. The molecular mechanisms underlying this synergy are currently unknown. My research aims to dissect the intrinsic mechanisms by which PI3K and Wnt pathways cooperate to promote aggressive mammary tumorigenesis. To examine this, I am establishing CRISPR-engineered MCF10A cell lines with individual and combined activating mutations in PI3K and Wnt pathway components. Comparative transcriptomic and proteomic analyses of these cell lines will be conducted to identify differences in signaling landscapes between single and dual pathway activation. Distinctive changes will be tested in cell-line derived xenograft and endogenous mouse models. Uncovering the molecular changes underpinning this synergy may reveal novel therapeutic targets for aggressive TNBC, offering a path to improved patient outcomes.

LAY SUMMARY

Co-activation of PI3K and Wnt signaling drives aggressive mammary tumorigenesis in mouse models and is associated with poor survival in TNBC patients. To identify distinct transcriptomic and proteomic profiles between single oncogenic driver and oncogenic pairs. To reveal potential mediators/targets and facilitate testing of more effective combination therapy with current immunotherapy for aggressive triple negative breast cancer to improve patient outcomes.

Characterizing SP1 Regulation of SAT1 Induction in Response to Doxorubicin Treatment - Poster #465

Presenter: Janice Wu, University of British Columbia

Other Authors: Caroline Liang, Thomas Velenosi

ABSTRACT_ID#3136

ABSTRACT

BACKGROUND Triple-negative breast cancer (TNBC) is a highly aggressive disease that is challenged by drug resistance. Therefore, early identification of chemotherapy effectiveness is important for TNBC prognosis. Doxorubicin (DOX) induces the expression of Spermidine/spermine N¹-acetyltransferase (SAT1), a metabolic enzyme involved in the production of diacetylspermine, which can be detected in urine during effective response to DOX treatment. However, the mechanism behind SAT1 induction is still unknown. Two recently published studies suggest conflicting SAT1 induction and repression mechanisms, respectively, by the transcription factor specificity protein 1 (SP1). **OBJECTIVE** This study aims to characterize the role of SP1 in SAT1 induction following DOX treatment in TNBC. **METHODS** Plasmid constructs containing CRISPR/Cas9 and a SP1-targeting single guide RNA were packaged into lentivirus for lentiviral transduction of the Hs578T and MDA-MB-231 TNBC cell lines. Individual transduced cells were isolated through flow cytometry to culture monoclonal cell populations. Sanger sequencing and western blot were used to identify and validate potential SP1 knockout (KO) clones. **RESULTS** Multiple clones with frameshift-inducing indels in the Hs578T and MDA-MB-231 cell lines were identified. Western blot analysis confirmed the absence of the SP1 protein in all tested KO clones. **CONCLUSIONS** The development of SP1 KO cell lines provides the foundation to explore the role of SP1 on SAT1 induction. Investigating this mechanism may lead to a deeper understanding of urinary diacetylspermine as a biomarker of DOX effectiveness, with potential applications to other cancers and chemotherapies.

LAY SUMMARY

Previous evidence suggests that doxorubicin induces SAT1 expression, but the exact mechanism remains unclear. The transcriptional regulation of SAT1 by Specificity Protein 1 (SP1) is controversial, with studies suggesting that SP1 may be a transcriptional repressor or activator of SAT1 expression. The role of SP1 in doxorubicin mediated SAT1 induction was investigated by deleting SP1 in two triple-negative breast cancer cell lines through CRISPR/Cas9, followed by confirmation of successful knockout through sequencing and protein analysis. Future rescue experiments with modified forms of the SP1 protein will allow us to explore the two proposed mechanisms, which could support better utilization of chemotherapy regimens and prediction of chemotherapy-induced metabolic changes.

Hypoxia-Resistant Lymphatic Endothelial Cells are Sensitive to Lipotoxicity: Implications for Cancer-related Lymphedema - Poster #466

Presenter: Lazina Hossain, University of Alberta

Other Authors: Karina Pereira Gomes, Xiaoyan Yang, Nikki Boerkamp, Spencer B Gibson

ABSTRACT_ID#2668

ABSTRACT

Cancer-related lymphedema (CRL) is a common complication following cancer treatment, characterized by swelling of the arms or legs, inflammation, and fibrosis. The CRL microenvironment contains areas of hypoxia (low oxygen) due to the accumulation of lymph fluid. We found that CRL patients have an increased relative level of plasma saturated fatty acid (SFA), indicating a role in lipotoxicity. Cancer patients also have elevated plasma levels of fatty acid binding protein 4 (FABP4), which transports fatty acids into cells. However, the role of hypoxia, SFA, and FABP4 in the CRL microenvironment remains poorly investigated. In this study, we showed that hypoxia marker, Carbonic anhydrase IX (CA-IX) levels were increased in lymphatic endothelial cells (LECs) in the lymphedema tail model under a high saturated fat diet (HSFD). We also found increased hypoxic LECs (CAIX+) undergoing apoptotic cell death, measured by the TUNEL assay, when mice were fed with an HSFD compared to the normal diet. In addition, human dermal lymphatic endothelial cells (HDLEC) under hypoxia (1% oxygen) showed increased reactive oxygen species (ROS) and cell death following stearic acid treatment, the SFA, which is high in HSFD. To determine the role of FABP4, HDLEC cells were treated with a pharmacological inhibitor of FABP4 under hypoxia in the presence or absence of stearic acid. The results showed that the FABP4 inhibitor reduced hypoxia-induced cell death in the presence of stearic acid. Overall, this indicates a role for SFA lipotoxicity in the CRL microenvironment.

LAY SUMMARY

Cancer-related lymphedema (CRL) is a debilitating disease characterized by swelling of the arms and legs due to damage to the lymphatic vessels. Cancer treatment, such as surgery, chemotherapy, and radiation therapy, causes damage to the lymphatic vessels. But not all cancer patients develop CRL, indicating the role of microenvironmental factors in CRL progression. In CRL, the fluid accumulation creates areas with low oxygen levels (hypoxia) in the affected tissues. In our preliminary data, we observed that CRL patients often have higher levels of saturated fatty acids (SFAs) in their blood, which can be harmful to cells, a condition known as lipotoxicity. Additionally, these patients show increased levels of a protein called fatty acid binding protein 4 (FABP4), which helps transport fatty acids into cells. We found that mice fed a high saturated fat diet show prolonged lymphedema. The lymphedema tissue is hypoxic and more prone to cell death. Similarly, human lymphatic cells exposed to low oxygen and treated with stearic acid (a type of SFA) showed increased stress and cell death. However, using the FABP4 inhibitor reduces cell death. This suggests that SFAs and FABP4 contribute to cell damage in CRL, and targeting FABP4 might offer a new therapeutic approach.

Identifying Novel Modifiers of EGFR Induced Tumourigenesis - Poster #467

Presenter: Jana Jajarmi, BC Cancer Research Centre

Other Authors: Amy Nagelberg, Raj Chari, Lok-In Josephine Ma, William Lockwood

ABSTRACT_ID#2893

ABSTRACT

Lung cancer is the deadliest form of cancer worldwide, and accounts for 24% of all cancer deaths in Canada. The most common subtype of lung cancer is lung adenocarcinoma (LUAD) which is frequently driven by activating mutations in epidermal growth factor receptor (EGFR). While the development of targeted therapies against mutant EGFR (mEGFR) such as tyrosine kinase inhibitors (TKIs) has significantly improved patient outcomes, they are not curative and acquired resistance remains a major challenge. It is therefore essential to identify other genes that cooperate with mEGFR in driving LUAD tumourigenesis so new therapies can be developed. Our lab previously utilized an in vivo Sleeping Beauty (SB) insertional mutagenesis screen to identify such genes, and comparison with human LUAD data from TCGA produced a list of 385 candidate genes mutated in both mouse and human tumours. We then validated these using a targeted CRISPR screen in mEGFR NIH-3T3 cells under multiple conditions including in the presence of clinically used TKI. After stringent analysis we have identified 5 candidate oncogenes with available small molecule inhibitors for further study implicated in driving tumourigenesis, TKI resistance, or both. The results of this work will greatly expand our understanding of mEGFR driven LUAD tumourigenesis, progression, and drug resistance and lay the groundwork for future therapies against this devastating disease.

LAY SUMMARY

- Mutations in the gene epidermal growth factor receptor (EGFR) are very common in lung cancer. This type of cancer is treated using drugs called tyrosine kinase inhibitors (TKIs), but patients become resistant very quickly so we need to find other ways to treat them.
- Our lab has used a combination of genetic approaches to identify other genes that when mutated, can work with mutant EGFR to drive lung cancer formation and progression. We have narrowed down our target list to 5 potential genes that may be related to tumour formation, TKI resistance, or both. Importantly these targets have drugs that shut them down already developed, so the timeframe from research to clinical use is shorter.
- This work will enhance our understanding of mutant EGFR driven lung cancer and TKI resistance, and lay an important foundation for future therapies.

Elucidating a novel role for the Bcl-2 family member BAD in breast cancer metastasis - Poster #468

Presenter: Leila Pirayeshfard, University of Alberta

Other Authors: John Maringa Githaka, Shu Luo, Han Huang, Arashdeep Saini, Nicolas Touret, Olivier Julien, Ing Swie Goping

ABSTRACT_ID#2642

ABSTRACT

Breast cancer is the most diagnosed cancer in women and metastasis remains the leading cause of mortality. Understanding the molecular mechanisms underlying metastasis will provide tools for improving clinical outcomes. Our lab identified the BCL2 family protein BAD as a novel regulator of metastasis and a prognostic marker for breast cancer patient survival. BAD is regulated by phosphorylation, and using a genetic mouse model wherein the three serine sites are mutated to alanine (BAD3SA), we demonstrated that BAD3SA inhibits mammary epithelial cell migration in both mammary gland development and breast cancer metastasis. BAD3SA only inhibits cell migration in 3D-culture but not in 2D, suggesting regulation of 3D-specific cell motility processes. To gain molecular insight, we used BioID to identify BAD3SA-binding proteins in 2D and 3D culture by mass spectrometry. Reactome analysis of BAD3SA 3D hits identified significant pathways including Rho GTPase and Integrin cell surface interactions. Importantly, the integrin family protein, ITGB1, was the most enriched BAD3SA interactor. Using proximity ligation assay, we validated BAD3SA:ITGB1 interactions. ITGB1 is a cell surface receptor crucial for cell-cell and cell-extracellular matrix adhesion and motility by modulating adaptor proteins, signaling molecules and cytoskeletal components. Notably, BAD3SA diminished actin and tubulin clustering, consistent with impairment of ITGB1-mediated downstream pathways. For functional validation, we will disrupt BAD3SA:ITGB1 interactions using BAD3SA deletion constructs and assess rescue of the invasion defect. Our data suggest that BAD3SA blocks 3D migration/invasion by interacting with ITGB1. Thus, we have uncovered a new regulatory mechanism of cell invasion in breast cancer metastasis.

LAY SUMMARY

Metastasis is the leading cause of death from breast cancer. Understanding what causes cancer cells to migrate is crucial, as it could lead to prevent metastasis by targeting specific molecules involved in the process. Our lab has identified a protein called BAD as a new regulator of cell migration and metastasis. BAD exists in two forms: phosphorylated and non-phosphorylated. We found that the non-phosphorylated form, called BAD3SA, can inhibit cancer cell migration, though the exact mechanism is still unclear. Our research focuses on understanding how non-phosphorylated BAD achieves this by examining its location within the cell and identifying other proteins it interacts with. We discovered that BAD3SA interacts with Integrin beta-1 (ITGB1), a protein on the cell surface that helps cells attach and move through tissues. This interaction appears to disrupt key structures inside the cell, such as actin and tubulin, which are critical for cell movement. These findings suggest that BAD3SA may block cancer cells spread by interfering with ITGB1 signaling. We aim to further explore how this interaction works to help identify new therapeutic targets for preventing metastasis.

Dysregulated RAP1GAP expression is associated with poor prognosis in TNBC - Poster #469

Presenter: Tobias Karakach, Dalhousie University

Other Authors: Karla Valenzuela, Jasmine Barra, Dean Chamberlain, Evelyn Teh, Barbara Karten

ABSTRACT_ID#3214

ABSTRACT

Triple-negative breast cancer (TNBC) accounts for ~15% of invasive breast cancers and is marked by high aggressiveness and poor event-free survival. Despite extensive research, Ras family small GTPases remain underexplored as biomarkers for TNBC, although they are key regulators of cell signaling and frequently dysregulated in cancer. One such regulator, RAP1GAP, is a GTPase-activating protein for Rap1 and is known to function as a tumor suppressor in several epithelial cancers. RAP1GAP is often downregulated through promoter hypermethylation, LOH, or proteasomal degradation, leading to constitutive Rap1 activation and increased oncogenic potential. We investigated the role of RAP1GAP in TNBC progression using the MDA-MB-231 cell line. Lentiviral shRNA was used to silence RAP1GAP, and a recombinant construct enabled its overexpression. Cell proliferation and migration were measured using CCK-8 and scratch assays, respectively. Knockdown of RAP1GAP enhanced both processes, while its overexpression suppressed them. To explore the underlying molecular mechanisms, we performed tandem mass tag (TMT)-based quantitative total proteomics and phosphoproteomics with synchronous precursor selection-MS3. Total proteome analysis confirmed that RAP1GAP regulates protein expression. Gene enrichment of the phosphoproteome revealed key alterations in pathways related to cytoskeletal remodeling and transcriptional regulation in response to RAP1GAP modulation. Together, these results demonstrate that RAP1GAP functions as a molecular switch influencing TNBC cell proliferation and migration, likely via MAPK-mediated cytoskeletal reorganization. Our findings suggest that RAP1GAP downregulation contributes to TNBC progression and may serve as a potential biomarker or therapeutic target in this aggressive cancer subtype.

LAY SUMMARY

Triple-negative breast cancer (TNBC) is a fast-growing and hard-to-treat type of breast cancer with very poor outcomes. Scientists have studied TNBC for many years, but one group of important proteins called GTPases has not received much attention even though these proteins act like switches that turn signals on and off. One of them, called RAP1GAP, helps control another protein named Rap1, which is linked to cancer growth. RAP1GAP slows down cancer by turning off Rap1, but when its levels are low, Rap1 stays active and can make cancer worse. We wanted to know if RAP1GAP plays a similar role in TNBC used a cell line in the lab. We decreased the amount of RAP1GAP in some cells and increased it in others. Our study shows that RAP1GAP helps slow down the growth and movement of TNBC cell. When it is missing, the cancer becomes more aggressive We found that when RAP1GAP was low, the cancer cells grew and moved faster. When we added more RAP1GAP, the cells slowed down. We also studied the proteins inside these cells and found that RAP1GAP affected proteins involved in how cells move and grow. This means RAP1GAP may help control how TNBC spreads.

Improving efficacy of PARP inhibition by targeting ribosome biogenesis in triple-negative breast cancer - Poster #470

Presenter: Mallory I. Frederick, Centre de recherche du CHUM

Other Authors: Mallory I Frederick, Nicolas Pouderous, Kenan Aloss, Shufeng Zhou, Gerardo Ferbeyre, Saima Hassan

ABSTRACT_ID#2338

ABSTRACT

Breast cancer is the most common cancer in women and a significant contributor to cancer-related mortality. Poly (ADP-Ribose) Polymerase inhibitors (PARPi), such as olaparib, target PARP-1, demonstrating success in patients with DNA repair gene mutations due to synergistically lethal effects. However, PARP-1 activity is not limited to DNA repair. Approximately 40% of PARP-1 molecules reside in the nucleolus, and PARP-1 is involved in regulation of Pol I transcription and shuttling of proteins between the nucleolus and nucleus. Thus, targeting PARP-1 also impacts ribosome biogenesis. Ribosomal proteins are enriched in triple-negative breast cancer (TNBC), an aggressive subtype of breast cancer. We measured sensitivity to a PARPi, olaparib, and two Pol I inhibitors, CX-5461 and BMH-21, in TNBC cell lines. The combination of olaparib with either Pol I inhibitor was synergistic in three PARPi-resistant cell lines, BT549, Hs578T, and MDAMB231. Using immunofluorescence, we showed that in MDAMB231 cells, the combination approach increased induction of DNA damage, indicated by increased percentage of γ -H2AX-positive cells, along with nucleolar stress, shown by increased RSL1D1 and translocation of nucleophosmin from the nucleolus to the nucleoplasm. Similarly, the combination increased senescence, demonstrated by increased SA-B-gal staining. Finally, in a recovery assay, we found significant reduction of growth in cells treated with the combination of olaparib and CX-5461, up to 20 days after drug removal. Together, these data show the potential for targeting ribosome biogenesis in combination with PARPi in TNBC.

LAY SUMMARY

Triple-negative breast cancer (TNBC) is an aggressive form of breast cancer. PARP inhibitors are used in TNBC to target the protein PARP-1, which acts in the part of the nucleus where ribosomes are assembled. Combining ribosome biogenesis inhibitors with PARP inhibitors increased the ability of PARP inhibitors to kill TNBC cells by causing DNA damage. The combination was also linked to senescence, a phenotype in which cells stop replicating to avoid specific types of stress. TNBC pre-treated with the combination of PARP inhibitors and ribosome biogenesis inhibitors were unable to recover from the treatment in a 20-day recovery assay.

Molecular and Cellular Biology

Monday, November 3, 2025

Targeting the Mitochondria in Pediatric Early T-cell Precursor Acute Lymphoblastic Leukemia - Poster #472

Presenter: Marissa Rashkovan, CHU Sainte Justine

Other Authors: Juliette Durocher

ABSTRACT_ID#2463

ABSTRACT

T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive hematologic malignancy, accounting for 10-15% of pediatric ALL cases. A distinct subgroup of immature T-ALL, Early T-cell Precursor ALL (ETP-ALL), is associated with early relapse and poor prognosis. ETP-ALLs are difficult to treat, due to the increased failure of early-stage induction chemotherapy. Furthermore, there are currently few approved therapies for patients who relapse. Thus, better leukemia-specific therapies are needed to treat ETP-ALL in particular. We have found that pediatric ETP-ALL is particularly dependent on the essential mitochondrial protein MFN2, and that loss or inhibition of MFN2 leads to leukemia cell death. This observation led us to hypothesize that ETP-ALL rely upon mitochondrial function for survival and proliferation, and that MFN2 is integral to coordinate mitochondrial dynamics. The inhibition of MFN2 could therefore act as a novel target for the treatment of pediatric ETP-ALL. Here, we define the genes, cellular processes and pathways regulated by MFN2 in ETP-ALL, and determine the clinical relevance of MFN2 inhibition. We believe that targeting mitochondrial dynamics has the potential to bring novel therapies to the clinic, to treat pediatric patients with relapsed or refractory ETP-ALL who have no other recourse.

LAY SUMMARY

MFN2 is essential for pediatric ETP-ALL cell survival and MFN2 inhibition induces many molecular and cellular changes within ETP-ALL cells, leading to cell death. Targeting mitochondrial dynamics may be a viable pharmacologic strategy to treat ETP-ALL.

FOXO1 Promotes Dermal Fibrosis via Smad Activation Independent of PI3K/Akt Signaling - Poster #473

Presenter: Liliane Kreuder, University Health Network

Other Authors: Pierre-Antoine Bissey, Kenneth W. Yip, Fei-Fei Liu

ABSTRACT_ID#2635

ABSTRACT

Introduction: Radiation-induced fibrosis (RF) continues to be one of the most challenging long-term side effects experienced by cancer patients treated with radiation therapy. While previous studies have linked Forkhead Box O1 (FOXO1) to a variety of different fibrotic diseases, its role in RF has yet to be thoroughly investigated. In this study, we aim to investigate the Akt and Smad upstream pathways in regulating FOXO1 activity in an in vitro RF model. **Methods:** Collagen expression as a marker for fibrosis was investigated in human primary dermal fibroblasts (hPDFs) using western blot analysis of whole cell lysates and RT-qPCR for gene expression. Temporal changes in FOXO1 expression following TGF- β 1 treatment were analyzed using western blot, alongside evaluation of Akt and Smad pathway activation. FOXO1 knockdown was performed using both siRNA and shRNA approaches to investigate its role in fibrotic signaling. **Results:** FOXO1 protein levels increased one hour after TGF- β 1 treatment. The presence of phosphorylated-Smad2 upon TGFB1 treatment confirmed the activation of Smad pathway in our model. In contrast, Akt remained inactive by TGF- β 1 treatment. Loss-of-function experiments targeting FOXO1 resulted in an increase in collagen. **Conclusion:** Our data indicate that TGFB1 activates FOXO1 via Smad pathway rather than Akt. Increased collagen level following FOXO1 knockdown suggests that FOXO1 may act as a negative regulator of collagen synthesis. Future experiments will delve into understanding the mechanisms governing FOXO1-Smad signaling pathways and identifying downstream targets through which FOXO1 modulates collagen expression.

LAY SUMMARY

- TGF- β 1 treatment led to phosphorylation of Smad2 but did not activate Akt, suggesting FOXO1 is regulated via the Smad-dependent pathway in this model of radiation-induced fibrosis.
- Knockdown of FOXO1 (via siRNA/shRNA) resulted in increased collagen levels, indicating that FOXO1 likely functions as a negative regulator of collagen synthesis.
- Since loss of FOXO1 enhances fibrotic signaling (collagen upregulation), the data suggest that FOXO1 may act as an anti-fibrotic or protective factor in the context of radiation-induced fibrosis.

Molecular and Cellular Biology

Monday, November 3, 2025

Synthetic dosage lethal relationships between hTERT and nucleolus associated proteins - Poster #474

Presenter: Mary Lazell-Wright, University of Saskatchewan

ABSTRACT_ID#3274

ABSTRACT

While individual cancers display heterogeneity and diversity, 85% of these cancers share a common mechanism that allows them to maintain immortality; the use of the enzyme hTERT for the continual maintenance of telomeres. Although directly targeting hTERT has been shown to be ineffective in the past, we hypothesize that the indirect targeting of hTERT via a synthetic dosage lethal (SDL) approach will allow for the selective killing of these high hTERT cancers. The SDL approach is a method that allows for the indirect targeting of an overexpressed trait, via a genetic dependency not found in those cells that do not harbour the overexpression of the trait, therefore having minimal off target effects. The Vizeacoumar lab hypothesized that certain nucleolus associated proteins will have SDL relationships with hTERT overexpression. This relationship was explored via genetic inhibitory means, as well as using inhibitory drugs. Through these assays, the nucleolus associated protein(s) that exhibit an SDL relationship with hTERT have been determined, and further steps are being taken to elucidate the dependency mechanism.

DHODH connects de novo pyrimidine synthesis and mitochondrial activity in prostate cancer cells - Poster #476

Presenter: Maxime Labroy, Centre de recherche du CHU de Québec - Université Laval

Other Authors: Marc-Olivier Paré, Line Berthiaume, Mélissa Thomas, Martin Pelletier, Frédéric Pouliot, Jean-Yves Masson, Etienne Audet-Walsh

ABSTRACT_ID#2597

ABSTRACT

Prostate cancer (PCa) remains the most commonly diagnosed cancer in men in Canada. The cornerstone clinical therapy is to inhibit the androgen receptor (AR) signalling pathway. Even if AR inhibition is successful in the early stages of the disease, tumours will eventually develop treatment resistance and developing new therapeutic strategies is necessary to enhance patient care. To discover new anti-metabolic approaches, we performed a high-throughput anti-metabolic screening of 1.280 compounds in PC3 cells, an AR-negative human PCa cell line. Five inhibitors of the dihydroorotate dehydrogenase (DHODH) enzyme decreased proliferation by 60%. Results were validated in a second AR-negative cell line, DU145, along with the neuroendocrine PCa model LASCPC-01, revealing the inhibition of DHODH as a potential new therapeutic approach for PCa cells resistant to AR inhibition. DHODH catalyzes the fourth reaction of the de novo pyrimidine synthesis pathway and is involved in nucleotide synthesis. It is also the sole mitochondrial enzyme of this pathway, connecting nucleotide synthesis to mitochondrial activity. In respiration assays, the inhibition of DHODH was shown to decrease oxygen consumption rates and disrupt the activity of the mitochondrial respiratory chain. Metabolomics analyses further demonstrated a reprogramming of the TCA cycle, notably altering fumarate levels by more than 10-fold. Finally, DHODH inhibitors were tested in patient-derived organoid models, showing a strong blockade of growth in these models. Taken together, these results highlight the DHODH as a new potential therapeutic target in AR-negative PCa, linking both de novo pyrimidine biosynthesis and the TCA cycle activities to sustain cell proliferation.

LAY SUMMARY

Prostate cancer (PCa) is originally treated by anti-androgenic therapies. Tumors can develop a resistance to those treatments, leading to castration-resistant prostate cancer (CRPC). CRPC is the lethal form of PCa and remains without efficient therapy. We performed an anti-metabolic high-throughput screening on a PCa model and revealed the Dihydroorotate Dehydrogenase (DHODH) inhibition as a new potential therapy. DHODH is the unique mitochondrial enzyme of the de novo pyrimidine synthesis and can also interact with the mitochondrial respiratory chain to produce energy. DHODH inhibition blocks tumoral growth in in vitro CRPC models. The neuroendocrine prostate cancer model is particularly sensitive to DHODH inhibition.. Inhibition of DHODH leads to a metabolic reprogramming and a connection between the TCA and urea cycles and de novo pyrimidine synthesis.

Investigating the role of intermittent hypoxia on breast cancer metastasis - Poster #477

Presenter: Laura Paulus, BC Cancer Research Centre

Other Authors: Kevin Bennewith

ABSTRACT_ID#2927

ABSTRACT

Introduction Breast cancer is the most common malignancy in Canadian women, with one in 36 Canadian women projected to die from the disease. This mortality is due to development of metastatic disease. Many solid tumours, such as breast cancers, experience abnormally low oxygen in the tumour microenvironment (TME), known as hypoxia. Hypoxia in the TME elicits molecular responses that promote tumour cell survival and metastasis. As the tumour grows, aberrant and improperly formed vasculature may result in transiently compromised oxygen delivery to tumour cells, causing intermittent hypoxia. Under in vitro conditions, chronic hypoxia was shown to promote the pro-metastatic amoeboid migration type in tumour cells. We hypothesize that intermittent hypoxia will accelerate breast cancer cell migration and invasion through a 3-dimensional matrix, with a greater proportion of tumour cells adopting the amoeboid-like phenotype in a tumour spheroid. **Objectives** To improve our understanding of how intermittent hypoxia in tumours can promote metastasis. **Methods** We developed a spheroid-based assay to assess whether intermittent hypoxia induces a more metastatic phenotype in tumour cells. Tumour spheroids are formed from murine breast cancer cells and subsequently embedded in a collagen matrix prior to being incubated under intermittent hypoxic conditions. The area and distance of migration away from the initial spheroid, as well as the type of migration, is assessed by microscopy and quantified. **Conclusions** Intermittent hypoxia is a common feature of the solid TME and is often linked to metastasis and cancer recurrence, as well as resistance to treatments such as chemotherapy and radiation.

LAY SUMMARY

We have developed a 3-dimensional tumour spheroid assay to assess differences in tumour cell invasion through a collagen matrix with a range of oxygen conditions. Cycling oxygen concentrations in tumour spheroids between 1% and 5% O₂ has distinct effects on cancer cell migration and invasion through collagen, as compared to physiological oxygen concentrations. Our results imply a link that we aim to further explore between transiently compromised oxygen delivery to tumour cells in the tumour microenvironment and the metastatic capabilities of breast cancer cells.

KSHV kaposin RNA employs a dual mechanism to disassemble p-bodies which may contribute to viral tumourigenesis - Poster #478

Presenter: Maxwell P. Bui-Marinis, University of Calgary

Other Authors: Mariel Kleer, Jennifer A. Corcoran

ABSTRACT_ID#2472

ABSTRACT

Kaposi's sarcoma-associated herpesvirus (KSHV) causes the endothelial malignancy, Kaposi's sarcoma (KS). Latent KSHV dominates in KS suggesting that viral genes expressed in latency promote tumours. Kaposin, a GC-rich, repetitive transcript, encodes for three proteins and is abundant in latency. In prior work, overexpression of the kaposin protein, KapB, elicited the disassembly of processing (p-)bodies, cytoplasmic granules that repress or decay inflammatory and tumourigenic mRNAs. However, we recently showed that kaposin is a nuclear-retained non-coding RNA during infection and overexpression, making it largely unavailable for translation. This creates a new question; since both KapB protein and kaposin transcript are present after overexpression, is p-body loss elicited by KapB (protein) or kaposin (RNA)? We created three kaposin constructs to distinguish between RNA- and protein-mediated functions: authentic KapB, 'recoded' KapB (makes authentic protein from alternatively coded RNA), and 'nostarts' KapB (makes authentic RNA, fails to translate protein). As not all constructs produce KapB, protein-based techniques cannot validate expression. To address this, I developed a TaqMan assay and determined kaposin transcript copy number on a per cell basis. Overexpression of each construct followed by immunostaining of p-bodies and RNA-FISH for kaposin showed that both KapB protein and kaposin RNA elicit p-body disassembly. Using a CellProfiler pipeline to filter kaposin-positive cells, preliminary analysis suggests that kaposin elicits p-body disassembly in a cell autonomous manner while KapB induces p-body loss through a paracrine mechanism. This work unveils that kaposin RNA uses a dual mechanism to disassemble p-bodies, contributing to KSHV-mediated inflammation and tumourigenesis.

LAY SUMMARY

Kaposi's sarcoma-associated herpesvirus (KSHV) encodes for the kaposin gene locus. The resulting kaposin transcript, which is highly expressed in KS tumours, is a bifunctional RNA that I show is capable of eliciting p-body disassembly at the RNA- and protein-level. P-bodies are cytoplasmic granules that repress or decay inflammatory and tumourigenic mRNAs; disassembly of p-bodies makes these mRNAs available for translation. We recently found that kaposin is nuclear-retained and unavailable for translation during overexpression and infection. Here, I show that cells with nuclear-retained kaposin display an enhanced ability to disassemble p-bodies. This suggests that kaposin may elicit p-body disassembly in a cell autonomous manner, whereas the kaposin protein may induce p-body loss through paracrine signalling. This work unveils that kaposin RNA uses a dual mechanism (through RNA and protein) to disassemble p-bodies which may contribute to KSHV-mediated inflammation and tumourigenesis.

Molecular and Cellular Biology

Sunday, November 2, 2025

Elucidating the Role of the Insulin Receptor Isoforms in Pancreatic Ductal Adenocarcinoma - Poster #479

Presenter: Lan Valerie Tao, BC Cancer Research Institute

Other Authors: James Topham, Joanna Karasinska, Michael Lee, Erica Tsang, Andrew Metcalfe, Hassan Ali, Dawn Ashforth, Rachel Goodwin, Patricia Tang, Oliver Bathe, Janessa Laskin, Marco Marra, Daniel Renouf, David Schaeffer

ABSTRACT_ID#2746

ABSTRACT

Pancreatic ductal adenocarcinoma (PDAC) is associated with a dismal five-year survival rate of 13%. The identification of new predictive and prognostic biomarkers is essential to address the heterogeneity of PDAC and enhance therapeutic outcomes for patients. Hyperinsulinemia, an abnormal condition resulting in high levels of circulating insulin in the body, is both a risk factor and cancer-induced phenomenon associated with PDAC that may contribute to the dysregulation of the insulin receptor gene, INSR. Two isoforms of INSR contribute to its expression: IR-A and IR-B. IR-A has been documented as the oncogenic isoform that may play a greater proliferative role, while IR-B may contribute predominantly to cellular metabolism. However, the exact distinction between the two isoforms across different cancer types remains unclear, which suggests that their function may be tissue- and cancer-dependent. Through whole genome and transcriptome sequencing of 92 PDAC patient tumours from both PanGen (NCT01855477) and Personalized Oncogenomics (NCT02155621) trials, we demonstrated that about 20% of patient tumours contain a higher proportion of IR-A than IR-B isoforms, while 39% have a heterozygous loss of INSR that increases the IR-A:IR-B ratio. PurlST classical subtype tumours also have higher INSR and IR-A expression. Gene set enrichment analysis reveals that IR-A correlates with genes related to serine peptidase inhibitor activity, while IR-B correlates with various metabolic pathways. In vitro studies are underway to investigate IR-A and IR-B levels in PDAC cell lines and patient-derived organoids, while a stable overexpression model of the isoforms is being established to assess proliferation, signaling and metabolism.

LAY SUMMARY

Using the genetic information from 92 pancreatic cancer patient tumours, we have conducted a study to better understand the role of the insulin receptor and its two forms, IR-A and IR-B. IR-A and IR-B are associated with distinct metabolic roles in pancreatic cancer.

The COPII secretory complex protein SEC23B governs MDA-MB-231 breast cancer cell migration by regulating collagen I processing - Poster #480

Presenter: Michael Olson, Toronto Metropolitan University

Other Authors: Emily Joo, Jonathan St-Germain, Audrey Astori, Brian Raught

ABSTRACT_ID#2772

ABSTRACT

Cell migration requires dynamic reorganization of the actin-myosin cytoskeleton. To characterize how the initiation of cell migration affects the actin-associated proteome, we developed a robust method that combines the mini-Turbo version of the promiscuous biotin ligase BirA with the 17 amino acid actin-binding peptide LifeAct (MiniBioID-LifeAct) under the transcriptional control of a doxycycline promoter. To validate this system, MDA MB 231 human breast cancer cells expressing MiniBioID-LifeAct were treated with cytochalasin D to inhibit actin polymerization, followed by a biotin pulse, and differences in protein biotinylation were identified by mass spectrometry. Biotin labelling of well-characterized F-actin binding proteins was reduced, while biotinylation of G-actin proteins was increased. To determine changes in actin-cytoskeleton proximal protein complexes in the early stages of cell migration, MiniBioID-LifeAct expressing cells were pulsed with biotin after making scratch wounds in monolayers to induce actin polymerization and coordinated cell motility. Consistent with the cytochalasin D results, migrating cells had increased biotin labelling of several F-actin binding proteins. Intriguingly, a component of the COPII secretion complex SEC23B was more extensively biotinylated by MiniBioID-LifeAct following scratch wounding. SEC23B knockdown revealed a role in promoting efficient migration by governing the processing of collagen I protein. These findings indicate a previously unknown role for SEC23B in regulating extracellular matrix organization to enable cell movement.

LAY SUMMARY

Cell migration induces changes in actin-associated protein complexes. The COPII core component SEC23B is re-distributed during cell migration. Post-translational collagen processing is dependent on SEC23B to enable efficient cancer cell migration.

Harnessing a marine worm hemoglobin potential to enhance tumor biobanks preservation and 3D living avatars viability for personalized cancer research - Poster #481

Presenter: Maëlle Cottin, Centre de recherche du centre hospitalier de l'université de Montréal (CRCHUM)

Other Authors: Samuel Bourdages, Alexandre Bernèche, Guillaume Cardin, Apostolos Christopoulos, Thomas Gervais, Franck Zal, François Yu, Francis Rodier

ABSTRACT_ID#2547

ABSTRACT

Tumour biobanks have been used for decades and play a crucial role in translational biomedical research. However, their quality can be compromised by conditions of ischaemia and hypoxia, affecting tissue integrity, representativeness and viability. Hemarina, a pharmaceutical laboratory, is studying the oxygenating properties of haemoglobin from a marine worm (M101). This non-immunogenic extracellular haemoglobin can transport up to 154 oxygen molecules. Our hypothesis is that the addition of M101 during standardized bio-banking protocols could improve the representativeness, conservation and viability of samples. The first objective is to optimise hypoxic culture conditions in order to assess the oxygenating properties of M101 in vitro. The second objective is to analyse the properties of M101 on the conservation and stabilisation of non-living bio-banks. In the final objective, we will analyse the properties of M101 on increasing the viability and representativeness of living 3D avatars using micro-fluidic devices. We have demonstrated that M101 has significant oxygenating and anti-inflammatory properties in vitro on Pc3 cells. Preservation of PC3 xenografts in a solution supplemented with M101 significantly reduced the expansion of hypoxia and tissue metabolic stress without causing oxidative stress. Finally, the addition of M101 into MDTs culture chips improved their structure, stabilize their size and increase their proliferation over-time. In conclusion, M101 demonstrates significant oxygenating potential in vitro and ex-vivo, supporting its strong potential for the preservation of tumour samples. Its widespread use could revolutionize sample preservation and live avatar models, opening up new avenues for oncology research and personalized medicine.

LAY SUMMARY

M101, a special haemoglobin from marine worms, can deliver much more oxygen than human haemoglobin, and adding it to tumour biobanking protocols helps keep tissue samples healthier and more representative during the pre-analytical period. During our experiments we showed that M101 not only significantly reduce hypoxia and metabolic stress in preserved tumor samples, but also improves 3D tumor models grown on microfluidic chips structural stability and increases cell proliferation over time, making these models more reliable for research. By enhancing both preserved tissue samples and live tumor avatars, the use of M101 could greatly improve how tumour samples are preserved and studied, making research more reliable and opening up new possibilities for personalized cancer treatments.

High Throughput Drug Screen Reveals Synthetic Lethal Targets with KRAS in Intrinsically Resistant KRAS Non-Small-Cell Lung Cancer - Poster #482

Presenter: Mutian Wang, University Health Network

Other Authors: Nikolina Radulovich, Zhijuan Luo, Ming-Sound Tsao

ABSTRACT_ID#2415

ABSTRACT

Introduction: KRAS G12C mutations accounts for 13% of non-small cell lung cancers (NSCLC). Although sotorasib is the only KRAS G12C-specific inhibitor approved in Canada, it elicits responses in only approximately 37% of patients; the remaining 60% exhibit intrinsic resistance. Exploiting synthetic lethality, in which simultaneous inhibition of two targets is lethal but inhibiting either one alone is not, could enable combination therapies to overcome this resistance. Methods: Four patient-derived KRAS G12C organoid models showing intrinsic resistance were treated $\pm$ 1 μ M sotorasib and screened against the Ontario Institute for Cancer Research's kinase-inhibitor and toolkit libraries. Hits were defined as genes whose co-inhibition with KRAS reduced viability below 50%, whereas single-gene inhibition retained $>$ 50% viability. Results: Across all models, overlapping targets Clustered RTK, the cell-regulation and mitosis, and the MAPK families. JAK2 stood out among these shared targets, as the JAK/STAT pathway, which has been linked to both cancer progression and MAPK activation. We hypothesize that JAK/STAT mediates resistance by bypassing KRAS to reactivate MAPK pathway and by upregulating Mcl-1, an anti-apoptotic gene. NSCLC patients often exhibit elevated Mcl-1 levels, which was associated with poorer prognosis; MCL1 is a commonly essential gene in cancer. In three of four organoids, combined KRASG12C and Mcl-1 inhibition yielded significant synergistic cytotoxicity; single-agent inhibition of either target had negligible effect. One organoid was uniquely dependent on Mcl-1 and responded robustly to Mcl-1 inhibition alone. Conclusion: Our findings support the development of a KRAS + Mcl-1 co-inhibition strategy as a treatment avenue in resistant NSCLC.

LAY SUMMARY

A large percentage of patients with KRAS-driven non-small cell lung cancer do not respond to KRAS-targeted drugs like sotorasib. In most of our resistant tumor organoids, inhibiting the antiapoptotic protein Mcl-1 alone has no effect, but when both Mcl-1 and KRAS are inhibited in combination, a large percentage of cancer cells die. This suggests that these resistant tumors rely on Mcl-1 for survival when KRAS is inhibited and that shutting down Mcl-1 restores the effectiveness of sotorasib. These results point toward a new combination treatment strategy: using sotorasib together with an Mcl-1 inhibitor may overcome resistance in patients who do not respond to sotorasib. The literature also suggests that the JAK/STAT pathway can promote Mcl-1 expression. Our initial discovery screen revealed JAK2 as a possible synthetically lethal target with KRAS. Testing whether blocking JAK/STAT reduces Mcl-1 could identify additional targets to help disable the tumor's resistance.

BlindfoldING the HDAC1/Sin3A complex: ING1 antagonizes LBH589-mediated invasion in breast cancer in an integrin-dependent manner - Poster #483

Presenter: Mahbod Djamshidi, Arnie Charbonneau Cancer Institute, Cumming School of Medicine, University of Calgary

Other Authors: Katayoun Heshmatzad, Ryota Tanida, Karl Riabowol

ABSTRACT_ID#2349

ABSTRACT

The pan-histone deacetylase inhibitor (pan-HDACi) LBH589 is FDA-approved for the treatment of several hematological cancers and some solid cancers including triple-negative breast cancer (TNBC). TNBCs respond poorly to both standard cancer therapies like chemotherapy and novel approaches like immune checkpoint inhibitors. Although clinical trials investigating the effect of LBH589 on TNBC progression have been promising, our recent data suggest that while higher concentrations of LBH589 induce cellular mortality, cancer cells that avoid death increase their motility and invasion potential in response to LBH589 in an EMT hallmark-independent manner. The HDAC1/Sin3A/ING1 complex is frequently downregulated in TNBCs. It is normally targeted to the promoter-adjacent histone mark, trimethylated lysine 4 of histone H3 (H3K4me3). This precise targeting is largely dependent on the INhibitor of Growth proteins ING1 & ING2, which are stoichiometric members of Sin3/HDAC1 complexes and bind H3K4Me3 by interactions with their PHD domains. Comparing the single cell (SC) transcriptomic profiles of human TNBCs against bulk-TNBC transcriptome profiles while manipulating the expression of HDAC1 and/or the ING1 targeting subunit, revealed strong effects of integrin signaling activity toward the FAK-Src-NEDD9 signaling axis. In this study, we investigate the upstream regulatory role of the HDAC1/Sin3A/ING1 complex at the chromatin and protein levels through inside-out (tumor cell) and outside-in (tumor microenvironment) regulatory mechanisms using engineered cancer-associated fibroblasts (CAFs) in vitro and in vivo. We anticipate the outcome of this study will clarify the mechanism(s) involved in the LBH589 side-effect of increased breast cancer invasion and propose mechanisms to selectively inhibit and reverse this concerning side-effect.

LAY SUMMARY

FDA-approved HDAC inhibitor LBH589 inhibits tumor proliferation, but unexpectedly induces cellular invasion in triple-negative breast cancer in an EMT-hallmark independent manner. Disturbing the HDAC1-Sin3A/ING1 complex induced HDACi-mediated invasion through regulating the FAK/NEDD9 signaling axis. Activation of the HDAC1-Sin3A/ING1 complex antagonizes LBH589-induced invasion both at the chromatin and protein levels. Single-cell RNA-sequencing (scRNA-seq) analysis of patient-derived TNBCs reveal an enrichment of the integrin signaling cascade in tumor cells. NEDD9, the FAK/Src complex scaffolding protein, is highly expressed in TNBCs while being significantly less active in non-tumorigenic mammary gland epithelial cells. Inhibition of integrin signaling both inside-out (disturbing NEDD9-FAK-Src complex through the Sin3A complex) and outside-in (disturbing membrane integrins using engineered cancer associated fibroblasts (e-CAFs)) improves HDACi treatment efficacy by decreasing invasion and further inhibiting proliferation, while minimizing toxicity.

Roles of Grainy head-like Transcription Factors in Mammary Epithelial Differentiation - Poster #484

Presenter: Nancy Aloysius, Université de Montréal

Other Authors: Faustine Gorse, Jessica Adams, Annabelle Chaput, Lucas Porras, Salwa Haidar, Sylvie Mader

ABSTRACT_ID#2628

ABSTRACT

Breast cancer (BC) is one of the most prevalent and life-threatening diseases affecting women worldwide. The epithelial-mesenchymal transition (EMT) and the mesenchymal-epithelial transition (MET) play key roles in the progression of all BC subtypes. However, the transcription factors (TFs) regulating the MET process remain poorly understood compared to EMT. GRHL2 has been implicated in BC epithelial phenotype establishment and maintenance. The roles of the other GRHL family members, GRHL1 and GRHL3, expressed preferentially in basal-like breast tumors, are less characterized. We hypothesize that GRHL factors play partially overlapping roles in suppressing mesenchymal gene expression and inducing differentiation into different epithelial subtypes. Our goals are to characterize the contribution of each GRHL TF in (i) inducing MET in mesenchymal breast cell lines and (ii) maintaining an epithelial phenotype in cell models of different breast cancer subtypes. GRHL1, GRHL2, and GRHL3 could all activate transcription from a consensus GRHL-binding sequence, although isoforms of GRHL3 had variable transcriptional activity. Overexpression of GRHL1 and GRHL2 were detected in the luminal vs myoepithelial compartment of the normal mammary gland by spatial profiling. Stable overexpression of GRHL1 and GRHL2 in MDA-MB-231 mesenchymal breast cancer cells resulted in a partial MET with both shared and distinct sets of regulated genes. shRNA-mediated knockdown and sgRNA-mediated knockout of GRHL TFs in cell lines representative of different breast cancer subtypes is in progress. Clarifying and understanding the roles of GRHL family members in different breast cancer subtypes will provide insight into their contribution to mammary epithelial cell differentiation and tumorigenesis.

LAY SUMMARY

GRHL1 and GRHL2 can promote epithelial features in breast cancer cells Breast cancer cells can switch between more invasive, mesenchymal-like states and more organized, epithelial-like states. We found that overexpressing GRHL1 or GRHL2 in mesenchymal breast cancer cells induced features of the epithelial state—a process known as mesenchymal-to-epithelial transition (MET), which is associated with changes in cell shape, behaviour, and gene expression. GRHL1 and GRHL2 are more active in the luminal part of the mammary gland Using spatial profiling of normal breast tissue, we found that GRHL1 and GRHL2 are more highly expressed in luminal epithelial cells compared to the surrounding myoepithelial cells. This supports the idea that these proteins play a key role in maintaining differentiated epithelial cell types in the breast. Different GRHL proteins may control unique aspects of cell identity in breast cancer Although all three GRHL proteins (GRHL1, GRHL2, GRHL3) can activate gene expression, they don't all behave the same. GRHL3 isoforms showed varying activity, suggesting that isoform-specific properties may influence their regulatory roles in breast cancer cells.

Fatty Acid Binding Protein 12 Dependent Metabolism in Prostate Cancer - Poster #486

Presenter: Noah Wood, University of Alberta

Other Authors: Cécilia Bernier, Maryam Lo, Rong-Zong Liu, Roseline Godbout, Hélène Lemieux

ABSTRACT_ID#2544

ABSTRACT

Prostate cancer (PCa) accounts for nearly a quarter of new cancer diagnoses in Canadian men. Although early-stage PCa has a nearly 100% 5-year net survival rate, this decreases drastically to ~40% once PCa metastasizes. Unlike many cancers that primarily rely on glycolysis for energy production, PCa depends on mitochondrial fatty acid oxidation. Fatty acid binding proteins (FABPs) are a family of lipid-binding proteins that facilitate the transport, subcellular distribution and utilization of their fatty acid ligands. The newest member of this family, FABP12, is overexpressed in metastatic PCa compared to primary tumours. This project investigates the role of FABP12 in energy production and lipid metabolism in metastatic PCa. Our lab has previously shown that FABP12 expression in PCa supports epithelial-to-mesenchymal progression and stemness in vitro, key traits associated with metastatic phenotypes. High-resolution respirometry revealed a fatty acid-dependent increase in mitochondrial fatty acid β -oxidation in FABP12-expressing PCa cells compared to controls. We hypothesize that FABP12 and its fatty acid ligands contribute to PCa progression by enhancing mitochondrial fatty acid oxidation. Our aims are to: (i) identify the preferential energy substrates in FABP12-expressing versus non-expressing cells, and (ii) uncover the genes and key steps driving enhanced fatty acid β -oxidation in FABP12-expressing cells. Our data, to date, indicate that FABP12-expressing cells have a greater capacity than non-expressing cells to metabolize long and medium chain fatty acids, potentially conferring metabolic advantages to PCa in lipid-rich environments.

LAY SUMMARY

Prostate cancer has an excellent outcome in its early stages, with a 5-year survival of almost 100%. Upon spreading, there is a dramatic drop in the 5-year survival, down to ~40%. Prostate cancers primarily rely on fats for energy, unlike many other cancers. The fatty acid binding protein family is a group of proteins that transports and distributes lipids within cells. A member of this family, fatty acid binding protein 12, is highly expressed in aggressive prostate cancer. Our lab has previously shown that this protein contributes to aggressive traits and that it increases the energy production from lipids in prostate cancer cells. Our goal is to identify specific lipids preferred by aggressive prostate cancer. We also aim to identify the genes and cellular processes that are altered with the expression of fatty acid binding protein 12, allowing for the metabolism of these lipids. A technique called high-resolution respirometry allows us to measure the energy production of cells that express and do not express this protein. We have shown cells that express fatty acid binding protein 12 prefer certain lipids of varying size, indicating that they may have an advantage in energy production in certain lipid-rich environments

Exploiting Multidimensional Synthetic Lethalities to Overcome PARPi Resistance in Ovarian Cancer - Poster #487

Presenter: Mandana Bigdeli, CRCHUM

Other Authors: Skye Sauriol, Euridice Carmona, Diane Provencher, Anne-Marie Mes-Masson, Francis Rodier

ABSTRACT_ID#3085

ABSTRACT

Introduction: High-grade serous ovarian cancer (HGSOC) is the most common and lethal subtype of ovarian cancer. Despite therapeutic advances, most patients relapse. PARP inhibitors (PARPi) are initially effective in homologous recombination-deficient HGSOCs by exploiting synthetic lethality, but resistance frequently develops due to therapy-induced adaptations. We previously demonstrated that NAMPT inhibitors (NAMPTi) can re-sensitize PARPi-resistant cells, though the underlying mechanism remains unclear. We also showed that PARPi induces a reversible senescence state, which can be exploited with senolytics in a “one-two punch” strategy, currently being tested in a phase I clinical trial (NCT05358639). Here, we hypothesize that the new NAMPTi-PARPi combination triggers therapy-induced cell fate decisions, particularly senescence, leading to enhanced cell death via a temporally coordinated synthetic lethal mechanism. **Methods:** We evaluate cell fate dynamics in response to NAMPTi-PARPi using isogenic sensitive and resistant HGSOC cell lines, assessing proliferation, apoptosis, mitotic catastrophe, senescence, and the secretome via live-cell imaging, biomarker assays, and mass spectrometry. We identify altered pathways and markers using a multiomics approach (transcriptomic and metabolomic profiling). Resistance markers and therapeutic targets will be validated using multiplexed antibody imaging and patient-derived xenografts. **Results:** The NAMPTi-PARPi combination re-sensitizes resistant HGSOC cells, induces extensive DNA damage, and triggers senescence-to-death transitions. Transcriptomic data reveal dysregulation of senescence, OXPHOS, cholesterol biosynthesis, and metabolic pathways, which are under validation. **Conclusion and Impact:** Targeting therapy-induced cell fate adaptations offers a rational strategy to overcome PARPi resistance. This approach supports precision treatment design and may inform therapeutic strategies in other cancers with similar resistance mechanisms.

LAY SUMMARY

Ovarian cancer often becomes resistant to treatment, especially to a class of drugs called PARP inhibitors (PARPi), which are designed to target tumors with specific DNA repair defects. Once resistance develops, treatment options are limited. Our research explores a new two-step strategy to target and eliminate resistant cancer cells. We combine PARP inhibitors with another drug that blocks cancer cell metabolism (called a NAMPT inhibitor). This combination pushes cancer cells into a vulnerable state (senescence), which ultimately leads to their death. This approach re-sensitizes tumors to treatment and could improve outcomes for patients with ovarian cancer that no longer responds to standard therapies. By understanding how cancer cells adapt to therapy at the molecular level, we aim to design smarter, more precise and effective treatment combinations.

CD13 Loss Disrupts Tissue Architecture to Promote Cancer Progression - Poster #489

Presenter: Marie-Ève Proulx, McGill University

Other Authors: Rachel Catterall, Shruti Rughani, Luke McCaffrey

ABSTRACT_ID#2420

ABSTRACT

Breast cancer is the most diagnosed cancer among women in Canada. A requirement for breast cancer initiation is the disruption of epithelial tissue architecture. In healthy breast tissue, luminal epithelial cells are highly organized, forming ducts with hollow lumens and well-defined apical-basal polarity. The apical membrane faces the central lumen, and the basolateral membrane faces the extracellular matrix and neighbouring cells. The development of breast cancer is characterized by filling of the lumen and subsequent dispersion of malignant cells from the duct. Despite the knowledge that disrupted tissue architecture is permissive for cancer initiation and progression, our understanding of the mechanisms contributing to this tissue disruption in early cancer remains largely unknown. Our previous research identified CD13, a transmembrane metalloprotease expressed on the surface of epithelial cells, as a pioneer factor essential for lumen formation and maintenance. Notably, CD13 protein levels are reduced in breast cancer. Here, we demonstrate that the loss of CD13 is sufficient to recapitulate early architectural changes observed in breast cancer using a 3D mammary organoid model. In this model, CD13 depletion leads to increased epithelial stratification and loss of lumen integrity. Strikingly, while many oncogenes require long latency to develop cancer, we observed that loss of CD13 dramatically accelerates these steps in a 3D organoid model. These findings uncover a previously unknown role for CD13 as a potent tumour suppressor in the mammary epithelium.

LAY SUMMARY

We demonstrate that the loss of a protein, CD13, can recapitulate the architectural changes observed in early breast cancer in a spheroid model. We found that loss of CD13 dramatically accelerates breast cancer progression by disrupting the structure in a spheroid model.

Influencing the Modality of Programmed Cell Death during Cancer Therapy - Poster #490

Presenter: Noah Conohan, Memorial University of Newfoundland

Other Authors: Kensuke Hirasawa, Lingyan Wang, Maria Licursi, Lin Ling, John Thoms

ABSTRACT_ID#2564

ABSTRACT

Introduction During cancer therapy, radiation or chemotherapeutic agents are used to cause cellular damage and initiate programmed cell death (PCD) in tumors. Many modalities of PCD exist, such as apoptosis and necroptosis. Apoptosis generally does not stimulate the immune system. In contrast, necroptosis is a form of immunogenic cell death that activates the immune system by releasing inflammatory mediators and tumor antigens into the microenvironment. Immunogenic cell death is often preferred during cancer treatment as it can initiate antitumor immunity in patients. Caspase-8 is a regulator of PCD pathways that initiates apoptosis but suppresses necroptosis. We hypothesize that inhibiting caspase-8 during cancer therapy will polarize cells to preferentially undergo necroptosis and promote the generation of antitumor immunity. **Methods** Mouse cancer cell lines (MC38 and EO771) were irradiated or treated with chemotherapy agents (Cisplatin, Doxorubicin, Etoposide and 5-FU). To determine roles of caspase-8 in regulating PCD pathways, RNAi knockdown of caspase-8 was conducted in cells prior to radiation or chemotherapy. Cell viability was measured by crystal violet assay. Activation of PCD pathways was determined by western blot analysis. RT-qPCR analysis was conducted to examine production of inflammatory mediators. **Results and Conclusion** Caspase-8 knockdown promoted necroptosis induction in irradiated MC38 and EO771 cells, accompanied by increased expression of inflammatory mediators in MC38 cells. Caspase-8 knockdown also increased necroptosis induction in cells treated with chemotherapy dependent on combination of cell line and chemotherapy agent. Our results indicate that caspase-8 could be a therapeutic target for increasing immunogenic cell death during standard cancer therapies.

LAY SUMMARY

During cancer treatment, radiation and chemotherapy are regularly used to kill the cancer cells that comprise tumors. Cancer cells die through various controlled processes that can differ in the engagement of the immune system. This is important because the immune system can help remove tumors throughout the body. In our experiments, we have observed that during radiotherapy and chemotherapy, cancer cells predominantly undergo a type of death called apoptosis, which generally does not encourage immune responses against tumors. We hypothesized that targeting a specific protein called Caspase-8, which regulates the fate of dying cells, could trigger a form of cell death known as necroptosis. This form of cell death may enhance the recruitment of the immune system and increases the likelihood of developing immunity to tumors. Our results show that depending on the type of cancer cells and treatment, targeting Caspase-8 can increase necroptosis in cells. These findings show that this approach may be an effective strategy to increase the involvement of the immune system and improve patient outcomes during standard cancer therapy.

Sunday, November 2, 2025

Elucidating the impact of the p53 checkpoint throughout breast cancer progression in the presence of chronically elevated oxidative stress - Poster #491

Presenter: Meghan Lancaster, McGill University

Other Authors: Rachel La Selva, Eduardo Cepeda Cañedo, Emma Ciccolini, Elias Maldonado, Caitlynn Mirabelli, Valarie Sabourin, Josie Ursini-Siegel

ABSTRACT_ID#2466

ABSTRACT

The p53 transcription factor is a ubiquitously expressed protein that helps cells respond to stress by preventing the accumulation of DNA damage, initiating cell cycle arrest, giving cells time to repair themselves or initiating apoptosis if the damage is too severe. Without p53, DNA damage accumulates and makes cells susceptible to oncogenic transformation. Because of this, p53 suppresses tumor formation and mutations in TP53 can lead to cancer development. A paradox arises in the case of breast cancer. More than half of breast cancer tumors retain wild-type TP53. I will test the hypothesis that in these instances, p53 endows breast cancers with the ability to adapt to stressors, contributing to their increased survival. My research focuses on understanding the importance of the p53 checkpoint in breast cancer progression in the context of elevated oxidative stress. My research utilizes immortalized mammary gland epithelial cell lines NMuMG and MCF10A, as well as transformed models NMuMG-NT and MCF7, each of which have an intact p53 checkpoint. Using these models, we will ask how p53 engages with elevated oxidative stress to alter breast cancer progression using 2D clonogenic assays, soft agar assays, and in vivo mouse studies. I will compare wild-type TP53 expressing cells and those engineered to lose p53. In both cases, we will induce elevated levels of oxidative stress by increasing the expression of p66ShcA, a known ROS producer. We will ask how p53 and p66ShcA mechanistically interact during cancer evolution to contribute to normal breast epithelial cells becoming cancerous.

LAY SUMMARY

Despite more than half of breast cancer tumors retaining the wildtype tumor suppressor TP53, these tumors can still have aggressive phenotypes p53 may be engaging with elevated oxidative stress to alter breast cancer progression Maintaining wild-type TP53 could be advantageous to breast cancer cells when in the presence of chronically elevated oxidative stress

Molecular Crosstalk Between LMO2 and ZEB2 in Early Thymic Progenitor Acute Lymphoblastic Leukemia - Poster #492

Presenter: Kenneth Osafire, Paul Albrechtsen Research Institute, Cancer Care Manitoba

Other Authors: Jody Haigh, Cedric Tremblay

ABSTRACT_ID#2722

ABSTRACT

Background Early Thymic Progenitor Acute Lymphoblastic Leukemia (ETP-ALL) is a high-risk, chemoresistant subtype of T-cell ALL, affecting 10–15% of cases, particularly in children and young adults. It is marked by stem-like and myeloid gene expression and poor response to standard treatments. Persistent leukemic stem cells (LSCs) contribute to relapse and resistance. The transcription factors LMO2 and ZEB2 are implicated in ETP-ALL, but their functional role remains unclear. Objective This study investigates the cooperative role of LMO2 and ZEB2 in promoting LSC survival and evaluates a potential therapeutic strategy using dual inhibition with SP2509 (LSD1 inhibitor) and Dynole 34-2 (dynamin inhibitor). Methods Human T-ALL cell lines (LOUCY, PEER, ICHIKAWA) co-expressing LMO2 and ZEB2 were identified. CRISPR/Cas9 knockouts of each gene assessed their impact on IL7R, BCL2 (anti-apoptotic), and BIM (pro-apoptotic). Apoptosis and proliferation assays were conducted. Murine ETP-ALL lines (TG39, TG50) and a mature T-ALL line (WT-1) were treated with SP2509 and Dynole 34-2 to assess cytotoxic synergy. Results Knockouts of LMO2 or ZEB2 reduced IL7R and BCL2, increased BIM, induced apoptosis, and decreased proliferation. LMO2 also regulated ZEB2 expression, suggesting a feedback loop. The SP2509/Dynole 34-2 combination showed synergistic cytotoxicity in ETP-ALL but not in WT-1 cells. Conclusion LMO2 and ZEB2 cooperate to sustain LSC survival in ETP-ALL. Dual LSD1/dynamin inhibition disrupts this survival axis, offering a promising therapeutic strategy now advancing to in vivo validation.

LAY SUMMARY

We discovered that two genes, LMO2 and ZEB2, work together to help leukemia cells survive in a high-risk form of blood cancer called ETP-ALL. When we removed either gene, the cancer cells became weaker, grew less, and were more likely to die—suggesting these genes help leukemia cells resist treatment. Our study showed that a drug combination targeting both LSD1 (an enzyme that controls gene activity) and dynamin (a protein involved in cell survival) kills ETP-ALL cells more effectively than current treatments. This approach was especially powerful in drug-resistant leukemia cells that mimic early blood development. The two drugs—SP2509 and Dynole 34-2—not only stop survival signals in cancer cells but also increase cell death signals, working together in a way that's stronger than either drug alone. This combination may offer a new, more effective option for patients who don't respond to standard therapies.

Multi-omic Profiling of Extracellular Vesicles in Leiomyosarcoma - Poster #494

Presenter: Pateel Markarian, McGill University

Other Authors: Philippe Jolivet, Deirdre Lum, Kristen Ganjoo, Matt van de Rijn, Rebecca Gladdy, Jonathan A. Fletcher, Joanna Przybyl

ABSTRACT_ID#2406

ABSTRACT

Extracellular vesicles (EVs) – nanosized lipid-enclosed particles released from cells – are widely recognized as a potent analyte for liquid biopsy. Tumor-derived EVs released into body fluids of cancer patients carry oncogenic molecules and transfer them to healthy cells, modulating their behaviour and promoting tumor dissemination. This positions EVs as valuable diagnostic markers for early detection and monitoring of cancer. There is currently a poor understanding of the role of tumor EVs in leiomyosarcoma (LMS). LMS is a highly malignant mesenchymal tumor that frequently metastasizes and is associated with poor prognosis. Investigating LMS-derived EVs may provide insights into early diagnosis and prediction of metastasis via liquid biopsy. Therefore, this study seeks to characterize LMS-derived EVs, investigate their molecular cargo, and identify currently unknown pathological processes contributing to LMS metastasis. We isolated EVs from cultured cells of LMS and leiomyoma (LM, benign counterpart of LMS), as well as from plasma samples of LMS and LM patients and healthy donors. Nanoparticle Tracking analysis revealed particle size and concentration within the size range characteristic of small EVs. Western blot confirmed EV markers (e.g., syntenin, TSG101, CD63), validating EV population in our samples. In the next steps, we will study the differential expression of EV-proteins and evaluate the genomic profile of EV-DNA using mass spectrometry and shallow whole genome sequencing, respectively. This study will pave the way for future functional studies of the role of EVs in LMS metastasis and may contribute to developing liquid biopsy tests to improve clinical outcomes in LMS patients.

LAY SUMMARY

1. Cancer cells release small particles called extracellular vesicles (EVs) into body fluids of patients, carrying molecules that can promote tumor growth and spread to distant sites in the body.
2. Leiomyosarcoma (LMS) is an aggressive tumor associated with high risk of metastasis and poor survival rate, highlighting the need for improved markers for early detection of disease progression.
3. Investigating EVs shed by LMS tumors can aid in understanding the molecular mechanisms of tumor spread and develop non-invasive tests for early diagnosis and treatment monitoring of LMS patients.

Investigating the role of the RNA cytosine methyltransferase NSUN2 in ovarian cancer - Poster #495

Presenter: Zorica Nakevska, University of Alberta

Other Authors: Farzaneh Afzali, Huachen Chen, Tyler Cooper, An Nhien Tong, Congxiang Yu, Zhihua Xu, Rui Zhe Yang, Holly Zhao, Guihua Zhang, Dupreez Smith, James Key, Gilles Lajoie, Helen Steed, Martin Köbel, Lynne-Marie Postovit, Cheng-Han Lee, YangXin Fu

ABSTRACT_ID#2471

ABSTRACT

Epithelial ovarian cancer (EOC) is the most lethal gynecologic malignancy, with no effective treatments for advanced disease. The RNA modification 5-methylcytosine is catalyzed by NSUN family methyltransferases, regulating gene expression. NSUN2 influences mRNA stability and translation and has been implicated in various cancers. However, its role in EOC remains unclear. Thus, we aim to identify NSUN2's functional role in EOC. We knocked down NSUN2 using shRNA and knocked out NSUN2 using CRISPR/Cas9 in various EOC cell lines and assessed the phenotypic effects of NSUN2 depletion on cell viability, proliferation, and tumor formation. RNA sequencing, proteomic analysis, and downstream validation were performed to identify differentially expressed genes in NSUN2-deficient cells. NSUN2 knockdown reduced cell growth and colony formation, whereas NSUN2 knockout had a similar but less pronounced effect. The severity of phenotype caused by NSUN2 deficiency also varied between cell lines. RNA sequencing revealed more differentially expressed genes in NSUN2 knockdown compared to knockout cells with respect to their controls, suggesting an adaptive response, where the NSUN2 knockout cells restore some of the altered genes in response to NSUN2 deficiency over time. Our findings indicate that NSUN2 has cell-context dependent effects in EOC, urging us to identify in which cellular contexts targeting NSUN2 could be beneficial in EOCs. In conclusion, depletion of NSUN2 alters gene expression and mitigates the tumorigenic phenotypes in EOC cells. However, these effects are only observed in certain cellular contexts. Thus, identifying these contexts and examining the molecular pathways regulated by NSUN2 in EOC warrants further investigation.

LAY SUMMARY

Epithelial ovarian cancer (EOC) is a deadly disease with few treatment options, especially in advanced stages. We are studying how changes in the chemical “decorations” that are normally added to RNA affects EOC growth. We focused on a molecule called NSUN2, which is a protein that chemically modifies RNA. When we depleted NSUN2 from EOC cells, the cells grew more slowly, suggesting that NSUN2 plays a role in promoting tumor-like behavior.

Loss of the CHD6 chromatin remodeling factor is synthetic lethal with BRCA1- and BRCA2-deficiency and is a potential strategy to alleviate poly-(ADP)-ribose polymerase inhibitor resistance - Poster #496

Presenter: Peter Brownlee, University of Calgary

Other Authors: Luc Provencher, Reece Hall, Wilson Nartey, Dustin Pearson, Askar Yimit, Gareth Williams, Aaron Goodarzi

ABSTRACT_ID#3338

ABSTRACT

Poly-(ADP)-ribose polymerase (PARP)-trapping small molecule inhibitors (PARPi) such as Olaparib are established as potent anti-cancer therapeutics for tumors displaying homologous recombination deficiency (HRD). Mutations in the BRCA1 and BRCA2 genes are amongst a growing list of genetic and epigenetic alterations giving rise to PARPi-sensitive tumors. However, the therapeutic success of PARP inhibitors in the clinic has been somewhat tempered by the eventual emergence of patient tumor resistance, leading to relapse. As such, there is an ongoing need to improve our understanding of the mechanisms governing PARP inhibitor resistance, and identify novel factors that modulate PARPi sensitivity in general. We have identified the CHD6 chromatin remodeler as a factor that sensitizes cells to loss of BRCA1 or BRCA2, as well as to PARPi in a manner that is distinct from HRD. CHD6 itself is frequently mutated in multiple human cancers, and therefore has potential as a target of interest for novel anti-cancer therapeutic discovery. I will present unpublished biological data that demonstrates loss of CHD6 expression or function produces innate synthetic lethality to BRCA1- and BRCA2-loss, and multiplies human cell sensitivity to PARP inhibitors. Importantly, CHD6 loss is able to overcome clinically important PARPi resistance mechanisms that have been observed in BRCA1- and BRCA2-deficient tumors, such as reduced 53BP1 expression. We will present data outlining the molecular mechanism underlying CHD6 loss-mediated PARPi sensitivity, as well as biochemical and structural biological evidence for a mechanism by which CHD6 might be inhibited therapeutically.

LAY SUMMARY

Loss of the chromatin remodeling factor CHD6 is synthetic lethal with BRCA1/2-deficiency CHD6 loss can overcome clinically relevant PARP inhibitor resistance mechanisms CHD6 might be an attractive target for therapeutic intervention in PARP inhibitor resistant tumors

Investigating the epigenetic landscape governing glioblastoma state plasticity and tumor heterogeneity -

Poster #497

Presenter: Nayantara Govindrajan, University of Calgary

Other Authors: Michael Van Vliet, Kateryna Yakubets, Ana Nikolic

ABSTRACT_ID#2804

ABSTRACT

Glioblastoma multiforme (GBM) is the most common malignant adult brain cancer, with a median survival of just 15 months. Recurrence usually occurs despite radiation and chemotherapy treatment. GBM cells exist along a dynamic spectrum of transcriptional states- oligodendrocyte progenitor-like (OPC), astrocyte-like (AC), neural progenitor-like (NPC), and mesenchymal-like (MES)- and can transition between these subtypes in response to microenvironmental and epigenetic cues. To test the hypothesis that GBM tumors have different degrees of epigenetically mediated cell state plasticity, we generated novel patient-derived fluorescent reporter lines using state-specific epigenetic markers identified via single-nucleus ATAC-seq of primary GBM. These reporters target GBM cell states with distinct fluorophores, with a method we call “epigenetic state painting”. Preliminary characterization revealed state-specific differences in proliferation, sphere-forming ability, and plasticity between AC/MES and OPC/NPC tagged reporter lines. This data can be used to calculate a relative plasticity index, which we will use to stratify lines for ATAC-seq profiling to identify epigenetic contributors of GBM state plasticity. Significance: We developed a novel epigenetic reporter system to track and quantify GBM cell state plasticity, enabling identification of epigenetic drivers of tumor progression and recurrence.

LAY SUMMARY

Brain tumors like glioblastoma (GBM) often recur after treatment. This could be due to how tumors are made up of different cell states that can transition from one to the other. Our research focuses on understanding how and why these changes happen. We created special fluorescent reporter lines that light up in different colors, based on the state of the cell. These reporters are attached to genes that control how accessible the chromatin is. These modifications of chromatin without changes in DNA sequence are termed the “epigenetic state” of a cell. Our reporter lines allow us to track how cells of each type behave and how they shift over time, and link that to the epigenetic state of the GBM cell. Using these tools, we found that different GBM cell states grow and behave differently, with some showing more ability to switch states. Our next steps will be to quantify this ability to change state and identify how that relates to the epigenetic state.

Molecular and Cellular Biology

Sunday, November 2, 2025

Defining the Proximity Interacting Partners of the Tyrosine Kinase SRMS - Poster #499

Presenter: Nayoung Kim, University of Saskatchewan

ABSTRACT_ID#2479

ABSTRACT

Tyrosine kinases play an important role in intracellular signaling cascades and are involved in regulating various key biological processes including growth, metabolism, and differentiation in response to both external and internal stimuli. SRMS is a member of the BRK family kinases alongside BRK and FRK. Previous research has found BRK to display oncogenic properties and FRK playing an opposing role in tumour suppression. Unfortunately, the role of SRMS is understudied. Recent publications show promising insight for SRMS in promoting tumour growth through phosphorylating the scaffolding protein FKBP51, as well as its promise as a biomarker for colorectal and gastric cancers. Additionally, SRMS was found to play a potential role in platinum resistance in ovarian cancer. SRMS displays an interesting, primarily cytoplasmic, punctate localization within the cell, known as the SRMS Cytoplasmic Punctae. Currently, it is unknown what proteins SRMS may be interacting with to form these structures. Therefore, we aim to identify the proximity partners of SRMS using a technique known as BioID in the hopes to better understand its cellular functions. Recently, we have conducted biotin affinity purification and mass spectrometry analyses for the identification of SRMS proximity partners. We predict that proteins involved in the regulation of the PI3K-AKT pathway interact with SRMS and have conducted functional assays to validate this potential, novel SRMS signaling pathway. Defining the proximity partners of SRMS may result in a better understanding of the regulation and function of SRMS and the role this kinase plays in diseases such as cancer.

LAY SUMMARY

Proteins play various important roles in body. One class of proteins known as kinases, are involved in cell signaling, acting as on and off switches or as messengers in the game of cellular telephone. Cancer can be a result of these switches or messengers not functioning properly and relaying too much, not enough, or the incorrect information. One family of kinases are known as the BRK family kinases. There are three members in this family of protein: BRK, FRK and SRMS. BRK has been found to play a role in promoting cancer, while FRK has been found to play the opposite role by suppressing tumour growth. SRMS is the least studied member, and its role in the cell warrants further research. Just like how one can read what a person is like by looking at the people they surround themselves with, the same can be said about proteins. By studying where a protein goes in the cell and what other proteins it surrounds itself with, one can gain a better understanding of its function. Therefore, we aim to study SRMS' interacting proteins to better understand this kinase and the role it may be playing in diseases such as cancer.

The role of Lin28B in promoting NEPC development - Poster #500

Presenter: Qiongqiong Jia, Vancouver Prostate Centre

Other Authors: Ning Xie, Faraz Hach, Baraa Orabi, Xuesen Dong

ABSTRACT_ID#2562

ABSTRACT

Therapy-induced neuroendocrine prostate cancer (t-NEPC) is a highly aggressive form of prostate cancer often marked by the acquisition of stemness phenotypes. Our previous work demonstrated that Lin28b regulates a stemness gene network to promote t-NEPC development. However, under stress conditions, Lin28b is sequestered in stress granules (SGs), and its role in RNA compartmentalization and translational regulation during NEPC progression remained unclear. In this study, we show by immunofluorescence microscopy that Lin28b promotes the formation of stress granules (SGs) in NEPC cells. In contrast, Ln268, a Lin28b-specific inhibitor developed through computational drug design, suppresses SG formation. Using sucrose gradient polysome fractionation (SGPF) and APEX-based proximity labeling followed by RNA-sequencing, we found that under stress conditions, Lin28b actively excludes transcripts associated with stemness and neuroendocrine differentiation from SGs. These transcripts are instead enriched in polysomes (PS), facilitating their continued translation. Additionally, we identified that Lin28b forms a protein complex with SALL4 and enhances its transcriptional activity, thereby promoting prostate cancer cell survival. Together, these findings reveal that under stress, Lin28b reprograms the translational landscape to favor the expression of genes with cancer stemness functions. This highlights Lin28b as a promising therapeutic target in NEPC.

LAY SUMMARY

Lin28b helps cancer cells survive stress: In aggressive prostate cancers like therapy-induced neuroendocrine prostate cancer (t-NEPC), cancer cells often face stress from treatments. We found that Lin28b helps these cells adapt by changing how certain genes are used during stress, allowing the cancer to keep growing. Lin28b controls where gene messages go: When cells are under stress, they can temporarily store gene messages (mRNAs) in structures called stress granules (SGs), which pauses their use. Lin28b prevents key gene messages—especially those related to cancer stemness and neuroendocrine traits—from being stored in SGs. Instead, it moves them to areas (polysomes) where they can still be turned into proteins. Lin28b work together with another protein to boost cancer traits: Lin28b works with a protein called SALL4 to turn on genes that help cancer cells act like stem cells—making them more resistant to treatment and able to survive longer.

Functional role of HMGB3 in tumor progression and DNA damage response in neuroendocrine tumors - Poster #501

Presenter: Liyana A. Nilakshi Kulathunga, Sunnybrook Research Institute

Other Authors: Nilakshi Kulathunga, Areeba Qureshi, Manjusha Muralidharan, William You, Taraneh Hajian, Mark Mabanglo, Benjamin Lok, Masoud Vedadi, Kathrin Tyryshkin, Anne-Claude Gingras, Iacovos P. Michael

ABSTRACT_ID#2604

ABSTRACT

Introduction: Our previous work demonstrated that upregulation of High mobility group box 3 (HMGB3) contributes to the transition of the relatively benign pancreatic neuroendocrine (PanNET) molecular subtype to a more aggressive and highly metastatic subtype through dedifferentiation. Moreover, elevated HMGB3 expression has been implicated in metastasis and therapeutic resistance across multiple solid tumor types. **Aims:** This study aimed to characterize and validate the HMGB3 protein interactome and to understand its functional role in PanNET progression. **Material and Methods:** We employed two protein-based approaches: proximity-dependent biotin identification (BioID) and FLAG-based immunoprecipitation (FLAG-IP), to investigate the HMGB3 protein interactome. To elucidate the functional role of HMGB3, we performed both gain-of-function and loss-of-function studies. **Results:** The HMGB3 protein interactome revealed its involvement in chromatin remodeling and organization through interaction with various complexes, including Polycomb Group (PcG) proteins, SWI/SNF complex. Additionally, it contributes to the DNA damage response and repair by interacting with proteins such as XRCC1, XRCC5, XRCC6, PARP1 and PARP2. Knockdown of HMGB3 significantly reduced the colony-forming ability of high-grade mouse and human PanNET cell lines, and this clonogenic ability was further reduced when the cells were exposed to radiation. **Conclusions:** These findings suggest that HMGB3 may contribute to PanNET aggressiveness through its effect on chromatin modifications and the DNA damage response pathway. Ongoing experiments are being carried out to validate the Hmgb3 protein interactome and elucidate the underlying mechanisms to improve the sensitivity to therapeutic response to radiation-based treatments such as radioligand therapy.

LAY SUMMARY

1. HMGB3 supports pancreatic neuroendocrine tumor (PanNET) progression Pancreatic neuroendocrine tumors (PanNETs) originate in the hormone-producing cells of the pancreas and become challenging to treat when they progress to high-grade cancers. A protein known as High-Mobility Group Box 3 (HMGB3) is found in high amounts in these advanced tumors and plays a critical role in helping the tumor spread to other parts of the body.
2. HMGB3 works with other proteins to control DNA organization and repair HMGB3 works with other key proteins to maintain the structural organization of chromatin, and therefore DNA organization inside the cells, and to repair DNA errors after damage. In cancer cells, these key functions of HMGB3 may help tumors grow faster.
3. HMGB3 makes cancer cells resistant to radiation treatment Cancer cells with high levels of HMGB3 can tolerate radiation treatment and survive better compared to cells that lack HMGB3. This protective role of HMGB3 is a major challenge in treating patients with radiotherapy. Targeting HMGB3 might be a promising strategy to make radiation therapy more effective and improve outcomes for patients with PanNETs and other cancers.

Unraveling the role of the CH25H–TFE3–RagB/D axis in cellular senescence - Poster #502

Presenter: Romain Villot, Frédérick Antoine Mallette Lab - Hôpital Maisonneuve-Rosemont

Other Authors: Paul Lemire, Karine Boulay, Pault Minaya Ferruzo, Christina Sawchyn, Agnieszka Dejda, Mathieu Neault, Peng Gao, Erwan Goy, Coralie Tambon, Laurence M.Gagné, Erlinda Fernández Díaz, Dagmar Glatz, Marine Régnier, Roxanne St-Georges, Cyril Carvalho, Nicolas

ABSTRACT_ID#2864

ABSTRACT

Cellular senescence is a stress response in which the cells remain viable but with altered and compromised function. Although cellular senescence could be harmful in some aging-related pathologies, it serves as a strong barrier to cancer which must be disassembled during tumoral transformation. Senescent cells secrete numerous inflammatory cytokines, named the senescence-associated secretory phenotype (SASP), which alters the tissue microenvironment and promotes tumor progression in some contexts. However, the molecular mechanisms underlying regulation of the SASP notably in cancer cells remain to be elucidated. We have obtained compelling data suggesting that a metabolic enzyme (cholesterol 25-hydroxylase, CH25H) implicated in cholesterol oxidation is upregulated during multiple in vivo senescence models. In addition, the elevation of CH25H is observed in vitro upon RAS activation or irradiation. Our results indicate that 25-hydroxycholesterol (25-HC) product by CH25H triggers cellular senescence in a p53-dependant manner and regulates the SASP by stimulating the mTOR pathway via indirect transcriptional events involving the Transcription Factor Enhancer 3 (TFE3). Thus, we propose a mechanism by which cellular senescence leads to the expression of CH25H and subsequently the potential elevation of 25-HC. In turn, 25-HC could help to prevent tumor transformation by reinforcing cellular senescence and SASP.

LAY SUMMARY

1. Senescence is a dynamic cellular state playing controversial roles in cancer development.
2. The enzyme CH25H is elevated during cellular senescence.
3. The 25-hydroxycholesterol (25-HC) is product by CH25H and induces cellular senescence via p53 and TFE3.

Epigenetic Targeting of the TEAD Repressive complex: A Promising Strategy Against Small Cell Lung Cancer - Poster #503

Presenter: Nivitha Bhaskar, Paul Albrechtsen Research Institute, Cancercare Manitoba

Other Authors: Joel Pearson

ABSTRACT_ID#2339

ABSTRACT

Lung cancer remains the leading cause of cancer-related mortality in Canada. Among the different subtypes of lung cancer, small cell lung cancer (SCLC) is the most aggressive, with a median survival of less than 10 months and five-year survival rate of less than 10%. Unfortunately, these outcomes have barely improved in 40 years, highlighting the urgent need for better therapies for SCLC. Recent work from our lab identified an essential function for the TEAD family of transcription factors in SCLC, where they cooperate with the CoREST transcriptional repressive complex to silence critical genes. This suggested that targeting the TEAD transcriptional repressive complex could serve as a novel therapeutic strategy for SCLC. This study evaluated whether inhibiting components of the TEAD repressive complex impacts cell viability and gene expression in SCLC. We tested Corin (a dual KDM1A/HDAC inhibitor), Entinostat (HDAC inhibitor), SP2577 (KDM1A inhibitor), and Vorinostat (pan-HDAC inhibitor) on multiple SCLC cell lines. Alamar Blue assays assessed viability, while qPCR and RNA-seq examined TEAD-regulated transcriptional changes. Corin exhibited the most potent effect, reducing viability and mimicking TEAD knockdown gene signatures. However, due to its limited clinical applicability, we investigated the synergy between Entinostat and SP2577. Their combination significantly lowered IC₅₀ and IC₇₅ values compared to individual treatments, supporting a rationale for combination therapy. Overall, our findings show that inhibiting the TEAD repressive complex—especially through combined HDAC and KDM1A targeting—offers a promising therapeutic strategy for this aggressive cancer.

LAY SUMMARY

Small cell lung cancer (SCLC) is one of the most aggressive and deadly forms of lung cancer, with a five-year survival rate below 10%. Sadly, these outcomes haven't changed in over 40 years, highlighting the urgent need for new treatment options. Recent work from our lab identified that a group of proteins called the TEAD repressive complex helps cancer cells survive by switching off genes that would normally stop tumor growth or trigger cell death. Based on this, we proposed that targeting this complex with inhibitors could lead to cancer cell death. We found that Corin, a dual inhibitor targeting two key members of the TEAD repressive complex, was highly effective at killing cancer cells by mimicking the effect of turning off this complex. This suggests a promising new treatment strategy for small cell lung cancer.

Cell fate shift dictated by L-glutamine in response to 25-hydroxycholesterol - Poster #504

Presenter: Roxanne St-Georges, Université de Montréal

Other Authors: Caroline Capdevielle, Sarah St-Amand, Romain Villot, Élise Latour, Karine Boulay, Frédérick Antoine Mallette

ABSTRACT_ID#3210

ABSTRACT

Normal cells possess intrinsic tumor suppressor mechanisms to prevent the spreading of damaged cells. Upon oncogene activation or in presence of DNA damage, normal cells can undergo cell death or cellular senescence, a cell fate characterized by a stable cell cycle arrest and an active metabolism. While cellular senescence has been initially associated to tumor suppression and to therapeutic response to cancer therapy, it is now known that accumulation of senescent cells rather promotes metastasis and tumor relapse. Our group recently demonstrated the enzyme Cholesterol 25-hydroxylase (CH25H), converting the cholesterol into 25-hydroxycholesterol (25-HC), is upregulated in different models of cellular senescence. Preliminary observations showed that cells exposed to 25-HC enters cellular senescence and displays a strong pro-inflammatory phenotype. Interestingly, we revealed a cell fate shift upon a 25-HC treatment dictated by the presence or absence of L-glutamine; promotion of cellular senescence in absence of L-glutamine, and cell death stimulation with its supplementation. Thus, we suggest that the L-glutamine supplementation modulates cell fate induction by 25-HC, representing a potentially new cell fate modulator. Results from flow cytometry against cell death markers of cells cultured in an incomplete amino acid media suggest a dependency of primary cells for certain non-essential amino acids upon 25-HC treatment. Metabolomic analysis by Seahorse and L-glutamine tracing of cells treated with 25-HC will offer a better understanding of the disruptions encountered by this metabolite, causing a metabolic failure which is leading to cell death, and open new avenues to develop senescent cells clearance therapies.

LAY SUMMARY

Cell fate is a set of physiological responses to intra and/or extracellular stress. Depending on the type of stress, cells have the option to fight against the aggression to survive, they can enter in a permanent cell cycle arrest to prevent mutation spreading, or they can die if the stress is too major, dangerous for the cell to stay alive. The cell fate outcome can be beneficial or deleterious based on the cellular environment context and a better understanding of the mechanisms playing a role in these decisions can lead to the development of new anti-cancer therapies. Different stimuli and molecules can act as a cell fate modulator. A supplementation, a restriction or an imbalance of a nutrient, a metabolic derivative or a cellular messenger (molecule transmitting a message to another cell, by activating or inhibiting a cellular mechanism) can lead to a cell fate switch, compared to a normal cellular environment.

Effect of the Ikaros isoform IK-4 on IMiD therapies and hematopoiesis - Poster #505

Presenter: Norrreen Quansah, Université de Montréal

Other Authors: Ailyn C. Ramon, Stefania Bottardi, Hugo Wurtele, Eric Milot

ABSTRACT_ID#3101

ABSTRACT

Acute lymphoblastic leukemia (ALL) and chronic myeloid leukemia (CML) are cancers that are frequently characterized by loss of Ikaros or by overexpression of dominant-negative (DN) isoforms of Ikaros. However, in multiple myeloma, therapies include immunomodulatory imides (IMiDs), which promote Ikaros proteasomal degradation. Importantly, the region required for IMiD's targeting Ikaros is absent in Ikaros DN isoforms over-represented in ALL and CML. We hypothesize that IMiDs differentially affect Ikaros isoforms and can favour the expression of DN isoforms over functional isoforms. We use Jurkat (T-ALL) and K562 (CML) cells with mutated Ikaros (Ik-KO), inserting either IK-1 (functional) or IK-4 (DN) isoform. These cells were characterized at steady state or after the addition of lenalidomide, an IMiD used in multiple myeloma therapies. Unlike IK-1, which is only detected in the nucleus, IK-4 is detected in both the nucleus and cytoplasm. Lenalidomide causes the degradation of IK-1 but hardly affects IK-4. Furthermore, the growth defects observed in Ik-KO cells are alleviated by the expression of either IK-1 or IK-4, as both are can restore proliferation in these cells. Our results demonstrate that IK-4 binds to chromatin, and interacts with the Ikaros-interacting partners NuRD and P-TEFb. However, unlike IK-1, which restores the regulation of all tested Ikaros target genes, IK-4 fails to fully regulate at least one tested IKAROS target gene. Thus, the DN isoform IK-4, frequently detected in ALL, localizes to the nucleus and is largely unaffected by lenalidomide. This study will help assess whether lenalidomide or other IMiDs pose unexpected risks.

LAY SUMMARY

In acute lymphoblastic leukemia (ALL), the Ikaros gene is often mutated or can produce non-functional versions of the protein, which leads to poor outcomes. These non-functional proteins are called Dominant Negative (DN) isoforms, which block functional Ikaros activity and are found at high levels in B-ALL and T-ALL cells. Interestingly, while losing functional Ikaros contributes to leukemia, some treatments for multiple myeloma aim to destroy Ikaros to kill cancer cells. Drugs called IMiDs (like lenalidomide) are used in the treatment of multiple myeloma to promote Ikaros degradation 2 % of multiple myeloma patients treated with IMiDs for a long period of time, are developing ALL or AML We discovered that IMiDs effectively degrade the normal Ikaros protein but hardly affect the DN isoform IK-4. We suspect that IMiD treatment might unintentionally increase levels of DN like IK-4, while degrading the normal Ikaros protein, possibly promoting leukemia. To study this, we use produced cell lines and mouse blood cells that produce either IK-1 (normal) or IK-4 (DN).We will analyze how IK-4 affects: cell growth, gene activity, response to lenalidomide, cell survival and differentiation This research will help determine whether IMiD therapy risks promoting non-functional forms of Ikaros and triggering leukemia.

Experimental and Theoretical evaluation of a copper (II) Schiff Base Complex: Interaction with Human Serum Albumin and Anticancer Properties - Poster #506

Presenter: Samaneh Ghofrani, University of Tehran

ABSTRACT_ID#2457

ABSTRACT

Remarkable therapeutic properties in metal compounds are still attracting scientists. We synthesized the $[Cu_2(HL)_2(\mu-Br)_2] \cdot H_2O$ complex and characterized it using IR, UV-Vis spectroscopy, and single-crystal X-ray diffraction. Using fluorescence quenching, circular dichroism, and molecular docking, we studied the interaction between HSA and the complex to better understand its pharmacological behavior. The fluorescence results indicated a static quenching mechanism with a binding constant (K_b) of 135 and $n \approx 1$, where n denotes the number of binding sites. In addition, CD spectroscopy revealed changes in HSA's secondary structure. Docking studies demonstrated that the complex binds in the hydrophobic pocket of subdomain IIA. The MTT assay revealed that this compound is toxic to cancer cells (HeLa and MCF-7). Cisplatin was included in the assay as a positive control to provide a benchmark for the activity of the synthesized compound. Overall, the findings highlight the promising anticancer potential of the synthesized copper(II) complex.

LAY SUMMARY

We synthesized and studied a copper-based compound that shows strong cytotoxic effects against two types of human cancer cells: breast (MCF-7) and cervical (HeLa). This suggests that the compound has potential as an anticancer agent. The compound was shown to interact with human serum albumin (HSA), a key blood protein that helps transport drugs in the body. This binding behavior may improve the compound's stability and effectiveness in drug delivery systems. Our findings revealed that the compound not only inhibits the growth of cancer cells but may also induce apoptosis, a natural form of cell death, which is often targeted in cancer treatment. Cisplatin was used as a benchmark to compare performance, and the results were promising.

EPB41L5 oligomerization as a potential target to prevent breast cancer progression - Poster #507

Presenter: Patrick Laprise, Université Laval/CRCHU de Québec-Université Laval

Other Authors: Lynn Martin, François Corriveau, Clémence Gamblin, Alexandra Jetté

ABSTRACT_ID#2342

ABSTRACT

BACKGROUND AND HYPOTHESIS. The protein EPB41L5 is essential for epithelial-to-mesenchymal transition (EMT), a process that facilitates cancer cell invasion and metastasis. EMT involves genetic reprogramming that downregulates epithelial markers (e.g., E-cadherin) and induces mesenchymal markers (e.g., fibronectin, N-cadherin, vimentin). While EPB41L5 is barely detectable in normal mammary epithelial cells, it is highly expressed in certain invasive breast cancers. Its depletion suppresses tumour cell invasion, metastatic spread, and drug resistance, highlighting EPB41L5 as a potential therapeutic target. Our preliminary data indicate that EPB41L5 functions as an oligomer. We hypothesize that disrupting EPB41L5 oligomerization could prevent tumour progression. **RESULTS.** By screening a compound library, we identified SUPER1, a small molecule that inhibits EPB41L5 oligomerization. Treating MDA-MB-231 breast cancer cells with SUPER1 phenocopies EPB41L5 knockout or expression of EPB41L5 mutant proteins unable to oligomerize. Specifically, SUPER1 reduces proliferation of MDA-MB-231 cells and represses their mesenchymal traits, as shown by modulation of EMT marker expression. Moreover, in 3D Matrigel culture, SUPER1-treated or EPB41L5-deficient cells form organized spheroids with a central lumen lined by a monolayer of cells, further supporting acquisition of an epithelial phenotype. Finally, SUPER1 sensitizes MDA-MB-231 cells to the chemotherapeutic drugs gemcitabine and paclitaxel. **CONCLUSION.** These findings suggest that EPB41L5 oligomerization is functionally relevant in aggressive breast cancer, and that SUPER1 may represent a novel tool to repress proliferation and cell characteristics associated with tumour progression.

LAY SUMMARY

We identified a molecule, named SUPER1, interfering with the function of a protein supporting cancer progression. Treating breast cancer cells with SUPER1 represses cell proliferation and invasion. SUPER1 sensitizes breast cancer cells to chemotherapeutic drugs used to treat patients.

CSDE1 regulates miR-486 function in leukemia - Poster #509

Presenter: Pavan Kumar Kakumani, Memorial University of Newfoundland

Other Authors: Arezou Arvand, Yunkoo Ko, Emily Tsao, Tony Chen, Kristin Hope, Chanseok Shin

ABSTRACT_ID#2590

ABSTRACT

MicroRNAs (miRNAs) are small non-coding RNAs that regulate gene expression at the post-transcriptional level, and misregulation of miRNA expression affects a broad range of leukemic processes, including proliferation, differentiation, and chemotherapy resistance. Despite the critical roles of miRNAs in leukemia, it remains unknown whether altered miRNA maturation contributes to its pathology. The non-canonical miRNA, miR-486, is highly expressed in maturing erythroblasts, and its loss leads to defects in erythroid differentiation and the development of leukemias. However, the factors regulating miR-486 function in erythroid differentiation and acute myeloid leukemia (AML) remain unexplored. In the current study, we demonstrate that the loss of the RNA-binding protein (RBP) Cold Shock Domain containing Protein E1 (CSDE1) results in decreased miR-486 function in erythroleukemia cells. CSDE1 interacts with the core miRNA protein AGO2 and promotes the release of the passenger strand in the miR-486 duplex. Furthermore, CSDE1 enhances the erythroid phenotype, while leukemia cells expressing its AGO2-interacting mutant exhibit defects in differentiation. Collectively, our results reveal, for the first time, CSDE1's role in controlling AGO2-dependent passenger strand release to achieve enhanced miR-486 function in the development of erythroleukemia.

LAY SUMMARY

The protein CSDE1 is essential for turning on a small RNA called miR-486, which is needed for healthy red blood cell development. Without CSDE1, miR-486 doesn't function properly, which can block blood cells from maturing—a problem often seen in leukemia. CSDE1 works by teaming up with another protein, AGO2, to help miR-486 do its job—this partnership could be a new target for treating leukemia.

Presenter: Seth Peyton, University of Alberta

Other Authors: Won Shik Choj, Pureunsol Jeon, H  l  ne Lemieux, Roseline Godbout

ABSTRACT ID#2676

Glioblastoma is the most common solid brain tumour, comprising ~60% of central nervous system malignancies. Despite aggressive therapies, outcomes remain poor with a median survival time of 12–15 months and a five-year survival rate below 5%. Recurrence and therapy resistance are major drivers of this lethality, in part due to glioblastoma stem-like cells (GSCs), which can migrate into healthy brain tissue and initiate secondary tumors. GSCs highly express Fatty Acid Binding Protein 7 (FABP7), a lipid chaperone we previously identified as promoting GSC migration. FABP7 preferentially binds polyunsaturated fatty acids including arachidonic acid (AA), which is a key activator of Protein Kinase C (PKC). PKC is a Ca^{2+} -dependent kinase that regulates signaling via phosphorylation. A downstream PKC target, Growth Associated Protein 43 (GAP-43), is enriched in glioblastoma tumour microtubules (TMs), ultralong protrusions that are critical for GSC migration and confer treatment resistance. Glioblastoma TMs, first identified in 2015, form a structural network between GSCs and rely on GAP-43 and the Ca^{2+} channel connexin-43 (CX-43) for their formation. Given PKC's activation by AA and Ca^{2+} , as well as FABP7's role in AA trafficking, both proteins may be critical in TM biology. Here, we show that targeting FABP7 and PKC disrupts TM formation in patient-derived glioblastoma cells. While PKC inhibitors are clinically limited by toxicity, FABP7's tumour-specific expression makes it a promising target. Additionally, we find that PKC activation leads to GAP-43 cleavage by calpains rather than phosphorylation, highlighting calpains as viable therapeutic targets for glioblastoma.

We have identified two proteins, FABP7 and PKC, which appear to play key roles in the formation of glioblastoma tumour microtubules, structures believed to promote the spread of glioblastoma cancer cells within the brain. Our data indicate that the formation of glioblastoma tumour microtubules is governed by a network of events driven by the binding of FABP7 and PKC to arachidonic acid. Arachidonic acid and the omega-3 polyunsaturated fatty acid DHA are abundant lipids in the brain. Both lipids bind to FABP7. We propose that altering the availability of DHA and AA may inhibit glioblastoma spread and improve patient outcomes.

Disruption of the Hexosamine Biosynthesis Pathway via GFPT2 Inhibition: A Novel Therapeutic Strategy for Leiomyosarcoma and Desmoid-Type Fibromatosis - Poster #511

Presenter: Pegah Rahimizadeh, McGill University

Other Authors: Richard Miallot, Philippe Jolivet, Matt van de Rijn, Raphael Pollock, Jonathan Fletcher, Livia Garzia, Joanna Przybyl

ABSTRACT_ID#2678

ABSTRACT

Introduction: Leiomyosarcoma (LMS) and desmoid-type fibromatosis (DTF) are sarcomas with limited treatment options. Cancer cells reprogram metabolic pathways to support malignancy, but the role of metabolic reprogramming in sarcomas remains unclear. We identified significant enrichment of the hexosamine biosynthesis pathway (HBP) genes in archival LMS and DTF tumor specimens. HBP, a glucose metabolism pathway responsible for protein glycosylation, regulates oncogenes that promote tumor survival. While HBP activation has been observed in various cancers and its targeting shows therapeutic promise, its role in LMS and DTF cell lines remains undefined. This study investigates HBP activation in LMS and DTF cell lines and evaluates the therapeutic potential of targeting GFPT2, the first and rate-limiting HBP enzyme, using the inhibitors DON and azaserine. **Methods:** We used six LMS cell lines (SK-LMS-1, SK-UT-1, SK-UT-1B, LMS03, LMS04, LMS05) and the DES23 DTF line. GFPT2 expression was assessed via Western blotting. Cells were treated with DON and azaserine for 24 hours, and viability was measured using the CellTiter-Glo assay. **Results:** GFPT2 expression varied across lines, with LMS05, SK-UT-1B, and DES23 showing highest levels. GFPT2 inhibition significantly reduced viability in a dose-dependent manner. Notably, LMS05, SK-UT-1B, and DES23 required higher inhibitor concentrations. **Conclusions:** GFPT2 inhibition reduces cell viability and shows promise as a therapeutic strategy for LMS and DTF. Future studies will explore combining GFPT2 inhibitors with standard therapies to assess synergy and further define the therapeutic potential of targeting HBP in LMS and DTF.

LAY SUMMARY

There are no effective treatments for selected types of sarcoma: Leiomyosarcoma (LMS) and desmoid-type fibromatosis (DTF) are difficult-to-treat tumors, and better therapies are urgently needed. We found a metabolic vulnerability: These tumors rely on the hexosamine biosynthesis pathway (HBP), which helps cancer cells grow and survive. We targeted the first enzyme in this pathway (GFPT2) using two known inhibitors. Blocking GFPT2 reduced tumor cell growth: In our experiments, inhibiting GFPT2 decreased the viability of LMS and DTF cells, especially those with high GFPT2 levels. This suggests a new potential treatment strategy that could be combined with existing therapies.

Investigating the role of ZEB1 and ZEB2 in the subtype evolution and drug resistance of small cell lung cancer

- Poster #513

Presenter: Reshani Perera, University of Manitoba

Other Authors: Joel Pearson

ABSTRACT_ID#2500

ABSTRACT

Small cell lung cancer (SCLC) is highly aggressive, with a poor 5-year survival rate of <10%. While SCLC initially responds well to front-line chemotherapy, most patients relapse within months. Understanding the mechanisms underlying this relapse will highlight novel therapeutic opportunities for SCLC, unfortunately these mechanisms are unclear. SCLC is now classified into subtypes based on transcriptional regulators, with SCLC-A and SCLC-N being the most common. Importantly, these subtypes exhibit different therapeutic vulnerabilities, and evidence suggests tumors switch between subtypes under therapeutic pressure. Although the mechanisms driving subtype switching remain unclear, they likely involve transcriptional and epigenetic reprogramming. ZEB1 and ZEB2 are transcription factors driving epithelial-to-mesenchymal transition, cellular plasticity and therapy resistance. We observed that ZEB1 and ZEB2 are highly expressed in the SCLC-N, but largely absent in the SCLC-A, suggesting a unique role for ZEBs in SCLC-N. This led us to explore the role of ZEB1/2 in SCLC subtype evolution and drug resistance. We established a novel CRISPR interference system to knockdown ZEB1 and/or ZEB2, and lentiviral vectors for their overexpression. To assess ZEB1/2 function in SCLC-N, we are knocking down ZEB1 and/or ZEB2 in SCLC-N cells to analyze the impact on cell survival and identify ZEB target genes and mechanisms. To study ZEBs in subtype plasticity and drug resistance, we are overexpressing ZEB1 and/or ZEB2 in ZEB1/2-deficient SCLC-A cells to evaluate phenotypic shifts towards SCLC-N and/or changes in chemosensitivity. This study will define ZEBs role in SCLC and identify drivers in subtype evolution and drug resistance, guiding targeted therapies.

LAY SUMMARY

- One of the major challenges in small cell lung cancer (SCLC) is that it quickly stops responding to treatments and becomes drug resistant.
- This project studies whether two proteins, called ZEB1 and ZEB2, help SCLC develop this drug resistance and escape treatments by changing its form.
- By studying how ZEB1 and ZEB2 help SCLC to survive and change in response to treatments, we can find new ways to treat SCLC.

Role of Active Beta Catenin (ABC) in Osteosarcoma Invasion & Metastasis - Poster #514

Presenter: Saima Ghafoor, University of Alberta

Other Authors: Aarya Ghimire, Elizabeth Garcia, Daniel J. Jay, Sujata Persad

ABSTRACT_ID#2506

ABSTRACT

Introduction. Osteosarcomas are aggressive primary bone tumors with a peak incidence in children <18-yrs age. The 5-year event-free-survival (EFS) is 60%, EFS drops to <30% for those with metastatic disease, and <15% in patients with widespread disease. These numbers have remained static. There is an urgent need for identifying prognostic markers to facilitate risk stratification and identify patient groups in which novel therapeutic interventions are most needed. The Wnt/ β -catenin pathway plays a crucial role in skeletal development and is dysregulated in osteosarcoma. However, its role in osteosarcoma/osteosarcoma progression remains unknown. We investigated whether a novel alternate form of β -catenin, Active β -Catenin (ABC), promotes metastatic osteosarcoma and can serve as a prognostic marker for aggressive disease. We identified that high levels of nuclear ABC is associated with significantly worse overall survival. We also reported that cellular/nuclear ABC levels increase with the degree of aggressiveness in osteosarcoma. **Methods.** To validate ABC's role in osteosarcoma progression, we used a GFP-tagged-ABC. We investigated the direct role of ABC in osteosarcoma progression using 3D-cultures and an in-vivo model. **Results.** We determined ABC preferentially accumulates in the nucleus and is likely responsible for driving the transcriptional activity and invasive phenotype in these cells. 3D-cultures showed that spheroids expressing GFP-ABC exhibited significantly greater invasive phenotype than control. In-vivo studies showed the presences of osteosarcoma in the distal femoral bone marrow cavity, inflammation and erosion of the bones due to tumor infiltration mainly in mice injected with ABC- expressing cells. **Conclusion.** ABC promotes osteosarcoma progression to metastatic disease.

LAY SUMMARY

Osteosarcomas are bone tumors with peak incidence in children <18-yrs age. Five-year event-free-survival is 60% that drops to <30% for metastatic disease, and <15% with widespread disease. There is an urgent need for identifying prognostic markers to facilitate risk stratification and identify patient groups in which novel therapeutic interventions are most needed. Wnt/ β -catenin pathway plays a crucial role in skeletal development and is dysregulated in osteosarcoma. However, its role in osteosarcoma/osteosarcoma progression remains unknown. We investigated whether a novel alternate form of β -catenin, Active β -Catenin (ABC), promotes metastatic osteosarcoma and serve as a prognostic marker for aggressive disease. We identified high levels of nuclear-ABC associated with significantly worse survival. We reported cellular/nuclear-ABC levels increase with osteosarcoma aggressiveness. To validate ABC's role, we used GFP-tagged-ABC to investigate ABC role in osteosarcoma progression using 3D-cultures and in-vivo model. We determined ABC preferentially accumulates in nucleus and is likely responsible for driving transcriptional activity and invasive phenotype in these cells. 3D-cultures showed; spheroids expressing GFP-ABC exhibited significantly greater invasive phenotype. In-vivo studies showed presences of osteosarcoma in distal femoral bone-marrow-cavity, inflammation and erosion of bones due to tumor infiltration mainly in mice injected with ABC-expressing-cells. **Conclusion:** ABC promotes osteosarcoma progression to metastatic disease.

The role of cholinergic signaling in lung cancer brain metastasis - Poster #515

Presenter: Ximena Mejia Delgadillo, University of Saskatchewan

Other Authors: Lara New, Anand Krishnan

ABSTRACT_ID#2665

ABSTRACT

Background Lung cancer (LC) is the leading cause of cancer-related death worldwide. LC dissemination to the brain [Lung cancer brain metastasis – LCBM] increases the risk of LC associated mortality. Effective therapies for LCBM are limited due to the inability of drugs to cross the blood-brain barrier, and due to lack of knowledge of therapeutic targets. Pre-clinical studies showed that cholinergic signaling promotes LC progression and metastasis, however, the role of cholinergic signaling in LCBM has not been explored. **Methods** In this study, we quantified the expression of cholinergic receptors CHRM1, CHRM3 in human primary LC samples from patients who developed or were protected from LCBM. The effect of CHRMs on cell proliferation, migration and invasion in vitro was examined using the LC lines NCI-H1993 and NCI-H2009. The cells were treated with natural/synthetic agonists (acetylcholine, carbachol, xanomeline) of CHRMs. In some experiments, the cells were pretreated with antagonists (pirenzepine, darifenacin) to confirm the role of CHRMs on cell dynamics. For mechanistic investigation, YAP1, a known promoter of LCBM, was examined in human samples and in vitro cultures downstream CHRMs' interventions. **Results** We found upregulation of CHRM1 and CHRM3 in primary LC samples of patients who developed LCBM. In vitro results showed that both CHRM1 and CHRM3 modify the invasive capabilities of LC cells. We also found YAP1 activation downstream of CHRMs interventions in vitro, confirmed with patient samples overexpressing CHRMs. **Conclusion** Our results indicate that cholinergic signaling facilitates LCBM, suggesting that CHRM1 and CHRM3 are potential therapeutic targets for LCBM.

LAY SUMMARY

Lung cancer (LC) is the leading cause of cancer-related death in men and women worldwide. Lung cancer dissemination to the brain (lung cancer brain metastasis - LCBM) significantly influences LC-mortality, and about 50% of LC patients develop LCBM. Cholinergic receptors respond to acetylcholine (ACh), a neurotransmitter that modulates biological and physiological functions throughout the body. Pre-clinical studies showed that cholinergic signaling promotes LC progression and metastasis. Nevertheless, the role of cholinergic signaling in LCBM requires further investigation. Our research indicates that cholinergic signaling promotes LCBM, suggesting that cholinergic receptors M1 and M3 are potential therapeutic targets for LCBM.

Discovery of Natural Products With Distinct Anti-mitotic Activities from Native Canadian Plants - Poster #518

Presenter: Tanner Lockwood, University of Lethbridge

Other Authors: David Williams, Raymond Andersen, Roy Golsteyn

ABSTRACT_ID#2911

ABSTRACT

Botanical species from Canadian ecozones produce unique natural product chemicals that enable them to survive harsh abiotic conditions and herbivory. We have generated one of the largest annotated collections of extracts from these species and identified compounds that induce mitotic arrest in human cancer cell lines. In this study, we investigated anti-mitotic natural products from *Arnica cordifolia* (heartleaf arnica), a Canadian species related to the European medicinal plant *Arnica montana*. Extracts prepared from *A. cordifolia* induced rounded cell morphology and arrested cells in the G2/M phase of the cell cycle without affecting mitotic spindle morphology, a phenotype unlike that of any mitotic inhibitor previously identified by our laboratory. Using biology-guided fractionation, five anti-mitotic sesquiterpene lactones were identified: three novel structures, and the known compounds aromaticin and pulchellin-2 α -O-isovalerate. These compounds all induced rounded cells positive for phospho-histone H3, yet had distinct effects on mitotic spindle morphology. Furthermore, aromaticin treatment formed ubiquitin foci in cells, suggesting that it may target the ubiquitin-proteasome pathway. This is the first report of three novel mitotic inhibitors, of the anti-mitotic activities of aromaticin and pulchellin-2 α -O-isovalerate, and of the effect of aromaticin on ubiquitin. Differences in the structures and biological activities of these anti-mitotic sesquiterpene lactones suggest that they act by distinct cellular targets and mechanisms of action. The discovery of novel mitotic inhibitors from Canadian plant species enhances our understanding of mitosis and may identify new targets for cancer treatments.

LAY SUMMARY

Arnica cordifolia is a botanical species from the Asteraceae family native to Alberta, Canada. It is related to the European medicinal plant *Arnica montana*. Extracts prepared from *A. cordifolia* induced rounded cell morphology in human cancer cell lines and arrested these cells in mitosis without affecting mitotic spindle morphology, a phenotype unlike that of any mitotic inhibitor previously identified by our laboratory. We isolated five anti-mitotic sesquiterpene lactones from *A. cordifolia* leaves by biology-guided fractionation. Three novel compounds were discovered, as well as the known compounds aromaticin and pulchellin-2 α -O-isovalerate. These compounds induced mitotic arrest with distinct effects on the mitotic spindle. Furthermore, aromaticin treatment formed ubiquitin foci in human cancer cells. This is the first report of the anti-mitotic activities of these compounds and of the effect of aromaticin on ubiquitin. Differences in the structures and biological activities of these anti-mitotic sesquiterpene lactones suggest that they act by distinct cellular targets and mechanisms of action.

Investigating PROX1 and NKX2-1 in TEAD-regulated Small Cell Lung Cancer - Poster #519

Presenter: Bea Ricarte, University of Manitoba

Other Authors: Joel Pearson

ABSTRACT_ID#3261

ABSTRACT

Lung cancer is the leading cause of cancer-related deaths globally. Among the two subtypes, Small Cell Lung Cancer (SCLC) is the most aggressive with 5-year survival rates <10%. Despite its severity, standard therapy for SCLC has remained the same for >30 years, highlighting a need for new therapeutic strategies. In contrast to Non-Small Cell Lung Cancer (NSCLC), which has been stratified into molecular subtypes that enabled development of targeted therapies, SCLC is treated as a single disease. However, recent research revealed that SCLC is more heterogenous than previously thought, and can be subdivided into several molecular subtypes: SCLC-A, SCLC-N, SCLC-P, SCLC-Y and SCLC-I, with SCLC-A being the most prevalent. Each subtype exhibits distinct characteristics that could unveil subtype-specific vulnerabilities. Our lab uncovered an essential role for TEAD transcription factors in SCLC and found that TEADs associate with specific homeobox proteins in the SCLC-A subtype. This suggested that these homeobox proteins may mediate the unique function of TEADs in SCLC-A. We validated the homeobox transcription factors PROX1 and NKX2-1 as TEAD-interacting partners in SCLC-A and found that they regulate various TEAD target genes in these cells. This suggests that PROX1 and NKX2-1 are key TEAD partners in the SCLC-A subtype. Building on these findings, we propose that PROX1 and NKX2-1 are essential partners of TEADs in the SCLC-A subtype. We aim to dissect the regulatory processes that underlie the unique biology of the SCLC-A subtype. Ultimately, this could contribute to the development of targeted therapeutic strategies to improve outcomes in SCLC.

LAY SUMMARY

- Small Cell Lung Cancer (SCLC) is the most aggressive lung cancer subtype, with low survival rates and high therapeutic resistance. Additionally, standard treatment has remained the same over the past 30 years, highlighting the need for new therapeutic strategies.
- Unlike its other subtype, Non-Small Cell Lung Cancer (NSCLC), SCLC was only recently recognized as heterogeneous with various molecular subtypes (SCLC-A, SCLC-N, SCLC-P, SCLC-Y and SCLC-I), each consisting of distinct therapeutic targets.
- Our lab found that TEAD transcription factors interact with homeobox proteins PROX1 and NKX2-1 to regulate key genes in SCLC-A, the most prevalent subtype of SCLC. Dissecting this pathway could reveal targets for subtype-specific treatment.

Clonal hematopoiesis of indeterminate potential may protect against radiation-induced toxicities in breast cancer patients - Poster #520

Presenter: Tirthankar Ray, United Health Network

Other Authors: Madison Hossack, Pierre-Antoine Bissey, Justin Williams, Wei Shi, Sagi Abelson, Steven Chan, Kenneth Yip, Fei-Fei Liu

ABSTRACT_ID#3055

ABSTRACT

Introduction: A common consequence of radiation therapy (RT) is fibrosis and/or lymphedema. A potential driver of these toxicities is clonal hematopoiesis of indeterminate potential (CHIP), an age-associated clonal expansion of mutated hematopoietic stem cells (HSCs) (e.g., Tet2). Because CHIP has been previously linked to chronic fibrotic states, we hypothesized that breast cancer patients with CHIP are at higher risk of RT-induced toxicities. Methods: DNA was extracted from breast cancer patient blood samples prior and 1 month post RT, and CHIP was detected through next-generation smMIP sequencing. CHIP status was cross-referenced with radiation-induced fibrosis and/or lymphedema clinical data. Additionally, a CHIP mouse model was generated by transplanting Tet2 heterozygous (+/-) HSCs into myelo-ablated mice. Mouse hindlimbs irradiated at 20 Gy were evaluated through joint angle measurements to study fibrosis progression. 16 weeks post-RT, skin samples were collected to assay dermal thickness and collagen deposition. Results: Amongst 138 breast cancer patients, 28 were CHIP-positive but only 1 developed radiation-induced fibrosis/lymphedema. Amongst the remaining 110 CHIP-negative patients, 24 developed fibrosis/lymphedema, demonstrating a significant association between toxicity outcomes with CHIP-negative patients (Fisher's exact test, $p < 0.05$). This was reflected in vivo, wherein CHIP-positive mice (carrying Tet2 +/- HSCs, $n=9$) exhibited reduced fibrosis, thinner skin (27% decrease), and less collagen (51% decrease) post-RT, compared to controls ($n=9$). Conclusion: Our findings unexpectedly demonstrated that patients with CHIP displayed a lower risk of fibrosis/lymphedema. These findings will be validated in other clinical cohorts and preclinical models to further understand the possible protective effects of CHIP against radiation-induced fibrosis.

LAY SUMMARY

Half of all cancer patients receive radiation therapy, but this can lead to the development of toxicities like fibrosis or lymphedema and these risks limit the doses that patients can receive. Some patients with unique driver mutations (normally associated with age) in their bone marrow appear less likely to develop these radiation-induced toxicities, suggesting that these mutations may protect against radiation therapy side effects. Our development of a fibrosis mouse model that mimics these bone marrow mutations shows preliminary results that match our findings in the clinical cohorts, wherein mice with these mutations show less severe fibrosis.

Isolation of Candidate Glioblastoma-Initiating Cells from the Cerebral Cortex of the Adult Mammalian Brain - Poster #521

Presenter: Sarah Sait, University of Calgary

Other Authors: Mathieu Meode, Hiba Omairi, Sophie Black, Gregory Cairncross

ABSTRACT_ID#2578

ABSTRACT

Glioblastoma multiforme (GBM) is an aggressive incurable brain cancer of older adults that arises in cortical and subcortical brain regions. Despite standard treatments, the median survival time remains 15 months. Current thinking suggests that GBMs arise from neural stem cells (NSCs) that persist in the postnatal brain, but whether such cells exist in cortical regions of the aging brain — where GBMs occur — remains unknown. In adults, NSCs are thought to reside in neurogenic regions, principally the subventricular zone (SVZ), located remotely from the site-of-origin of GBMs. To address this discrepancy, we sought to investigate whether NSCs exist in the cerebral cortex of the mammalian brain as it ages and whether such cells could give rise to GBMs. Here, we take advantage of two established model systems: first, a technique to culture dormant NSCs from the SVZ, and a second to transform p53-null NSCs into GBM-like cells. We demonstrate for the first time that quiescent NSCs can be isolated from the murine cerebral cortex using Platelet-Derived Growth Factor C (PDGFC). These SOX2+/Nestin+ cells form small minimally enlarging spheres that display a stem-like behaviour. We then demonstrate that p53-null cortical NSCs isolated using PDGFC become exogenous growth factor independent (i.e., tumorigenic) during chronic exposure to Platelet-Derived Growth Factor A. Overall, we show that NSCs capable of malignant transformation into GBM-like cells exist in the adult mammalian cerebral cortex. These results are consistent with the hypothesis that human GBMs arise peripherally in the brain from dormant NSCs that persist during aging.

LAY SUMMARY

- Neural stem cells (NSCs), a putative cell-of-origin of the incurable brain cancer glioblastoma (GBM), can be isolated from the cerebral cortex of young adult and elderly murine brains. Prior to this study, NSCs were only thought to exist in neurogenic regions postnatally, namely the subventricular zone and subgranular zone in the brain.
- NSCs isolated from the cerebral cortex assume a dormant state when cultured in Platelet-Derived Growth Factor C (PDGFC), and express NSCs markers. These cells form small spheres that do not significantly grow in size over 100 days, behaving like stem cells in the adult brain. This suggests that PDGFC provides a more authentic culture condition for NSCs compared to the gold standard Epidermal Growth Factor/Fibroblast Growth Factor condition, which causes cells to proliferate rapidly.
- Using a previously published model of GBM, we show that cortical NSCs, when p53-null, transform into growth factor independent GBM-like cells in Platelet-Derived Growth Factor A. These results are consistent with the hypothesis that GBMs might arise from NSCs resident in non-neurogenic regions of the adult brain including the cerebral cortex. These observations help address the concern that GBMs typically arise in areas of the adult brain thought to be deficient in stem cells.

Decoding the structure of a viral RNA essential for KSHV replication and tumourigenesis - Poster #522

Presenter: Tithi Ghosh, University of Calgary

Other Authors: Mariel Kleer, Jennifer Corcoran

ABSTRACT_ID#2637

ABSTRACT

Kaposi's sarcoma-associated herpesvirus (KSHV) is the etiological agent of Kaposi's sarcoma (KS), a malignancy predominantly affecting immunocompromised individuals. Unlike other tumour viruses, KSHV replication is essential for KS tumourigenesis. Kaposin, the most abundant viral transcript in KS tumors, is a polycistronic mRNA encoding three kap proteins and composed primarily of two sets of GC-rich 23-nucleotide direct repeats, DR1 and DR2. Previously, kaposin was viewed as a protein-coding mRNA; however, our lab's recent findings have redefined kaposin as a non-coding RNA retained in nuclear speckles (NSs) and largely unavailable for translation. In this way, kaposin acts like an architectural non-coding RNA, re-positioning NSs beside the KSHV DNA genome to optimize viral infection. The DR1 sequence is required for kaposin co-localization with the NS scaffolding protein, SRRM2; however, it remains unclear whether a specific nucleotide sequence or certain RNA secondary structure is responsible for kaposin-SRRM2 co-localization. The RNA structure prediction tool, RNAstructure, suggests that each 23-nucleotide DR1 repeat forms a stem-loop structure. I hypothesize that this stem-loop structure acts as a recognition motif to recruit SRRM2 to the kaposin RNA. To test this, I am using in vitro SHAPE and in vivo DMS-MapSeq to determine secondary structure of kaposin at single-nucleotide resolution. If each DR1 repeat indeed forms a stem-loop, I will introduce individual nucleotide substitutions to disrupt the stem-loop, validate the disruption using SHAPE, and evaluate the loss of NS co-localization via IF-FISH. Characterising the features of DR1 will reveal how precisely kaposin optimizes KSHV infection to promote replication and tumorigenesis.

LAY SUMMARY

- A cancer-causing virus rewires cells with RNA, not just proteins: Imagine a piece of RNA could rewire the control center of your cells. That's kaposin, an RNA made by Kaposi's sarcoma-associated herpesvirus (KSHV). Instead of making proteins, kaposin reshapes nuclear speckles, a hub that control RNA processing to help the virus thrive and promote cancer.
- Kaposin RNA shape with big impact: Identifying kaposin RNA's specific shape is like a VIP (Viral Invitation Party) pass addressed to certain proteins so the virus can throw a takeover party inside the host cell by boosting infection.
- Targeting RNA shape could lead to new cancer therapies: We are trying to identify unique shape of kaposin that may be essential for its cancer-promoting effects. Disrupting these RNA shapes could offer a new strategy to stop KSHV-related cancers at the primary infection stage by preventing it from setting up the cell's nucleus to support cancer.

Targeting the neutrophil extracellular trap (NET)-induced coiled-coil domain containing 25 (CCDC25)-integrin linked kinase (ILK) interaction as a therapeutic strategy in pancreatic cancer cells - Poster #523

Presenter: Sam Bakteria, BC Cancer Research Center

Other Authors: Shannon Awrey, Hossein Tavakoli, Paul McDonald, Shoukat Dedhar

ABSTRACT_ID#3064

ABSTRACT

Introduction: Pancreatic ductal adenocarcinoma (PDAC) remains one of the most lethal and difficult-to-treat cancers, with a 5-year survival rate of 9 percent. Emerging research highlights the role of neutrophils in promoting tumor progression through the formation of neutrophil extracellular traps (NETs). Our lab found that NETs, composed of modified DNA, histones, and proteases, activate integrin-linked kinase (ILK) in PDAC cells via the transmembrane receptor coiled-coil domain containing 25 (CCDC25), which detects extracellular NET-DNA. Given ILK's role in cytoskeletal reorganization, which drives metastasis and invasion, disrupting this pathway may offer a therapeutic strategy. **Objective:** This research investigates the specific binding domains between CCDC25 and ILK, enabling the design of targeted inhibitors that disrupt this interaction. **Methodology:** Peptide arrays were synthesized using SPOT synthesis to mimic CCDC25 fragments and incubated with recombinant ILK to identify critical binding regions. TALEN gene editing is being used to mutate these regions in mouse KPCY PDAC cells. Co-immunoprecipitation and Western blot analyses will assess the impact of these mutations on ILK recruitment and signaling. Additionally, novel cell-penetrating peptide inhibitors will be designed to competitively block CCDC25-ILK binding, leveraging expertise in peptide chemistry. **Results:** Through using peptide arrays, the binding between CCDC25 and ILK was narrowed down to a 20-amino-acid region within the intracellular domain of CCDC25, where hydrophobic and electrostatic interactions play a key role. This information will be used for inhibiting the CCDC25-ILK interaction to counteract the tumor-promoting effects of NETs without compromising neutrophil immune function, offering a novel therapeutic avenue for PDAC patients.

LAY SUMMARY

Pancreatic cancer is one of the deadliest and most difficult to treat cancers. We have learned that inflammation in the tumour environment, especially the buildup of immune cells called neutrophils, play a key role in how the disease progresses. Neutrophils release a special kind of DNA called NET-DNA that interacts with cancer cells, making them more invasive and helping them grow faster. This NET-DNA binds to a protein on pancreatic cancer cells called coiled-coil domain containing 25 (CCDC25), which then activates another protein, integrin-linked kinase (ILK), known to drive cancer progression. Our lab used a high-throughput method to map and pinpoint where ILK binds to on CCDC25. Currently, we have narrowed down to two potential binding regions and are now validating them using a gene-editing technology called TALEN. Once validated, this information can be used to develop new treatments that block the NET-DNA-CCDC25-ILK pathway and help prevent the cancer from spreading.

Amphiregulin regulates genomic integrity by increasing nuclear prelamin A in mammary epithelial cells to both maintain and augment heterochromatin formation in response to replication stress - Poster #524

Presenter: Tianqi Jiang, University of Ottawa

Other Authors: Aiman Zein, Christine Pratt

ABSTRACT_ID#2711

ABSTRACT

The EGFR ligand, amphiregulin (AREG) is a key growth factor during mammary gland development important for ductal cell differentiation and ductal elongation, however we and others have found that AREG is often localized in the nucleus of mammary epithelial cells (MECs) in histological sections. Using immortalized MECs, we have discovered that AREG undergoes retrograde trafficking to the nuclear membrane (nAREG) in close proximity with lamin A where it is required to both maintain constitutive heterochromatin and transiently increase heterochromatin in response to replication stress (RS). Both RS and expression of a C-terminal truncated, nuclear trafficked AREG(Δ C11) increased nuclear prelamin A, the H3K9 histone methyltransferase SUV39h1 and heterochromatin protein, HP1 α . In media supplemented with AREG, siRNA-mediated depletion of endogenous AREG reduced SUV39h1 and HP1 α proteins accompanied by decompaction and reduction in global H3K9me3 heterochromatin. The nuclear membrane (NM) was also impacted resulting in dissipation of the Ran gradient, reduced matrix lamin A and increased invagination. Moreover, loss of AREG slowed replication fork speed, increased new replication origins and increased global transcription while promoting and exacerbating DNA damage in response to RS. In BRCA2mut/+ MECs, AREG depletion exacerbated the effect of double-strand DNA breaks and induced senescence following RS, indicating that AREG is required to maintain genomic stability and enable continued proliferation under stress conditions. Overall, this study reveals a noncanonical but crucial role for AREG in heterochromatin maintenance and the response to RS, most critical in BRCA2mut/+ MECs.

LAY SUMMARY

The non-canonical role of AREG inside the nucleus is not well understood. Recent studies have linked nuclear AREG to early stages of cancer, including abnormal cell growth. In our work, we found that cells with BRCA2 mutations often have AREG in the nucleus. This was seen in our tissue staining of both mouse and human mammary glands sections. Nuclear AREG plays a role in shaping the epigenetic landscape, particularly in the maintenance and propagation of condensed heterochromatin. Loss of AREG leads to the depletion of heterochromatin marks, and promoting increased transcriptional activity. In the context of BRCA2-deficient cells, this transcriptional alteration may exacerbate cellular stress, contributing to genomic instability. Nuclear AREG plays a critical role in mediating DNA damage repair. Its absence sensitizes mammary epithelial cells to double-strand DNA breaks and halts cell growth following cellular stress. Mechanistically, we show that p38 (stress-activated protein kinase) promotes the nuclear translocation of AREG, and that inhibition of p38 disrupts AREG nuclear import. Thus, targeting nuclear AREG localization through p38 inhibition may offer a therapeutic strategy to selectively sensitize BRCA2mut/+ pre-cancerous cells to intrinsic DNA damage and prevent their continued proliferation.

Investigating Myt1 kinase as a link between lipid metabolism and cancer drug resistance - Poster #525

Presenter: Samantha Blackwood, University of Alberta

Other Authors: Hetagna Modi, Shelly Braun, Julia Nickols, Richard Fahlman, Gordon Chan, Armin Gamper

ABSTRACT_ID#3203

ABSTRACT

In recent years, cervical cancer (CC) has shown the highest increase among gynecological cancers in Canadian women, yet treatments for locally advanced CC have remained largely unchanged for decades, with a poor 5-year survival rate of around 30%. Kinase inhibitors are a promising class of anti-cancer drugs for CC, but our previous work showed that cervical cancer cells overexpressing the kinase Myt1 can develop resistance to these therapies. Altered lipid metabolism, such as increased fatty acid uptake and lipid droplet (LD) formation, is common in CC and has also been linked to cancer progression and drug resistance. We recently found that Myt1 co-localizes with LDs and that fatty acid supplementation can regulate Myt1 activity. However, the role that Myt1 may have in the interplay between lipid metabolism and drug resistance is unclear. To investigate this, we used a proximity-labelling approach to characterize Myt1's protein interaction network under steady-state and LD-inducing conditions. Among 45 candidate partners, PPP1R15B showed a heightened interaction with Myt1 under LD-inducing conditions. We validated PPP1R15B as a novel Myt1 interactor in CC cells using co-immunoprecipitation. While we continue to study the function of this interaction, we show that CC cells are more sensitive to dual inhibition of Myt1 and PPP1R15B compared to single inhibition. These findings suggest that co-targeting Myt1 and PPP1R15B could enhance cancer cell killing and overcome drug resistance in cervical cancer.

LAY SUMMARY

High levels of Myt1 kinase or altered fat metabolism have been shown to contribute to drug resistance in cancer cells. Myt1 kinase and PPP1R15B interact in cervical cancer cells, especially during lipid droplet-inducing conditions. Inhibiting both Myt1 and PPP1R15B simultaneously may be more effective in killing cervical cancer cells than targeting either one alone, offering a promising new treatment strategy to overcome drug resistance.

Molecular and Cellular Biology

Monday, November 3, 2025

An introduction to BEST4 cells and their development - Poster #526

Presenter: Tori McCabe, University of Alberta

Other Authors: Reegan Willms, Lena Jones, Edan Foley

ABSTRACT_ID#2504

ABSTRACT

The intestine is comprised of specialized epithelial cells that work in concert to maintain homeostasis and prevent the development of diseases such as colorectal cancer. Most cells have established functions and roles in disease, however newly identified bestrophin4 (BEST4) cells are a mystery in gut homeostasis. Recent studies on patient data identified BEST4 as a factor in colorectal cancer development and survival. Attempts to study BEST4 cells in vivo have been impeded by their absence in mice. Zebrafish are an excellent human intestinal model organism due to their structural similarity, genetic homology and malleability, and the cellular populations that comprise the intestinal epithelial layer as we previously described. This includes discovering BEST4 cells with comparable genetic profiles to their human counterparts in both larval and adult zebrafish. By studying the development and function of a newly discovered cell, we can identify the role they play in overall intestinal health and potential areas where errant differentiation may influence colorectal cancer. We used a combination of genetics, small molecule assays, imaging, and quantitative biology to examine the development of BEST4 cells in zebrafish. We found two key pathways for BEST4 cell differentiation, TNFRSF11A and Notch. By establishing avenues of BEST4 cells specification, we provide insight into an otherwise unknown cell. In future, we will identify the functional role of BEST4 cells using genetics and targeted cell ablation techniques followed by examining the consequences of intestinal homeostasis physiologically, environmentally, and the impact on gene expression throughout the gut.

LAY SUMMARY

BEST4 cells are a recently identified population of intestinal epithelial cells BEST4 cells rely on Notch and TNFRSF11A for development

SLK suppresses HER2/Neu-positive tumorigenesis by maintaining epithelial integrity and delaying expansion of SOX10-positive cells - Poster #527

Presenter: Samuel Delisle, University of Ottawa/Ottawa Hospital Research Institute

Other Authors: Luc Sabourin

ABSTRACT_ID#2773

ABSTRACT

Breast cancer accounts for approximately 12% of diagnosed carcinomas. HER2, a membrane-bound growth factor receptor which is overexpressed in approximately 25% of cases, leads to a more aggressive prognosis. Although HER2-targeted therapies have improved clinical outcomes, resistance to these treatments remains common, indicating a need for alternative therapeutic targets. Ste20-like kinase (SLK) is activated downstream of HER2 and has been shown to play a role in embryogenesis, cytoskeletal remodeling, and epithelial-mesenchymal transition. Conditional SLK knockout in a murine model for HER2-positive breast cancer (MMTV-NIC) accelerated tumor onset. SLK deficiency led to the induction of Sox10, a marker of triple-negative breast cancer and driver of stem/progenitor activity in mammary cells. Furthermore, conditional Sox10 knockout in MMTV-NIC transgenic mice failed to form tumors altogether. As such, obtaining a deeper understanding of Sox10 regulation and what induces SOX10-positive sub-population expansion could uncover new therapeutic targets. Using a novel kinase-dead (\$\$SLK^{K63R}\$\$) knock-in allele, we show that SLK kinase inhibition does not accelerate tumor onset nor Sox10 induction in MMTV-NIC transgenic mice. Furthermore, Sox10 induction in SLK-deficient mice is accompanied by breakdown of cell-junctions and mis-localization of apicobasal polarity proteins. Interestingly, although SOX10 expression is almost always lost in 2D cultures, it is consistently maintained in primary organoid models, suggesting that SOX10 marks a specific cell identity/state that is poorly maintained in 2D-growth. Taken together, our results support a phospho-independent role for SLK as a tumor suppressor, wherein, by maintaining epithelial integrity SLK delays the expansion of a Sox10-positive sub-population responsible for tumor initiation.

LAY SUMMARY

HER2-positive breast cancer is an aggressive subtype of breast cancer characterized by cancer cells with abnormally high levels of a protein called HER2. This protein accumulates on the surface of the cell where it transmits signals that promote growth. SLK is a protein that relays these signals inside the cell by adding chemical groups (or tags) to other proteins, activating, or inactivating them. When we eliminated SLK from a mouse with HER2-positive breast cancer, tumors presented sooner. Eliminating SLK caused a different protein, called SOX10, to become abundant. SOX10 is such a strong tumor-promoting protein that without it tumors will not form. Our goal is to better understand how SOX10 levels are controlled by the cells. Interestingly, SLK can also control the activity of proteins without tagging them. Does SLK need its tagging function to control SOX10? We test this by editing the SLK gene to shut off its ability to tag other proteins. Results showed that SLK controls SOX10 levels without tagging proteins. When cells break apart from each other and become less organized they turn on SOX10. SLK keeps the cells neatly packed together. By understanding how SOX10 is controlled, we can develop drugs that prevent its activation.

Soluble CD109 antagonizes EGFR-mediated pro-tumorigenic signaling and functional responses in head and neck squamous cell carcinoma - Poster #528

Presenter: Varsha Reddy Durgempudi, McGill University

Other Authors: Tenzin Kungyal, Anie Philip

ABSTRACT_ID#2677

ABSTRACT

Introduction: Multiple cell-signaling pathways have been dysregulated in SCC, including that of epidermal growth factor receptor (EGFR). Our group has shown that CD109 (a membrane-anchored protein) promotes tumor growth and metastasis in vivo, by activating EGFR signaling. Interestingly, we also showed previously that membrane CD109 (mCD109) is endogenously released from the cell surface; however, the function of released/soluble form of CD109 (sCD109) remains poorly understood. In the current study, we examined whether sCD109 regulates mCD109/EGFR-mediated pro-tumorigenic signaling in HNSCC progression. Objectives: (1) Does sCD109 regulates mCD109-EGFR interactions? (2) Does sCD109 modulates in vitro tumorigenicity? (3) Does sCD109 regulates angiogenesis tube formation? (4) Does sCD109 alter phospho-proteome? Results: (1) HNSCC cells treated with/without sCD109 recombinant protein in the presence/absence of EGF for 15 mins were processed using proximity ligation assay which showed that sCD109 binds with EGFR thereby inhibiting mCD109-EGFR interactions. (2) HNSCC cells allowed to form 3D spheroids in ultra-low attachment plates were treated accordingly for six days wherein we observed that sCD109 inhibits in vitro tumorigenicity. (3) HUVEC cells cultured on solidified Matrigel, were analyzed after 18 hours of treatment, and we observed that sCD109 inhibits angiogenesis tube formation. (4) Phospho-proteome array was used as per the Biotechne-protocol and our findings demonstrated that sCD109 inhibited EGF-induced AKT, ERK, p53 and GSK3 α/β activation. Conclusions: While our team has previously shown that mCD109 is pro-tumorigenic, our current findings suggest that sCD109 opposes pro-tumorigenic effects of mCD109. Together, our findings provide critical insights into understanding of molecular processes driving HNSCC progression.

LAY SUMMARY

Head and Neck Squamous cell carcinoma (HNSCC) is a type of cancer that originates from squamous cells in various organs, particularly in the head and neck region, such as the mouth, throat, and voice box. A protein called CD109, found on the surface of cancer cells, has been found more abundantly in HNSCCs and its increased presence in early abnormal growths is associated with a higher risk of cancer spreading to other parts of the body. Interestingly, CD109 can be released from the cell surface into the surrounding environment. The released form of CD109, referred to as soluble CD109 (sCD109), has been found (in our current study) to inhibit the tumor-promoting effects of its membrane counterpart, mCD109. In laboratory experiments, soluble CD109 blocked the signaling of a protein called EGFR, which is often overexpressed in cancer growth, by binding to EGFR with high affinity. Intriguingly, soluble CD109 could block the cancer cell's ability to form 3D tumor spheroids and nutrients-supplying new microvascular blood vessels formation. Additionally, soluble CD109 also inhibited key markers of cancer progression (such as AKT, ERK). Overall, our findings suggest sCD109 provide critical insights into understanding of molecular processes driving HNSCC progression, potentially improving patient outcomes.

p53-associated de novo activation of VWF expression in tumor cells - Poster #529

Presenter: Noorossadat Seyyedi, University of Alberta

Other Authors: Chu Shiun Lo, Parnian Alavi, Manijeh Pasdar, Nadia Jahroudi

ABSTRACT_ID#2354

ABSTRACT

INTRODUCTION. Expression of pro-coagulant protein von Willebrand Factor (VWF) under physiological conditions is exclusively restricted to endothelial cells and megakaryocytes. However, its de novo expression is detected in some cancer cells of non-endothelial/megakaryocytic origin and may promote their metastatic potential. We have previously demonstrated VWF expression in some cancer cells, with the transcriptional repressor NF-IB and activator GATA6 implicated in its regulation. NF-IB is a known target of tumor suppressor p53, one of the most frequently mutated genes in cancers. We have previously shown that the dual adhesion and signalling protein plakoglobin (PG) can restore the tumor suppressor function of some p53 mutants. We investigated whether mutant p53 contributes to the de novo activation of VWF expression in cancer cells, and if so, the underlying mechanism of its action. **METHODS/RESULTS.** Database analyses of 100 VWF-expressing cancer cell lines showed that VWF expression is associated with either NF-IB mutation (53%) and/or p53 mutations (~66%). To directly determine the role of mutant p53 in VWF expression, p53 and plakoglobin-null H1299 cells were transfected with p53- [WT, conformational, or contact mutants], with or without plakoglobin, and assessed for VWF, NF-IB, and GATA6 expression at the mRNA and protein levels. Specifically, expression of conformational mutant p53 induced VWF expression. It also disrupted NF-IB association with the VWF promoter, as shown by chromatin immunoprecipitation. Co-immunoprecipitation confirmed nuclear p53-NF-IB interaction. Plakoglobin co-expression reversed all these effects. **CONCLUSION.** Our study revealed a novel mechanism of de novo expression of VWF in cancer cells mediated by mutant p53.

LAY SUMMARY

Some cancer cells can produce a blood-clotting protein called von Willebrand Factor (VWF), which may help them spread in the body. Normally, this protein is produced by the cells lining blood vessels and by platelets, which assist in stopping bleeding, but surprisingly, we found it also in some cancer cells of different origins. Changes (mutations) in the p53 gene, which normally helps prevent cancer, can trigger VWF production in cancer cells by blocking the system that usually keeps it turned off. We found that a type of mutated p53, known as a conformational mutant, disrupts a repressor protein (NF-IB) that normally keeps the VWF gene inactive. Another protein, plakoglobin, can reverse the effects of mutant p53 and stop cancer cells from making VWF. This suggests that plakoglobin might help prevent cancer from spreading by restoring some of the lost control.

Defining common and context-specific protein sequences in the repair of double-strand breaks via non-homologous end-joining - Poster #531

Presenter: Shamika Shenoy, McGill University

Other Authors: Raquel Cuella-Martin

ABSTRACT_ID#2332

ABSTRACT

Safeguarding genomic integrity is paramount for maintaining cellular homeostasis. To achieve this, cells have evolved a complex network of tightly interconnected pathways collectively known as the DNA damage response (DDR). The DDR is a guardian for detecting and repairing a wide array of DNA lesions, including the double-strand breaks (DSBs). Upon sensing a DSB, a cascade of signalling events is initiated to mark the lesion site and recruit the necessary repair machinery. Notably, non-homologous end joining (NHEJ) machinery predominantly repairs DSBs in non-dividing cells and those in the G1 phase of the cell cycle. Additionally, NHEJ plays a crucial role in the programmed assembly of V(D)J gene segments in B- and T-cells and immunoglobulin class switch recombination in B-cells. The diverse nature of DSBs from blunt to staggered ends and in different contexts makes it crucial to identify these multifunctional NHEJ protein sequences involved in the repair. While extensive structure-function analyses of these genes have been conducted, only a few separation-of-function (SOF) mutations have been identified to date. This knowledge gap is particularly prominent in newly discovered NHEJ factors and proteins with expansive multidomain architectures. In this study, we propose a novel approach employing in situ deep mutagenesis with CRISPR-dependent base editors to elucidate structure-function relationships of NHEJ proteins. This systematic probing aims to unveil common and context-specific features governing DSB repair, potentially informing therapeutic strategies for preserving genomic integrity and addressing DNA damage-related diseases.

LAY SUMMARY

Our cells have a built-in “repair crew” to fix damage in our DNA and prevent harmful changes. One type of damage, called a double-strand break (DSB), is especially serious and needs quick and accurate repair to protect our genetic information. A key repair system, called non-homologous end joining (NHEJ), is active in many cell types and is especially important in the immune system, helping B and T cells develop properly. However, we still don’t fully understand how all the proteins in this system work especially the newer or more complex ones. In this project, we use gene-editing tools to make many tiny changes in NHEJ-related proteins directly inside living cells. This helps us see which parts of these proteins are essential for DNA repair and which aren’t. By mapping these functions more precisely, our research could improve our understanding of diseases linked to DNA damage and guide future therapies.

FAM72A promotes UNG2 degradation and mutagenesis in human cancer cells - Poster #532

Presenter: Yuqing Feng, York University

Other Authors: Philip Barbulescu, Chetan Chana, Melika Shirdarreh, Hong Yang, Lizhen Wu, Sami Mamand, Mohammad Kashem, Amin Zia, Ming Han, Julissa Tsao, Trevor Pugh, David Cescon, David Schatz, Frank Sicheri, Alberto Martin, Rossanna Pezo

ABSTRACT_ID#2543

ABSTRACT

Genetic lesions drive cancer development and progression, and understanding their origins will reveal the mechanisms of carcinogenesis. We showed that murine FAM72A promotes mutagenic DNA repair during antibody maturation by acting as a substrate adaptor of the CTLHMKLN1 E3 ligase to induce the proteasome degradation of Uracil DNA glycosylase 2 (UNG2), a pivotal enzyme of the base excision repair. In humans, the FAM72 gene has expanded to include four paralogues named FAM72A-D. Bioinformatic studies suggested that the human FAM72 genes are overexpressed in a broad range of cancers. However, the functional roles of FAM72A-D in human biology and cancer are unknown. Here, we show that FAM72 family members are minimally expressed in most healthy tissues except for thymus, and that FAM72A, B and D are overexpressed in primary tumorigenic tissues. Human FAM72 expression inversely correlates with UNG2 protein level in human cell lines and primary tumorigenic tissues suggesting that human FAM72 promotes UNG2 degradation. However, only FAM72A is able to bind to and induce UNG2 degradation in human cells. Our results suggest that the ability of FAM72A to induce UNG2 degradation contributes to neoplasia in a variety of cancer types by promoting mutagenic repair of genomic dUs.

LAY SUMMARY

1. Using a novel qPCR assay, we found that FAM72 paralogues are expressed at low levels in the majority of normal tissues, but highly expressed in human cancer cell lines and primary cancer tissues.
2. Similar to murine Fam72a, human FAM72 expression negatively correlates with UNG2 protein level in human cell lines and primary human tumours, and can promote mutations in a B cell cancer line.
3. Despite a high degree of sequence similarity, only FAM72A can bind to and induce UNG2 degradation.

Repeat Expansion in a Glycosyltransferase Gene: An Emerging Prognostic Factor in Kidney Cancer - Poster #533

Presenter: Shanna Ladouceur, Université Laval

Other Authors: Michèle Rouleau Rouleau, Lyne Villeneuve, Chantal Guillemette

ABSTRACT_ID#2444

ABSTRACT

Renal cell carcinoma (RCC) is the most common (85%) and aggressive form of kidney cancer. A recent study revealed a GAAA repeat expansion (RE) in the glycosyltransferase UGT2B7 gene, observed in 35% of RCC tumors. Repeat expansions are well-established contributors to neurological disorders, disrupting gene expression and offering pharmacological targets. Their role and pathogenic potential in oncogenic processes remain largely underexplored. Our objectives are to assess the impact of the expansion on the complex transcriptional landscape of the gene, to investigate its effect on the functional activity of the enzyme encoded by this gene, and to evaluate its associations with patient survival in RCC. Bioinformatics analyses, based on RNA sequencing and whole-genome sequencing data from RCC tumors, reveal that the presence of the repeat expansion alters the expression of the canonical and alternative UGT2B7 transcripts. Notably, some of these transcripts are associated with poor prognosis. Our investigations did not establish a link between the GPER axis, aldosterone, and the proliferation of RCC cells. This finding refutes one of our initial hypotheses, which proposed that the expansion disrupts aldosterone homeostasis, a known UGT2B7 substrate and a key regulator of the renin-angiotensin system. Our findings support that the expansion in the UGT2B7 gene modulates gene expression and may represent a promising prognostic marker in RCC. Ongoing clinical and functional studies aim to validate the prognostic value of this marker and its associated transcripts, and to further characterize the underlying molecular mechanisms, with the goal of evaluating its potential as a pharmacological target.

LAY SUMMARY

Renal cell carcinoma (RCC) is the most frequent and aggressive type of kidney cancer. A recent discovery identified an expansion of the repeated GAAA sequence up to more than 150 repeats in the DNA of 35% of RCC tumors. This genetic variation is located in the UGT2B7 gene, which encodes a metabolic enzyme with important functions in kidney homeostasis. The DNA repeat expansion may alter the function of this gene in kidney cancer cells. We observed that in RCC, the repeat expansion alters the expression of the UGT2B7 gene, favoring alternative versions. Some of these variants are associated with a more aggressive disease, suggesting that they could help predict how the disease will progress. Our findings support that the genetic variation in the UGT2B7 gene modulates its expression and may represent a promising prognostic marker in RCC. Our work will also help to understand the biology of RCC, and evaluate the therapeutic potential of the repeat expansion.

Cholesterol Dysregulation Enhances Viability and Migration in Ovarian Cancer Cells and Modulates Carboplatin Response - Poster #534

Presenter: Shuk On Annie Leung, Research Institute-McGill University Health Centre

Other Authors: Zahraa Qusairy, Yasaman Bagherian, Levana Xu, Xing Zeng, Basile Tessier Cloutier

ABSTRACT_ID#2879

ABSTRACT

Background: Cholesterol metabolism plays a pivotal role in cancer progression and treatment resistance. This study investigates the effects of exogenous cholesterol on cell viability and migration, both as a monotherapy and in combination with carboplatin, in ovarian cancer cell lines representing disease progression: PEO1, PEO4, and PEO6 that represent a spectrum from platinum sensitive to resistant disease. Methods: PEO1, PEO4, and PEO6 cells were treated with 1 µg/mL or 5 µg/mL cholesterol, with or without carboplatin (100 µM or 200 µM). Cell viability was assessed via standard metabolic assays, and migration was measured using wound healing assays. Statistical comparisons evaluated treatment effects across cell lines. Results: Cholesterol monotherapy significantly enhanced viability in all three-cell lines, with the most robust and consistent effects observed in PEO1. Co-treatment with cholesterol and carboplatin further increased viability compared to carboplatin alone, suggesting a cholesterol-mediated protective effect, most prominent in PEO1 and PEO4. Cholesterol also promoted cell migration in a dose-dependent manner across all lines, with PEO1 showing the strongest enhancement. While carboplatin inhibited migration, cholesterol partially reversed this effect, particularly in PEO1 and PEO4. PEO6 showed modest recovery. Conclusions: Exogenous cholesterol promotes ovarian cancer cell viability and migration and mitigates carboplatin-induced suppression in a cell line-specific manner. The most pronounced effects were seen in PEO1, suggesting a potential link between cholesterol dysregulation and chemoresistance. Future work will focus on identifying molecular biomarkers of cholesterol-mediated chemoresistance and elucidating related signaling pathways.

LAY SUMMARY

Cholesterol helps ovarian cancer cells grow and move: When ovarian cancer cells were exposed to cholesterol in the lab, they grew faster and moved more easily—two signs of more aggressive cancer behavior. Cholesterol may protect cancer cells from chemotherapy: Adding cholesterol made the cancer cells less sensitive to carboplatin, a common chemotherapy drug. This effect was strongest in cells that are usually more sensitive to treatment, suggesting cholesterol might contribute to drug resistance. Some cancer types are more affected than others: The PEO1 cell line, which is sensitive to platinum drugs, showed the most dramatic changes, highlighting the need to consider individual tumor characteristics when designing treatment strategies.

Pharmacological inhibition of LKB1-NUAK1 signalling in a spheroid model of ovarian cancer metastasis - Poster #535

Presenter: Sarah McArthur, University of Western Ontario

Other Authors: Yudith Ramos Valdés, Ahmed Abdelhameed, Babu Joseph, Methvin Isaac, Radek Laufer, David Uehling, Trevor Shepherd

ABSTRACT_ID#2700

ABSTRACT

Epithelial ovarian cancer (EOC) is a lethal gynecologic cancer, usually only diagnosed after widespread metastasis. EOC metastasis is unique, spreading through multicellular aggregates or spheroids. Previously, we demonstrated Liver kinase B1 (LKB1)-NUAK1 signalling promotes spheroid integrity, reattachment, and fibronectin production, in response to bioenergetic stress. Using genetics-based CRISPR deletion cell lines, we have solid evidence that LKB1-NUAK1 signalling plays crucial roles in EOC metastasis, therefore representing new therapeutic targets. In collaboration with the OICR, we have developed several novel small molecule LKB1-specific inhibitors; NUAK1 can be inhibited by the commercially-available compound ON123300/Narazaciclib. Based upon previous work, we hypothesize that pharmacologic inhibition of LKB1 will have a more significant impact on EOC metastatic potential as compared with NUAK1 blockade. Dose-response curves and spheroid reattachment assays were performed to evaluate LKB1-NUAK1 inhibition on spheroid cell viability and cell dispersion, respectively. Spheroid viability and dispersion following reattachment were reduced to a greater extent following treatment with several LKB1 inhibitors as compared with ON123300. Although ON123300 had little effect on spheroid cell viability, fibronectin expression was potently decreased in drug-treated spheroids resulting in reduced spheroid integrity that was distinct from LKB1 inhibitor effects. We are the first to develop and characterize potential LKB1 small molecule inhibitors, which represent important tools to provide new insights into LKB1-NUAK1 stress signalling as a crucial mediator during advanced EOC disease.

LAY SUMMARY

Two stress response proteins called LKB1 and NUAK1 are crucial for ovarian cancer cells to survive as clusters called spheroids during disease spread. Our lab has partnered with the Ontario Institute for Cancer Research to develop new chemical compounds that can block the function of LKB1; a drug compound called Narazaciclib is already available to target NUAK1. Using lab-based methods to study ovarian cancer metastasis, we show that blocking LKB1 with these new drug compounds has a more dramatic effect to kill ovarian cancer spheroids as compared with blocking NUAK1 using Narazaciclib.

Inhibitor-of-growth 5 (ING5) knockout affects stem cell development and the maintenance of tissues during natural aging - Poster #536

Presenter: Tom Bartizal, University of Calgary

ABSTRACT_ID#2648

ABSTRACT

Robust stem cells are essential throughout the bodies of humans and animals to sustain tissues and organs. The impact of stress and injury on tissues during biological aging results in the steady depletion or dysregulation of adult stem cell populations. This directly contributes to diminished functionality in tissues during advanced age and elevates the risk of various organ diseases and cancers. Multiple factors play a role in maintaining balance and controlling the rate of terminal differentiation in stem cell populations. In this context Inhibitor-of-Growth 5 (ING5), a member of the ING family of proteins known for their role as chromatin adaptors, is relatively understudied in specific tissues. ING5 has been proposed as a stemness factor influencing stem cell development and maintenance, owing to its significant expression in embryonic stem cells and its subsequent decline upon induced differentiation. Recently, ING5 has been linked to the maintenance of various adult stem cell populations, with its dysregulation apparent in cancer stem cells such as brain tumor initiating cells (BTICs). The depletion or dysregulation of stem cells is a significant contributor to several age-related disorders. Hence, by utilizing a recently developed ING5 knockout mouse model, our objective is to explore the role of ING5 in sustaining stem cell populations. This is a crucial step towards developing a comprehensive understanding of what factors influence deficiencies in stem cell pools and how these impact the functionality or health of tissues.

LAY SUMMARY

The loss of the inhibitor-of-growth 5 (ING5) has shown to affect different parts of development and long-term health. Earlier preliminary data showed a significant increase in lymphoma, dermatitis, increased apoptosis in testes, abnormal wound healing and differentiation, as well as increased expression of differentiation associated markers in the neural stem cell niche within the brain. Mutant animals are somehow affected at the early stages of fertilization or development. Size of ING5 knockout litters is reduced by half, with a skewed sex ratio favoring males. ING5 KO animals become progressively more susceptible to certain malignancies at old and advanced ages. Mutant animals have a higher incidence of lymphoma compared to wild-type animals, while only mutant females at advanced age show signs of pituitary adenoma.

The Discovery of Novel Neuroendocrine Prostate Cancer Biomarkers Utilizing a Proteomic Approach - Poster #537

Presenter: Unnati Yagnik, University of British Columbia

Other Authors: Syam Somasekharan

ABSTRACT_ID#3071

ABSTRACT

Neuroendocrine prostate cancer (NEPC) is an aggressive form of prostate cancer (PCa) with a poor survival rate of 14.3%. NEPC is marked by a cellular transformation to neuroendocrine phenotype and an increased expression of neuroendocrine markers. An earlier detection of NEPC is crucial for its timely intervention and prolong patient life; however, this is hampered by the limited number of biomarkers available to differentiate NEPC from other PCa types. We hypothesize that molecular profiling of PCa patient specimens at the proteomic level will uncover novel NEPC biomarkers. We performed Tandem Mass Tag (TMT)-labelled mass spectrometry to profile the proteome of nine PCa patient specimens. We discovered that one of the nine specimens expresses a significantly higher level of known neuroendocrine markers, CgA, CgB, and DDC; its NEPC features were further validated through immunohistochemistry. We identified a unique set of 39 proteins that are upregulated in this specimen by >2.5-fold change compared to all other specimens. We conducted pathway analysis and a database search which unveiled a set of novel proteins from this subset, including GRN, GNS, KCTD12, and THSD7A, that are associated with neuroendocrine phenotypes and have not been previously studied in NEPC. We intend to further validate these proteins by performing immunohistochemistry on a larger tissue microarray of NEPC and non-NEPC tumors to assess their sensitivity and specificity, thereby further characterizing them as potential biomarkers of NEPC.

LAY SUMMARY

Neuroendocrine prostate cancer (NEPC) is an aggressive form of prostate cancer with a 14.3% survival rate. Unfortunately, there are very few molecular markers that can be used in the clinic to detect the presence of NEPC, which has made it difficult to identify and treat this deadly disease in its early stages. By analyzing the molecular makeup of nine different prostate cancer samples, including one NEPC sample, we found 39 proteins that were unique to NEPC. From this group of 39, we identified four proteins in particular that are linked to neuroendocrine traits and processes; further, none of these four proteins have been previously studied in the context of NEPC. These four proteins hold potential to be utilized to identify the presence of NEPC. This could allow for earlier diagnosis and treatment, prolonging patient life and survival.

Investigating the role of the RNA-binding protein DEAD box ATPase 55 (DDX55) in castration resistant prostate cancer - Poster #538

Presenter: Ziwei Cai, Vancouver Prostate Centre

Other Authors: Syam Somasekharan, Eliana Beraldi, Devika Prakash, Jordan Lam, Elias Orouji

ABSTRACT_ID#2840

ABSTRACT

Background: Castration-resistant prostate cancer (CRPC) is an aggressive prostate cancer (PCa) form with a survival rate of less than two years. CRPC is difficult to manage and has very few treatment options available. CRPC mainly develops as a result of selective pressures of androgen deprivation therapy (ADT), a treatment given to ~50% of PCa patients. Our project focuses on identifying potential therapeutic targets for the improved management of CRPC and prolonging patient survival. Hypothesis: We focus on a novel DDX55, a member of the DEAD-box family of RNA helicases. We hypothesize that DDX55 regulates the activity AR to promote cancer progression in aggressive PCa. Aim: We aim to validate the expression of DDX55 in aggressive PCa and study the DDX55-regulated molecular mechanisms of AR activation. Results: We found that DDX55 expression is enhanced in aggressive CRPC, where it regulates AR activation. Mechanistically, AR interacts with CBP to enhance the H3K27ac in the promoter regions of AR-regulated genes. The H3K27ac led to increased chromatin accessibility and the recruitment of AR co-regulators and RNA polymerase II (RNAPII) to amplify AR-mediated transcription. In parallel, DDX55 uses its helicase activity to resolve RNA/DNA R-loops that accumulate near paused RNAPII, thereby preventing transcriptional stalling and ensuring the sustained elongation of AR target transcripts. Loss of DDX55 in PCa cell lines leads to reduced AR activity, global chromatin condensation, increased ssDNA breaks, and impaired cell proliferation, migration, and invasion. Impact: DDX55 represents a promising therapeutic target for CRPC where AR signalling persists despite androgen deprivation therapy.

LAY SUMMARY

DDX55 expression is associated with aggressive prostate cancer and poor patient survival. DDX55 promotes the proliferation and survival of PCA cells, potentially contributing to cancer progression and metastasis. DDX55 can interact with AR and enhance AR activity. Silencing DDX55 significantly decreases the expression of AR-target genes and binding of AR co-regulators. DDX55 promotes prostate cancer progression by enhancing chromatin accessibility at AR-regulated loci. DDX55 represents a promising therapeutic target for CRPC where AR signalling persists despite androgen deprivation therapy. Our project will characterize DDX55 as an aggressiveness marker of PCA, investigate the molecular mechanism through which DDX55 regulates AR activation, with the goal of identifying new therapeutic targets for prostate cancer, and develop an animal model to study the role of DDX55 in cancer progression. This research is expected to advance our understanding of the mechanisms of prostate cancer progression and help identify ways to target it for improved management of the disease.

Next-Generation Strategy for Cervical Cancer through Dual Inhibition of Wee1 and Myt1 Kinases - Poster #539

Presenter: Sharon Wu, University of Alberta

ABSTRACT_ID#2855

ABSTRACT

Cervical cancer is a major global health burden for women, with few therapeutic advancements since cisplatin-based chemoradiation became standard. Owing to HPV-driven inactivation of tumor suppressors like p53 and Rb, cervical cancer cells lose G1 checkpoint control and rely heavily on the G2/M checkpoint for survival. Wee1 and Myt1 kinases are key regulators of this checkpoint, inhibiting CDK1 to delay mitosis and allow DNA repair. Analyses have shown that both kinases are overexpressed and essential in cervical cancer cells, highlighting them as promising targets. Inhibiting Wee1 and Myt1 disrupts G2/M control and triggers mitotic catastrophe, a promising route to cancer cell death, though its regulation and escape mechanisms remain unclear. We demonstrate that second-generation inhibitors, Debio-0123 (Wee1 inhibitor) and RP-6306 (Myt1 inhibitor), synergize to induce mitotic catastrophe and reduce cell viability in cervical cancer cells. While RP-6306 shows strong preclinical activity, resistance can still develop through compensatory survival mechanisms. Although biomarkers for RP-6306 sensitivity have been explored, its resistance pathways remain poorly defined. To address this, we employed transcriptomic and proteomic profiling of RP-resistant cervical cancer models and identified upregulation of ERK1/2 and senescence-associated signaling. Functional validation studies and mechanistic analysis of mitotic catastrophe is currently underway. Conclusion: Dual Wee1/Myt1 inhibition is a promising strategy to induce mitotic cell death in cervical cancer. Resistance via ERK1/2 kinase and senescence pathways may be overcome with combinatorial approaches. Significance: Targeting the Wee1/Myt1 axis may overcome treatment resistance and improve outcomes in cervical cancer.

LAY SUMMARY

Most cervical cancers rely on backup "emergency brakes" in the cell cycle—targeting those brakes (Wee1 and Myt1) can force cancer cells into cell death. New targeted therapies are more effective than traditional treatments but can still encounter resistance. We are uncovering how cancer cells escape cell death and working on new strategies to block their survival tricks.

Patient Experience and Advocacy

Monday, November 3, 2025

Exploring the Impact of Socioeconomic and Geographic Factors on Immunotherapy Discontinuation and Success in Northern Ontario: A Retrospective, Single-Center Cohort Study - Poster #540

Presenter: Alesha Bishop, NOSM University

Other Authors: Olexiy Aseyev

ABSTRACT_ID#2308

ABSTRACT

Immunotherapy has effectively treated various cancers, enhancing outcomes and extending survival rates. Despite its benefits, the expansive geography of Northern Ontario, presents substantial challenges to immunotherapy efficacy, including insufficient healthcare infrastructure, professional shortages, and economic constraints, resulting in elevated discontinuation rates. This retrospective, single-center cohort study sought to investigate the socioeconomic and geographic factors contributing to immunotherapy discontinuation among patients from Northern Ontario with advanced, inoperable cancers. Data were extracted from the electronic medical records of 450 patients diagnosed with cancer between 2018 and 2022. Among these, 96 cases had complete data on a known outcome (death), providing a robust basis for analysis. The analysis encompassed a comprehensive range of factors: disease-related aspects including cancer type, stage, line of therapy, and the number and location of metastases; social factors like smoking, alcohol, and the distance from the patient's home to the hospital or chemotherapy unit. In this cohort of 96 cases, 53 (55.2%) received Pembrolizumab and 43 (44.7%) were treated with Nivolumab. Among these patients, the discontinuation rate for reasons other than disease progression was 25.6%. Notably, prevalent adverse events such as pneumonitis and thyroid dysfunction contributed significantly to treatment discontinuation. This study underscores the complex interaction between socioeconomic, geographic, and lifestyle factors in Northern Ontario and their impact on immunotherapy efficacy related to access of health care resources. To optimize immunotherapy outcomes and ensure equitable cancer care, it is crucial to address these multifaceted influences through comprehensive healthcare policies and infrastructure enhancements tailored to diverse populations.

LAY SUMMARY

- This study analyzed 96 patients that were treated at the Thunder Bay Regional Cancer Centre. * The Cancer centre, located within the regional hospital services Thunder bay, accounts for 55% of the population serviced within Thunder Bay, as well as the surrounding area which accounts for 43% of the sub-region.
- The regional hospital only receives funding based on the needs of Thunder Bay, not taking into account surrounding cities. * In a cohort of 96 cases, 73% were from urban and 26% of patients were within a rural community.
- 74% patients has metastatic disease with progression and 62% of patients had progression.
- One of the most notable findings was that 80% of rural patients did not tolerate Immunotherapy treatment compared to 60% of urban.
- These findings shed light on the limitations of rural healthcare, with contributions from scarce access to emergency departments, patient health literacy, professional shortages, delayed diagnosis, and multi-morbid factors.
- Next Step: Physician recruitment, nursing recruitment, improvements in ER access, Patient education on risk factors and signs to watch for.

Patient Experience and Advocacy

Sunday, November 2, 2025

Developing a Patient and Caregiver Engagement Plan to Amplify Diverse Voices in Canadian Cancer Society Policy Positions - Poster #541

Presenter: Apiramy Jeyapalan, Canadian Cancer Society

Other Authors: Olivia Kulbak, Elizabeth Holmes, Tracy Torchetti

ABSTRACT_ID#2574

ABSTRACT

People with lived experience of cancer, including patients and caregivers, provide valuable insights into cancer control issues. By incorporating patient and caregiver perspectives, we can align policy positions with the real needs and challenges faced by those directly affected by cancer. Developing a structured engagement plan ensures that a diverse range of voices are heard, leading to more comprehensive, equitable and impactful policy positions that truly reflect the needs of people with cancer and their caregivers. To develop the patient and caregiver engagement plan, we first identified two main goals: to either gather input from patients and caregivers to understand their experiences before developing a policy position or to get feedback on a draft policy position. We outlined key considerations like defining the scope of engagement, timelines, selecting target demographics to engage and establishing strategies to reach a diverse group of patients and caregivers. Lastly, we collaborated with internal stakeholders to refine the plan, creating a clear, adaptable and repeatable process for engagement. Standardizing patient and caregiver engagement ensures a consistent approach to including voices impacted by cancer and understanding their experiences. This approach provides representation across Canada, including people from rural and urban areas, various racial and ethnic backgrounds, gender identities, income levels, and residency statuses. Lessons learned highlight the need for clear, accessible language and flexibility throughout the process. By defining engagement purpose and standardizing steps, this plan offers a repeatable, adaptable framework for effective patient and caregiver engagement.

LAY SUMMARY

People with lived experience of cancer can provide valuable insights into cancer control issues. Standardizing patient and caregiver engagement ensures a consistent approach to including voices impacted by cancer and understanding their experiences.

Patient Experience and Advocacy

Monday, November 3, 2025

Your Voice Matters: Using Patient-Reported Experience Measures (PREMs) to Elevate the Cancer Care Experience - Poster #542

Presenter: Carrie O'Conaill, CancerCare Manitoba

Other Authors: Carrie O'Conaill, Munayie Wakie, Joyce Wei, David Dawe, Donna Turner

ABSTRACT_ID#2696

ABSTRACT

Background and Objectives: Patient-Reported Experience Measures (PREMs) are critical for shaping patient-centered cancer care, providing direct insight into satisfaction, unmet needs, and areas for improvement. The Ambulatory Oncology Patient Satisfaction Survey (AOPSS) is a standardized, validated PREM used by CancerCare Manitoba (CCMB) to ensure oncology services align with patient priorities. **Approach:** AOPSS captures experiences from Manitobans who received ambulatory cancer treatment in the previous three months. The System Performance team oversees survey distribution, collection, and analysis, compiling results from paper and online questionnaires twice a year. The survey measures overall satisfaction and key dimensions of patient-centered care. **Results:** Approximately 3,000 survey invitations are mailed each cycle, with response rates ranging 44-55% since 2004. In 2024, 1,455 patients completed the survey (49.0% response rate). Response rates were highest among patients 65 or older (56.9%) and lowest among 18–34-year-olds (21.1%). Overall satisfaction reached 96.9% with scores for dimensions of person-centred care ranging from 51.6% for Emotional Support to 80.3% for Respect for Patient Preference. Results varied across sex, health region, cancer type, and time since diagnosis. **Conclusion:** AOPSS is more than a survey—it's a strategic tool that ensures patient voices drive cancer care improvements. Supported by the Canadian Partnership Against Cancer, AOPSS is available to cancer programs nationwide, providing jurisdictions with a proven framework to measure and enhance oncology services. We invite stakeholders to join our cross-provincial working group to strengthen and expand the use of AOPSS, helping to build a national standard for patient-centered cancer care.

LAY SUMMARY

Listening to Patients: The AOPSS survey helps CancerCare Manitoba hear directly from patients about their experiences with cancer care, making sure their voices shape improvements. **Who Participates?** People who had cancer treatment in the last three months are invited to take the survey. Older patients tend to respond more than younger ones, and response rates vary across different regions in Manitoba. **Making a Difference:** The survey results help health experts plan better cancer care. Manitoba, Saskatchewan, and Alberta are working together to improve how this survey is used across Canada, ensuring patient feedback leads to real change.

Patient Experience and Advocacy

Sunday, November 2, 2025

Layers of Experience: A Multimodal Analysis of Digital Stories from Pre-Menopausal Women with Breast Cancer - Poster #543

Presenter: Amber Young, University of Calgary

Other Authors: Janet de Groot, Kathleen Sitter, Tiffany Yu

ABSTRACT_ID#3275

ABSTRACT

Pre-menopausal women with breast cancer experience unique challenges as they navigate their journey with the illness. However, their experiences and needs may often be overlooked in breast cancer psychosocial interventions which typically target younger and older women. Despite rising incidence rates and at more advanced stages in this demographic (Nichols et al., 2017), pre-menopausal women report challenges accessing well matched psychosocial services. The need to understand the experiences of this population emerges as both timely and necessary. Digital stories, a multimedia method that combines photographs, music, and first-person narratives to create a personal story in a three-to-five-minute video (Sitter et al., 2020), offer an accessible format to share the lived experiences of premenopausal women with breast cancer. By centring diverse narratives conveying experiential knowledge digital stories can deepen understandings of how women in this demographic navigate and make sense of their journey. As such, this modality has the potential to inform healthcare delivery and responses in meaningful ways that honour the complexities of patient realities. This presentation will share preliminary findings from the multimodal analysis of six digital stories created by pre-menopausal women (18-50 years of age) with breast cancer in Alberta, Canada who participated in a digital storytelling group intervention. The multimodal analysis assessed each digital story's dominant imagery, narrative, tone, content, and overall framing. Reflections from the research team's process of conducting this analysis will be offered, inviting audience engagement with the textured layers of the participant experiences.

LAY SUMMARY

The unique needs of pre-menopausal women with breast cancer may often be overlooked in existing psychosocial support, which tends to focus on either younger or older age women. With rising incidence rates in this demographic, there is a timely and necessary need to understand their experiences to shape and inform meaningful care responses and support from health professionals. In research, digital storytelling offers a powerful and accessible method for participants to share the richness and complexity of their lived experiences. These short, first-person narratives combine images, music, and voice to help individuals make sense of their journey and produce a valuable record of experiential knowledge that can be shared with multiple audiences, including health professionals. This presentation will share preliminary findings from a study involving six pre-menopausal women with breast cancer who participated in a digital storytelling group intervention. Using a multimodal analysis, the research team examined each digital story's dominant imagery, narrative, tone, content, and overall framing. Reflections on this analytical process will be shared with the goal of contributing to care responses that reflect the complex and layered experiences of the people we work alongside.

Patient Experience and Advocacy

Monday, November 3, 2025

Survivor Advocacy: Using social media to break stigma, raise awareness, and empower change in cancer care - Poster #544

Presenter: Harjeet Kaur, Co- Founder of Chai and Hope : South Asian Cancer Community

ABSTRACT_ID#2766

ABSTRACT

As a Stage 4 rare blood cancer survivor, my diagnosis was delayed by three months due to limited awareness and systemic gaps—compounded by my recent immigration to Canada. Diagnosed with Subcutaneous Panniculitis-like T-cell Lymphoma (SPTCL) and Hemophagocytic Lymphohistiocytosis (HLH), I navigated treatment in isolation during the COVID-19 pandemic, enduring immense physical and emotional tolls. I turned to Instagram (@hk_thrivers) as a digital diary, which evolved into a public platform for cancer education and advocacy aimed at breaking cultural stigma. Sharing my lived experience as a South Asian immigrant woman helped others feel seen and supported—especially those navigating language barriers, taboos, and systemic mistrust. In 2023, I co-founded Chai and Hope, a support initiative for South Asian cancer patients and caregivers. Our goal is to create safe spaces for open conversations around cancer, fertility loss, and mental health—topics often silenced by cultural beliefs related to karma or shame. Today, my platform blends advocacy, education, and policy engagement. I have collaborated with organizations such as the Canadian Cancer Society, Leukaemia Lymphoma Society of Canada and Wellspring Alberta, and have represented patient voices at Parliament Hill to advocate for equitable care and financial support. My experience highlights how survivor-led digital advocacy can influence healthcare systems and foster more inclusive, culturally responsive cancer care.

LAY SUMMARY

Social media is a powerful tool for advocacy. It helped me, as a Stage 4 cancer survivor, turn my personal story into a way to educate others, break cultural stigma, and support people facing similar challenges—especially in underrepresented communities. Many immigrants and South Asians dealing with cancer face silence, shame, and barriers in the healthcare system. By sharing my journey publicly and co-founding Chai and Hope, I created safe spaces for open conversations around cancer, mental health, and fertility loss. Survivor-led advocacy can lead to real change. Through collaborations with cancer organizations in Canada, the U.S., and India, and by speaking at Parliament Hill, I've shown how lived experiences can shape better, more inclusive cancer care.

Patient Experience and Advocacy

Sunday, November 2, 2025

A Roadmap for Participatory Cancer Research: Co-Creation of a Patient Navigator Program for Black-Identifying Patients with Breast Cancer - Poster #545

Presenter: Ashanté Lakey, Princess Margaret Cancer Centre

Other Authors: Jennifer Ryan, Michelle Audoin, Emily Peterson, Aisha Lofters, Clare Cruickshank, June Buckle, Lisa Kamran, Patricia Russell, Leila Nicholls-Springer, Danielle VandeZande, Jennifer Jones, Melanie Powis

ABSTRACT_ID#3041

ABSTRACT

There is growing interest in using Community-Based Participatory Research (CBPR) approaches to engage underserved communities in co-creating interventions to address health disparities. CBPR counters histories of exploitation in health research by centring the lived expertise and priorities of marginalized communities to inform more acceptable, effective, and sustainable strategies to advance equitable health outcomes. However, practical guidance on how to successfully undertake CBPR remains limited, particularly in clinical settings such as cancer care. Our team partnered with community organizations in the Greater Toronto Area to co-design a patient navigation program for Black-identifying patients with breast cancer. Patient navigators play a vital role in addressing persistent disparities across the cancer continuum by providing individualized support and advocacy. The program aims to improve access to culturally safe, high-quality breast cancer care through treatment and survivorship. We began with a six-month engagement process to build trust, recruit diverse key informants, and co-create expectations for involvement and knowledge governance. This led to the formation of a knowledge exchange advisory committee to guide the co-development and contextualization of the navigation program. The committee meets monthly and is co-facilitated by a patient partner and a social justice lead. Based on our initial needs assessment, meetings are structured to include shared learning, discussion, and time to spotlight upcoming community initiatives. Evaluation of the co-creation process will involve feedback via email and at pre-scheduled check-ins, surveys, an activity-based focus group, and a closing meeting to collectively reflect on the process and celebrate the collaboration.

LAY SUMMARY

Working closely with communities leads to the development of health programs that are more relevant and impactful. Community-Based Participatory Research (CBPR) supports the development of culturally safe, equity-focused cancer care by following community members' lead in shaping solutions that reflect their lived experiences and priorities at every stage of the research process. Developing meaningful collaboration requires researchers to create a safe space for trust-building, setting shared expectations, and collective decision-making. This kind of work is a long-term investment in community relationships that often extends beyond traditional, output-driven research timelines, but is essential to doing the work well. Ongoing feedback helps to guide and strengthen the co-creation process. Creating regular opportunities for participants to share input—through check-ins, surveys, and focus groups—enables course correction, keeps the work aligned with community needs and goals, and disrupts traditional research hierarchies by affirming community expertise and supporting equitable power-sharing. These practices also encourage researcher reflexivity, promoting cultural humility and culturally safe practice.

Patient Experience and Advocacy

Monday, November 3, 2025

Enhancing Pediatric Radiation Therapy Through Video-Based Distraction and Empowerment - Poster #546

Presenter: Heather Roy, RadFlix

Other Authors: Natalie Logie, Tatiana Ritchie

ABSTRACT_ID#3086

ABSTRACT

Radiation therapy can be a distressing experience for pediatric patients, often leading to high levels of anxiety and, in many cases, the need for sedation or general anesthesia to complete treatment. Video distraction therapy is an innovative, patient-centered tool that can be used to transform the radiation experience by allowing children to watch their favorite movies during treatment. Video-based distraction therapy addresses the psychosocial needs of pediatric patients by reducing fear, anxiety, and the emotional burden associated with cancer care. Preliminary implementation of RadFlix as a video distraction tool has demonstrated multiple psychosocial benefits. Children report feeling more in control during treatment sessions, with many stating that time passes more quickly and the overall experience is less frightening. Importantly, some younger patients who previously required anesthesia are now able to tolerate therapy while awake, reducing both clinical risks and healthcare costs. By incorporating a familiar and enjoyable activity into the clinical environment, RadFlix supports emotional well-being and helps restore a sense of normalcy and joy during a challenging time. This approach underscores the critical role of psychosocial care in pediatric oncology, where emotional support and engagement are essential components of effective treatment. RadFlix represents a scalable, non-invasive solution that aligns with holistic care models, emphasizing the importance of treating not just the disease, but the whole child.

LAY SUMMARY

Psychosocial interventions can reduce the need for sedation: Integrating positive distraction techniques into radiation treatment can enable younger patients to complete therapy awake, decreasing reliance on anesthesia and its associated risks and costs. Emotional support improves treatment adherence and experience: By addressing fear and anxiety through engaging, child-friendly interventions, patients are more likely to tolerate and complete treatment, enhancing both clinical outcomes and emotional well-being. Holistic care enhances quality of life during cancer treatment: Research in this area highlights the importance of treating the psychological and emotional aspects of illness not just the physical, especially in vulnerable populations like children, who benefit greatly from normalization and a sense of control.

Patient Experience and Advocacy

Sunday, November 2, 2025

Exploring Transitions in Care Among Individuals Living with and Beyond Cancer: A Qualitative Study - Poster #547

Presenter: Benny Rana, Cumming School of Medicine, University of Calgary

Other Authors: Jaling Kersen, Christine Smith, Areej Hezam, Khara Sauro

ABSTRACT_ID#2622

ABSTRACT

Background: Cancer care is multifaceted and involves multiple healthcare providers and settings, from diagnosis through to treatment and beyond. Each time the responsibility of care is transferred from one healthcare provider or setting to another (a transition in care or TiC), there is an increased risk of medical errors, patient dissatisfaction with care and poor outcomes.

Objective/Purpose: To explore patient perspectives and experiences with TiC during cancer care, and to identify perceived gaps in care during TiC among individuals living with and beyond cancer. **Methods:** An Interpretive Descriptive approach was used to generate clinically relevant insights while maintaining methodological rigour for this qualitative study. Participants were recruited between September 1, 2022, to September 9, 2023. Purposive sampling was used to obtain a diverse sample based on age, sex, type of cancer, and geographical location. Semi-structured interviews were conducted and analyzed with thematic analysis. **Results:** Thirty-one interviews were conducted; the average age of the participants was 62.5 years, 74.2% identified as women, and the majority resided in an urban city center in Alberta. Four themes, which included subthemes, were identified: 1) navigating personal struggles and growth during TiC, 2) system strengths and failures across TiC, 3) strategies for improving TiC, and 4) overall experiences of the TiC. **Conclusion:** The study highlights the complex nature of TiC among individuals living with and beyond cancer. The findings emphasize the need for patient-centred, coordinated approaches to improve the quality of care for individuals living with and beyond cancer.

LAY SUMMARY

Key Messages: Transitions in care for individuals living with and beyond cancer are vulnerable periods that often lead to anxiety and uncertainty, gaps in communication, and unmet needs for patients navigating between healthcare providers and settings. Individuals living with and beyond cancer value continuity, clarity, and compassionate communication during transitions in care, wanting to be informed and actively involved in their care. Understanding patient experiences during transitions is essential for designing more equitable, coordinated, and person-centred cancer care systems that support individuals across the full cancer journey.

Patient Experience and Advocacy

Monday, November 3, 2025

Reading the room: A joint perspective from a scientist- and patient-partnered working group on the importance of patient-centred language in the dissemination of cancer research - Poster #548

Presenter: Cara MacInnis, Research Institute of the McGill University Health Centre

Other Authors: Kyle Dickinson, Kirsten Efremov, Monyse De Nobrega, Jennifer Auchinleck, Louise Bird, Jennifer Coish, Isabelle Allain-Labelle, Antonia Palmer, Shuk On Annie Leung, Julia Burnier

ABSTRACT_ID#3142

ABSTRACT

Importance: Scientific communication in cancer research has historically focused on dissemination within professional circles, using technical language inaccessible to the broader public. However, growing emphasis on equity, diversity, inclusion, and accessibility has highlighted the need to engage PWLLEs in scientific discourse. Despite positive shifts, language in scientific communication often remains exclusionary, stigmatizing, and dehumanizing, impeding meaningful PWLLE engagement. **Observations:** Co-created by a diverse team of PWLLEs, patient advocates, and scientists, we identify key categories of exclusionary language in cancer science: (1) language that places blame on the patient, such as phrases implying fault for treatment failure or outcomes; (2) dehumanizing terminology that reduces patients to conditions or portrays them as objects of study; and (3) language that defines individuals by their diagnosis, disease characteristics or lifestyle factors, often neglecting socioeconomic and systemic factors. We propose alternative phrasing and a decision-making framework to guide more inclusive and compassionate scientific communication that can foster greater PWLLE engagement and empowerment. **Conclusions and Relevance:** Inclusive and respectful language in scientific communication is crucial to creating a welcoming environment for PWLLEs and ensuring that research priorities align with patient needs. This unique, PWLLE-led perspective exemplifies the value of patient-scientist partnerships in developing actionable guidance, offering a reflexive framework for improving communication. By adopting these recommendations, researchers can advance health equity, improve PWLLE engagement, and enhance the overall impact of cancer research. This collaborative approach strengthens the bridge between science and lived experience, ultimately driving research that is more relevant, compassionate, and impactful for everyone.

LAY SUMMARY

Words matter. Scientific language can unintentionally blame or dehumanize people living with and affected by cancer (PWLLEs). We co-developed better ways to communicate. Together with PWLLEs, patient advocates, and researchers, we identified harmful language patterns in cancer research and proposed more inclusive and respectful alternatives. Inclusive language builds stronger partnerships. When researchers use compassionate, accurate, and inclusive language, it helps PWLLEs feel more welcome and valued. This leads to more meaningful engagement and research that better reflects real-world needs and experiences, benefiting cancer research.

Patient Experience and Advocacy

Sunday, November 2, 2025

The Hidden Burden: Financial, Physical, and Psychosocial Impacts of Travel to Treatment for Canadian Blood Cancer Patients - Poster #549

Presenter: Christina Sit, Leukemia & Lymphoma Society of Canada

Other Authors: Colleen McMillan, Nadine Frisk, Deidra Brown-John, Kristin Dyck, Branon Peel, Paul O'Connell

ABSTRACT_ID#3217

ABSTRACT

Accessing care often requires patients with blood cancers in Canada to travel long distances, posing additional challenges beyond the disease itself. This study aimed to examine the financial, physical, and psychosocial impact of travel to treatment among Canadian blood cancer patients and their caregivers. A steering committee consisting of people with lived experience was convened to design and develop the survey. After community validation, data was collected from 487 respondents between November 2024 and January 2025. Results revealed that 64% of participants traveled over 100 km and 42.3% spent three or more hours traveling to receive care, with 66.6% having to do so for more than a year. The travel demands were associated with substantial financial strain—47.6% of respondents reported a $\geq 10\%$ reduction in household income, and those requiring a secondary residence near treatment centers incurred costs averaging over \$14,000. The physical toll was also significant, with 65% reporting worsened fatigue due to travel. Psychosocial impacts included extended separation from family (over 50% lived away for more than three months) and emotional distress, as 47% expressed feelings of guilt about being a burden. These findings underscore the need for targeted policy and support interventions to mitigate the adverse effects of travel for cancer care in Canada.

LAY SUMMARY

Many blood cancer patients in Canada must travel far for treatment, with most going over 100 km and some spending more than three hours to get there, often for over a year. This travel causes serious financial stress, including income loss and high costs for things like temporary housing near treatment centers. Travel also takes a toll on physical and emotional health, worsening fatigue and causing feelings of guilt and family separation for many patients and caregivers.

Patient Experience and Advocacy

Monday, November 3, 2025

Living with a life-limiting Cancer Diagnosis: A Longitudinal Qualitative Study - Poster #550

Presenter: Colleen Doylend, UBCO

Other Authors: Colleen Foran

ABSTRACT_ID#2495

ABSTRACT

This research aimed to understand the experiences of living with metastatic cancer with a particular focus on understanding the strategies persons used to continue to live and grow. This was a yearlong, qualitative study with 10 participants. They reflected on ongoing life experiences, relationships, and activities in the face of changing treatments and prognoses. Preliminary findings indicated participants strove to maintain a normal life within the bounds dictated by their illness and complex treatment plans. Analysis revealed the following codes: diagnosis; living in liminality; living with an uncertain future; relationships with family; relationships with friends; impact of being in the health care system; and personal strategies. They reported a sense of needing others to understand their new reality and their sense of being in liminality, which was a deeply personal, often indescribable, new sense of being, a new identity that pervaded all aspects of their lives. They faced intense and all-consuming interactions with health care, family, and friends while dealing with an uncertain future, a deep sense of loss, and hopes and fears in coming to terms with their ever-evolving prognoses. All participants spoke of an emergence of hope and a heightened desire to live after their diagnoses. Participants joined the study for various reasons including preserving their experiences in words as a legacy for family and allowing those in health care an opportunity to hear the patient experience. Participants acknowledged that having the opportunity to speak, put into words, and reflect clarified their experiences, thoughts, and feelings.

LAY SUMMARY

Life after a diagnosis of metastatic cancer was irrevocably changed, life dominated by doing what one had to do to stay alive rather than what they wanted to do. Participants spoke of feeling vulnerable in a new system that was indecipherable with its terminologies, pathways and hierarchies. They struggled with not understanding what a symptom of the cancer or treatment was, as opposed to other community/environmental exposures. They also experienced frustration in finding and accessing resources. Participants spoke of a perception of duality; being surrounded by loved ones yet feeling completely alone, feeling the beauty of a moment and sorrow of losing such moments, or looking and even feeling healthy while knowing they had a terminal illness. Participants expressed needing others to acknowledge their diagnosis and not deny or minimize their new reality. Most recognized that others didn't know what to say to them and did not mean to be unkind, yet they left participants feeling alone, misunderstood and even rejected.

Patient Experience and Advocacy

Sunday, November 2, 2025

Tailoring Patient-Reported Experience Measures: How Custom Questions Support Quality Improvement in Cancer Care Alberta - Poster #551

Presenter: Linda Watson, Alberta Health Services

Other Authors: Claire Link

ABSTRACT_ID#2363

ABSTRACT

Background: Patient-Reported Experience Measures (PREMs) help ensure that patient and family perspectives are routinely heard in Cancer Care Alberta (CCA). The Ambulatory Oncology Patient Satisfaction Survey (AOPSS), which includes validated questions grouped to represent six dimensions of person-centred care, is the primary PREM utilized. Adding custom questions relevant to Alberta's context and priorities has helped drive quality improvement (QI) within CCA. Methods: Custom questions include, "How involved has your primary care provider been while you have been receiving cancer treatments?", and "While waiting for your first appointment with the cancer program, which supports did you need and could not access?" Results: Patients with a "Very Involved" primary care provider reported significantly higher satisfaction across all six dimensions of care, compared to patients whose provider was "Not Very/Not At All Involved". "Emotional Support" showed the largest difference. Regarding pre-consult supports, 25% of respondents indicated needing at least one support and being unable to access it. Treatment information and access to patient navigation services were most commonly needed. Discussion: These findings have helped support QI within CCA. Recognizing that patients are more satisfied when primary care is involved provides evidence for embracing "shared care" models and encourages communication between cancer care and primary care. Additionally, an initiative is underway to implement standard questions to identify needs and connect patients with relevant supports shortly after diagnosis. Conclusion: Customizing PREMs to inform local priorities ensures the findings can help drive QI. AOPSS is used by multiple provinces, with tailored questions for each provincial context.

LAY SUMMARY

Patient-reported experience measures (PREMs) are a valuable way to routinely hear patient and family perspectives. Customizing questions to fit provincial priorities and contexts helps ensure the results are actionable and can be used to drive quality improvement initiatives at the local level. Cancer Care Alberta has been utilizing the Ambulatory Oncology Patient Satisfaction Survey (AOPSS) for many years. This consistency in collection has enabled comparisons over time, to understand how different aspects of patient experience change over time. Customizing questions each survey cycle ensures the results are meaningful and relevant to the current context in Alberta. Other provinces may want to consider utilizing the AOPSS to inform their own quality improvement work, and Alberta's custom questions may be relevant to these other provincial contexts. Multiple provinces have been collaborating since 2022 to share questions and approaches to further improve the utility of AOPSS.

Patient Experience and Advocacy

Monday, November 3, 2025

New Horizons for Peer Support in Cellular Therapies: Co-Designing Innovative Support Pathways - Poster #552

Presenter: Karine Bilodeau, University of Montreal

Other Authors: Ludovic Tamaro, Sandie Oberoi, Deborah Pascale, Kelley Kilpatrick, David Ogez, Marie-Pascale Pomey, Catherine Paquette-Gascon, Israel Fortin, Isabelle Fleury, Imran Ahmad

ABSTRACT_ID#2896

ABSTRACT

Cellular therapies, including allogeneic hematopoietic cell transplantation (HCT) and CAR-T cell therapy, offer promising clinical outcomes but are frequently accompanied by intense physical symptoms and profound emotional and social isolation. While professional healthcare is essential, it may not fully address the evolving psychosocial needs of these patients. Peer support, rooted in shared lived experience, presents a valuable yet underutilized resource in oncology care. This project aimed to co-develop structured peer support pathways tailored to the needs of individuals preparing for HCT and CAR-T therapy. The presentation will highlight participants' perspectives on the role and value of accompanying patients (peer supporters), as well as key challenges and strategies for sustainable implementation. Using a participatory research approach, two working groups (N=14), including patients, healthcare professionals, and managers, participated in three sessions to co-design the support pathway and discuss barriers and facilitators to implementation. All sessions were recorded, transcribed, and analyzed using content analysis. Participants recognized peer support as a meaningful complement to professional care, addressing emotional, psychological, social, informational, and practical needs. It was seen as enhancing continuity of care and potentially alleviating clinical workloads. Key considerations for successful implementation included clear role definitions, preparing accompanying patients to manage emotional strain and compassion fatigue, and integrating them effectively within care teams. Peer support can play a critical role in meeting the complex needs of patients undergoing cellular therapies. Ensuring clarity of roles and providing adequate training and support for accompanying patients are essential for creating effective, sustainable pathways within oncology care.

LAY SUMMARY

Co-designed peer support pathways were developed through participatory research involving patients, healthcare professionals, and managers. Peer support was found to complement professional care, addressing emotional, practical, and informational needs while easing clinical workloads. Sustainable implementation requires clear role definitions, preparation for emotional challenges, and integration into the care team.

Patient Experience and Advocacy

Sunday, November 2, 2025

Development of a Patient Partner Learning Hub: Building skillsets and trial knowledge for meaningful patient engagement across a pan-Canadian Network - Poster #553

Presenter: Diana Kato, Canadian Cancer Clinical Trials Network

Other Authors: Don Wood, Raisa Chowdhury, Stephen Sundquist, Judy Needham, Gretta Hutton, Katherine Zeman, Janet Dancey

ABSTRACT_ID#3051

ABSTRACT

Introduction. A goal of the Canadian Cancer Clinical Trials Network (3CTN) and its 51-member cancer centres is to improve recruitment, performance and access to academic cancer clinical trials and to ensure meaningful patient engagement throughout their conduct. As centres developed engagement strategies, an identified need emerged for connecting 3CTN's growing community of patient partners to suitable education resources providing knowledge and skills beneficial for fully contributing to cancer research activities. **Methods.** A project team was formed consisting of patient partners and a member cancer centre representative involved in patient engagement. An initial environmental scan of available resources and training programs was conducted and a framework for indexing subject matter according to defined engagement roles and knowledge areas. Each were linked within the framework to relevant resources, training modules, education materials and mentorship programs. **Results.** The Patient Partner Learning Hub (PPLH) was developed and includes a total of four knowledge areas and six patient engagement areas of involvement, along with glossaries and supporting materials. The environmental scan identified 110+ existing resources, training programs and education materials. The PPLH was created using Microsoft Power BI and is accessible on www.3CTN.ca. **Conclusion.** The PPLH addresses patient partners' need for a comprehensive, user-driven resource that provides an accessible, intuitive approach to sourcing learning materials relevant to their clinical research and skills-development needs. Knowledge gained across the patient partner community will aid in achieving greater capacity and potential for meaningful engagement.

LAY SUMMARY

Helping Patient Partners Get Involved in Cancer Research: The Canadian Cancer Clinical Trials Network (3CTN) provided patient partners with tools and knowledge to contribute meaningfully to cancer research. **Creating a Learning Hub for Patients:** A team of patient partners and research professionals worked together to find and organize over 100 helpful resources. They built an online tool called the Patient Partner Learning Hub (PPLH) to make it easy for patients to find training and educational materials. **Making Learning Easy and Accessible:** The PPLH is designed to be user-friendly and helps patients build the skills they need to be active partners in research. This shared learning helps improve the quality and impact of patient partner involvement in cancer trials.

Patient Experience and Advocacy

Monday, November 3, 2025

Learning Together: The Riddell Centre for Cancer Immunotherapy Co-Mentorship Model for Patient Engagement in Biomedical Research - Poster #554

Presenter: Laura Mah, University of Calgary/ Riddell Centre for Cancer Immunotherapy

Other Authors: Shannon Snelling, Sadia Ahmed, Don Wood, Nubia Zepeda, Judy Wang, David Spence, Harjeet Kaur, Hobin Seo, Jessica López Espinosa, John Corriveau, Kurt Hildebrand, Leanne Palichuk, Lucille Dupuis, Megan Kinzel, Taye Louie, Paul Fairie

ABSTRACT_ID#2913

ABSTRACT

Patient-mentor engagement has emerged as a critical requirement for successful grant funding and ethical research practices. Researchers, particularly graduate student trainees, often lack access to patient partners with lived cancer experience and professional life skills. Studies have shown that involving patients enhances research relevance, communication strategies, and opportunities for bi-directional learning. To address this gap, the Riddell Centre launched an 8-month pilot program involving five patient partners and seven trainees. Patient partners selected one to two trainees for co-mentorship. Participants completed pre- and post-program surveys and attended three workshops led by Alberta Strategy for Patient-Oriented Research SUPPORT Unit (AbSPORU), focusing on the role of patients in fundamental research. Over four months, each team met once or twice monthly to co-develop a science communication project and foster reciprocal knowledge exchange. This program demonstrated feasibility in integrating patient voices into biomedical research. Patient partners reported increased understanding of cancer research and greater confidence in discussing scientific topics. Trainees gained a deeper appreciation for patient engagement, improved their research and public communication skills, and reported a positive impact on their academic and career trajectories. Additional outcomes included an expanded mentorship network, peer-to-peer trainee support, and increased transparency of our research with the public. Patient engagement ensures research remains aligned with the needs of those it ultimately serves. Despite challenges such as limited funding and awareness, our pilot program offers a scalable model for embedding patient perspectives into early-stage research. We aim to inspire broader adoption of similar initiatives across institutions in Canada.

LAY SUMMARY

Graduate student trainees learned how to incorporate patient perspectives and needs into research Gained awareness of the human impact of their research Increased confidence in research execution and communication Real-world mentorship beyond the laboratory Bridged the gap between patient partners and researchers through reciprocal education of the cancer experience and research efforts Encouraged increased dialogue and understanding between patient partners and researchers Other Canadian university institutions are actively engaged in adopting similar programs due to the success of our Co-Mentorship Pilot Program

Patient Experience and Advocacy

Sunday, November 2, 2025

From patient-scientist interactions to co-created actions: Reflections on the development of a strong patient-scientist partnership in cancer research - Poster #555

Presenter: Kyle Dickinson, RI-MUHC

Other Authors: Cara MacInnis, Kirsten Efremov, Jennifer Coish, Monyse de Nobrega, Jennifer Auchinleck, Isabelle Allain-Labelle, Antonia Palmer, Louise Bird, Shuk On Annie Leung, Julia Burnier

ABSTRACT_ID#3144

ABSTRACT

Background: Meaningful patient engagement is increasingly recognized as essential to impactful research. Yet, true co-creation between patients, advocates, clinicians, and scientists remains rare, with many efforts still rooted in tokenism. At CCRC 2023, informal conversations between people with lived and living experience (PWLLE) and researchers sparked the organic formation of a collaborative engagement group rooted in mutual respect and equal partnership. Objectives: To reflect on how this PWLLE engagement group was created and evolved into a collaborative model where patients are equal partners throughout the research process—from idea generation to dissemination. Methods: Rather than beginning with a formal framework, the group emerged from relationship-building and shared curiosity. Initial interactions built trust and laid the groundwork for deeper collaboration. As the group took shape, members jointly defined goals, values, and communication norms and early sessions focused on establishing shared priorities. The group chose to explore the role of language in research, drawing on PWLLE reflections and lived experiences. Co-creation was embedded through shared decision-making, co-authorship on papers and grants, and conference presentations. Reflective practices were used regularly to strengthen the group dynamic and ensure continuous improvement. Results: The group has contributed to several outputs, including a co-authored commentary on inclusive language, a grant submission, and a conference presentation. PWLLE insights meaningfully shaped how issues like stigma and care access were addressed and communicated. Conclusion: Authentic partnership requires listening, flexibility, and a willingness to challenge traditional hierarchies. This experience offers a model for patient engagement that is inclusive, equitable, and truly collaborative.

LAY SUMMARY

This group came together naturally, starting with honest conversations between people with lived and living experience (PWLLE) and researchers who wanted to learn from one another. Rather than being led solely by researchers, our group used a model where PWLLEs and scientists worked as equal partners. Together, we shaped research questions, co-authored articles, and gave conference presentations—addressing issues like stigma and access to care. True partnership requires listening, flexibility, and a willingness to rethink traditional roles. This experience offers a meaningful model for patient engagement that is inclusive, equitable, and genuinely collaborative.

Patient Experience and Advocacy

Monday, November 3, 2025

Voices of Resilience: Analysing #MetastaticBreastCancer Content on TikTok - Poster #556

Presenter: Ari Meguerditchian, McGill University

Other Authors: Carla Herman, Jillian Schneidman, Eric Belzile, Nina Morena

ABSTRACT_ID#2393

ABSTRACT

Background: Young women with metastatic breast cancer (YWMBC) have unique biopsychosocial challenges, and often turn to social media for support. This study aims to understand YWMBC engagement on TikTok and analyze content about MBC. **Methods:** Videos tagged with #metastaticbreastcancer on TikTok were collected in 07/24. Reviewers gathered video characteristics (username, caption, length, date posted, number of followers, likes, views, shares, comments, and presence of sponsorship) and creator demographics (age, location, engagement approach). Explanations, communication styles and themes were assessed. Spearman's correlation coefficient was calculated between variables. **Results:** 336 TikToks were included. 1% included sponsorship. Average length was 1:46 minutes. 161 used music. Mean follower number was 61,927 (SD 350,900). Mean number of views, likes, and comments was 209,882 (SD 625,961), 15,541 (SD 134,070), and 622 (SD 3899), respectively. The majority were filmed at home (56%). 21% were filmed in a hospital setting. Average age at diagnosis was 31.4 years old (SD 4.3). Topics discussed included: daily life with MBC (70%), diagnosis (42%), treatment options (38% systemic, 26% surgery and 16% radiation). The most relevant correlations observed for viewer engagement (likes, views) included display of surgical scars, explicit expression of emotions, and diagnosis story. Predictors specific to more comments included fear of recurrence, grief and motherhood. Common reasons for making videos about their MBC include wishing to share their experiences, provide hope, and to raise awareness of the Stage IV experience. **Conclusion:** TikToks from YWMBC present personal narratives of the daily experience of MBC and may represent a journaling equivalent.

Patient Experience and Advocacy

Sunday, November 2, 2025

Patient reported experiences of barriers and facilitators to colonoscopy follow-up after an abnormal FIT in Alberta - Poster #557

Presenter: Sharon Mah Mah, Provincial Population Public Health

Other Authors: Jawad Chistie, Kamala Adhikari

ABSTRACT_ID#2752

ABSTRACT

Background Colorectal cancer (CRC) screening reduces CRC incidence, mortality, and treatment costs through early detection and management. For the average risk population, the fecal immunochemical test (FIT) followed by colonoscopy (for FIT +) is an effective method for CRC screening, but it is under-utilized in Alberta. This study aims to understand Alberta patients' barriers and facilitators to colonoscopy completion after FIT+. **Methods** This is a pragmatic descriptive qualitative inquiry. From July 2024 to May 2025, we recruited FIT+ patients aged 50-74, with no family history of CRC from the North Zone and Northeast Calgary, Alberta. Recruitment strategies involved posters emailed to nonprofit community organizations, local newspapers, primary and colonoscopy clinics, patient advisory network, and the Alberta Survey Panel. Interviews were conducted virtually, video and audio-recorded, transcribed, and analyzed using NVivo 15. The Consolidated Framework for Implementation Research (CFIR 2.0) informed the interview guide and analysis. **Results** Nine FIT+ participants from Alberta were interviewed. Patients reported facilitators to colonoscopy completion were patients' capability – understanding the meaning of FIT+, colonoscopy process, bowel prep instructions; patients' family or friends' experiences with CRC or colonoscopy (local attitudes); patients have time (opportunity) enables preparation and colonoscopy procedure. Patient experienced barriers included uncomfortable bowel preparation and painful colonoscopy (need); primary care provider (PCP) did not listen to patients' concerns (patient centeredness), and face-to-face consultation with PCP was time intensive (opportunity). **Conclusion** Patient reported experiences provide insights into areas where the health system needs to target strategies for decreasing barriers to improve colonoscopy completion after FIT+.

LAY SUMMARY

1. FIT followed by colonoscopy is an effective way to screen, reduce, and improve colorectal cancer (CRC) outcomes, but many eligible Albertans are not following this pathway.
2. Albertans reported that their knowledge and understanding of the FIT result, colonoscopy process, and bowel preparation; as well as supportive friends, family, and family doctor facilitated their colonoscopy procedure. The barriers patients encountered was the bowel prep process was unpleasant, and colonoscopy was painful. A few were frustrated by their family doctor, while some had urgent health challenges that were prioritized over colonoscopy.
3. Understanding patients' experiences of barriers and facilitators to colonoscopy after a positive FIT enables us to develop strategies to support patients and providers.

Patient Experience and Advocacy

Monday, November 3, 2025

Development and evaluation of a pilot health communication program for graduate students in oncology - Poster #558

Presenter: Maud Bouffard, Research Center of the CHU de Québec - Université Laval

Other Authors: Antoine Frasier, Maxime Bouchard, France Locas, Denis François Gravel, Maya Obadia, Aline Hajj, Annie LeBlanc, Amélie Fradet-Turcotte, Hermann Nabi, Lynn Rollande Gauthier

ABSTRACT_ID#2640

ABSTRACT

Context: Patient and community engagement is central to CIHR's Institute of Cancer Research's 2024-2029 priorities. Thus, training emerging researchers in effective, audience-tailored communication is essential to advance transdisciplinary, patient-oriented cancer research. This project, co-developed and co-presented with patient-partners, evaluates a pilot health communication program for graduate students, aiming to promote two-way dialogue. Methods: A 25-hour, 8 week program grounded in best practices for clear, accessible and collaborative communication was delivered to seven graduate students in oncology. Using the Kirkpatrick training evaluation model, students completed a satisfaction questionnaire and participated in a focus group assessing their reactions to the program and perspectives on learning outcomes. Data were analyzed using descriptive statistics and thematic analysis. Results: Five students completed the questionnaire and four participated in the focus group. Most were doctoral students (80%), aged 35 ± 4.4 years [28-41]. Overall satisfaction was high (4.4 ± 0.5), especially for workshops on health literacy (4.2 ± 0.8), motivational interviewing (4.5 ± 0.9), the art of presentations (4.6 ± 0.5), and the Café Scientifique public engagement event (4.8 ± 0.4). Thematic analysis revealed that the multi-session format, combining individual and small-group activities, supported communication skill development. Participation in the Café Scientifique was described as challenging but also confidence-building for community-based presentations. Participants suggested more hands-on activities and mentorship from patient partners to enhance the program. Conclusions: This study demonstrated high satisfaction with the training, including opportunities for two-way dialogue. Refinements were identified to enhance the program's ability to catalyze a culture of patient and community-led cancer research. Future research will assess its impact on community engagement.

LAY SUMMARY

Clear, accessible, and collaborative communication is essential to ensure that all research interest-holders can understand and use the information to actively engage in discussions and contribute meaningfully as expert members of cancer research teams. To support the development of communication skills in the next generation of cancer researchers, a pilot health communication program was co-developed with people with lived experience of cancer for graduate students in oncology. Students were highly satisfied with the overall program, workshops on health literacy, motivational interviewing, the art of presentation to craft communications tailored to the audience, and most importantly the Café-scientifique public engagement event held in the community. Analysis of focus group data revealed that a program spread out over time, combining individual and small-group activities, along with the opportunity to prepare and deliver a community-based presentation, supported the development of communication competencies. Suggestions for improvement included incorporating more hands-on activities and pairing students with patient partner mentors to further enhance learning. Future studies will explore the program's impact from the perspectives of patients, caregivers, and members of the research community.

Patient Experience and Advocacy

Sunday, November 2, 2025

Adapting a Parent Testimony Video on the Impact of Childhood Cancer on Couples: Leveraging Artificial Intelligence for Inclusive Representation - Poster #559

Presenter: Nikita Guarascio, University of Motnreal

Other Authors: David Ogez, Katherine Pélouquin, Valérie Marcil, Daniel Curnier, Émélie Rondeau, Serge Sultan

ABSTRACT_ID#2437

ABSTRACT

A child's cancer diagnosis profoundly impacts the psychological well-being of parents. To alleviate parental distress, researchers developed and refined Taking Back Control Together, a manualized program to support parents navigating pediatric cancer. During the refinement process, a need for greater diversity in the Parent Testimony video was identified. However, diversity is multifaceted, encompassing gender, ethnicity, sexual orientation, culture, language, and more, making it difficult to represent all experiences through real videos. Moreover, using real testimonies raises ethical concerns. To address this, we aim to leverage artificial intelligence (AI) to generate video scenarios that reflect a broad range of backgrounds. We are collaborating with English- or French-speaking families whose child has completed cancer treatment and who identify with diverse cultural, sexual, socio-economic, gender, religious or geographic backgrounds. 1-3 parental couples are being guided to critically review the existing video and enrich its content to reflect diverse experiences. We are conducting semi-structured qualitative interviews, presenting the original video to family-partners and asking targeted questions to better understand their experiences. Based on their feedback, we will revise the script to incorporate key nuances that reflect inclusivity. We will use the adapted script to create AI-generated videos, which will be reviewed by family-partners. By engaging family-partners in identifying areas where original content lacks cultural relevance or emotional resonance, we expect them to provide valuable insights that will enhance the video's contextual sensitivity, accessibility, and relatability. This initiative may serve as a model for using AI to co-create inclusive psychosocial resources in pediatric oncology care.

LAY SUMMARY

In a previous refinement study, our research team discussed the importance of cultural representation in video content. We also recognize the need to represent sexually diverse couples and to consider how socio-economic barriers and varying levels of literacy may influence parents' experiences in navigating pediatric cancer. However, due to time and funding constraints, we did not have the means to produce new videos. To address this, we aim to collaborate with families from diverse backgrounds to revise the content and make it more inclusive. We will use artificial intelligence to create new videos that reflect these diverse perspectives. We anticipate that this approach will enable us to produce realistic, representative content, even with limited resources, thereby helping families feel seen, supported, and understood. Co-creating psychosocial resources with families ensures that interventions are grounded in real experiences, increasing their acceptability, relevance, and cultural sensitivity. Co-creation fosters trust and leads to more meaningful and inclusive support tools. Artificial intelligence offers a promising and cost-effective solution for creating new videos that reflect the lived experiences of a broader range of families.

Patient Experience and Advocacy

Monday, November 3, 2025

Beyond the Binary: Engaging Trans & Gender Diverse people to improve cancer health services research - Poster #560

Presenter: Morgan Stirling, University of Manitoba

Other Authors: Cath Poitras, Kael McKenzie, Kieren de la Rue, Jay Gallant, Julia Peterson, Benjamin Carroll, Tristan Bilash, Natalie Mulaire, Jenny Duong, Annette Schultz, Janice Ristock, Nathan Nickel, Alyson Mahar

ABSTRACT_ID#2791

ABSTRACT

Background. Community-engaged research is critical for addressing the inequities Trans and Gender Diverse (TGD) people experience across the cancer continuum. Despite the positive impact of engaged research, TGD people are rarely engaged in cancer research. This oversight increases TGD people's risk of harm in research (e.g. reinforcing damaging narratives about who TGD people are) and limits efforts to establish inclusive evidence-based care approaches. Engaging TGD people in cancer research supports developing equity-oriented models of care by privileging TGD people's voices and experiences. **Methods.** Beyond the Binary is a community-based, mixed-methods study investigating approaches for improving the inclusion and representation of TGD people in cancer health services research. A community advisory board comprised of 9 TGD individuals across Canada with various types of cancer experiences are active research partners guiding this study. Advisors receive an honorarium. Engagement is grounded in principles of partnership, co-learning, mutual benefit, and commitment. **Results.** Advisors actively participate in decision-making, priority-setting and developing study materials. We collaboratively created a terms of reference to guide our work together, which has included publishing a scoping review on TGD people's representation in cancer research and co-developing the study's data collection tools. Key to our success is establishing a supportive and transparent research environment, including employing flexible engagement methods to ensure all perspectives are heard. **Conclusion.** Engaging TGD people in cancer research is imperative for improving the evidence base. Lessons learned to foster engagement will be discussed. We will share recommendations that support researchers engaging TGD people in cancer research.

LAY SUMMARY

- Engaging Trans and Gender Diverse people in cancer research requires establishing relationships based on trust, respect, and reciprocity.
- Engaging Trans and Gender Diverse people significantly improves our ability to ensure this research matters to the community
- We think sharing our experiences will help other researchers with engaging Trans and Gender Diverse people in their own work.

Patient Experience and Advocacy

Sunday, November 2, 2025

Strengthening the role of the Canadian Cancer Society in addressing cancer misinformation - Poster #561

Presenter: Shefali Panicker, Canadian Cancer Society

Other Authors: Ariana Del Bianco, Elizabeth Holmes, Olivia Kulbak, Tracy Torchetti

ABSTRACT_ID#3197

ABSTRACT

Objective: Cancer misinformation and disinformation can lead to harm by distracting from known risk factors, delaying cancer screening, or promoting unproven treatments. This project aimed to (1) re-assess current efforts by the Canadian Cancer Society (CCS) to address cancer misinformation, (2) define what cancer misinformation means, and (3) develop a practical report with ways to respond to and reduce its spread. **Methods:** A mixed methods approach was used. A literature review examined existing definitions and strategies for addressing cancer misinformation, while an environmental scan identified common language and terms in the field. CCS's current efforts were evaluated by reviewing content on cancer.ca, conducting qualitative outreach with frontline teams who handle public inquiries, and analyzing Google webpage analytics to assess public interest and engagement with misinformation-related topics. **Results:** The project led to the development of a new organizational definition of cancer misinformation, updated language and messaging, and a practical toolkit to support teams at CCS. These tools provide guidance for CCS to respond more effectively and consistently, with preliminary evaluations informing ongoing improvements to cancer education and communication. **Conclusion:** CCS is now better positioned to counter cancer misinformation through an updated definition, refined messaging and actionable tools. This strengthened approach will enhance CCS' ability to deliver and improve reliable content on cancer.ca, bolster digital health literacy and empower Canadians to make informed health decisions.

LAY SUMMARY

Cancer misinformation and disinformation include false claims about cancer that are not supported by current evidence, such as ways to prevent, diagnose, treat or cure cancer. While both terms refer to false information, the key difference between misinformation and disinformation is the sender's intention. Misinformation is typically shared without mal-intent and a belief that it is true and useful. Disinformation is shared with intent to mislead, motivated by financial, ideological, religious, political or social agendas. Both can still cause harm. Checking facts, clearly marking false information, and sharing consistent messages help people trust cancer information. When reliable sources verify claims and label misinformation, it's easier to tell what's true and what's not. Being transparent and making sure health claims match what medical professionals and recognized health organizations say also builds trust and supports better decision-making. Organizations should plan and build response teams, form strong community partnerships and set up ways to monitor what's being said online before cancer misinformation spreads. This proactive preparation—knowing your audience, having clear strategies, and ready-to-go messaging—can help quickly counter cancer misinformation, maintain public trust and reduce harm.

Patient Experience and Advocacy

Monday, November 3, 2025

In-context learning for detection of cancer stigma in medical narratives - Poster #562

Presenter: Lisa YW Tang, University of British Columbia

ABSTRACT_ID#3273

ABSTRACT

Implicit bias in clinical documentation, particularly in oncology care, can subtly undermine the quality of patient-provider relationships and contribute to inequitable care outcomes. Negative descriptors such as "difficult patient" often reflect subjective judgments that may disproportionately affect marginalized groups. To address this challenge, we propose the development of a prototype system that leverages large language models (LLMs) to identify stigmatizing language in real time and suggest neutral, patient-centered alternatives. This system will be integrated into clinical documentation workflows to assist healthcare professionals in producing respectful, bias-aware records. The proposed system employs in-context learning, where a small set of examples is provided to LLMs during inference. We leverage publicly available datasets containing domain-specific oncology terms to evaluate the accuracy of bias detection. In the current prototyping stage, the system also presents suggestions for alternative wording alongside brief rationales, grounded in well-established frameworks for respectful clinical communication. Qualitative evaluation includes a blinded study involving professionals trained in knowledge translation and public health. Future work includes usability testing with clinicians to collect qualitative feedback. Our research and proposed system represent a step toward embedding equity-aware language tools into electronic medical record systems. When presented to medical students, the prototype will also serve as a training tool to promote bias recognition and respectful clinical writing. Once integrated into clinical practice and education, this system has the potential to improve the quality of care and foster more respectful, equitable oncology care.

LAY SUMMARY

Biased language in medical records can harm patient care: terms like "difficult patient" often reflect personal judgments by healthcare providers. These words can unfairly shape how other providers view and treat the patient, especially for those from marginalized groups. Our tool alerts doctors and medical trainees about potentially biased language found in a piece of input text: using large language models (LLMs), our system automatically detects stigmatizing words in real time as clinicians write patient notes. It then suggests neutral, respectful alternatives and most importantly, explains why the change is recommended. This tool can support both practicing doctors and medical students: in addition to improving clinical records, it will also help train future healthcare professionals to recognize and avoid biased language. By encouraging more respectful communication in patient charts, this work can help promote fairness and dignity in cancer care.

Exploring individual quality of life responses among pediatric cancer patients who participated in a tailored physical activity intervention: A visual analysis - Poster #564

Presenter: Amanda Wurz, University of the Fraser Valley

Other Authors: Emma McLaughlin, Eden Baerg, Jenson Price, S. Nicole Culos-Reed, Gregory M.T. Guilcher, Sara Fisher, Carolina Chamorro-Viña, Members of the Advisory Board (JW, LW, BP, and in memory of MP)

ABSTRACT_ID#2399

ABSTRACT

Introduction: IMPACT (IMplementation of Physical Activity for Children and adolescents on Treatment) is a 12-week tailored physical activity (PA) intervention for pediatric cancer patients. To gain an early understanding of potential effects, this visual analysis explored individual quality of life (QoL) responses to IMPACT. Methods: As part of an ongoing hybrid effectiveness-implementation trial, QoL (patient/caregiver validated questionnaire) and physical function (patient assessment) data were collected at baseline and post-intervention and experiences with IMPACT (patient/caregiver semi-structured interviews) were gathered post-intervention. Visual analysis prioritized qualitative data and involved dyadic-level coding to inform preliminary visualizations of patient's QoL responses and iterative refinements using QoL and physical function data. Results: Four female patients (5-14 years) diagnosed with acute lymphoblastic leukemia (n=3) or atypical teratoid/rhabdoid tumor (n=1) were included. IMPACT enhanced patients' emotional, social, and physical QoL by helping them: (i) feel more confident and joyful, (ii) experience support, and (iii) build/maintain strength; though nuances were evident. Three patients experienced briefly diminished emotional QoL post-intervention due to completing IMPACT and 2 patients experienced increases in physical QoL while 2 patients experienced modest improvements and could have been "pushed harder. Notable variations in QoL were described at baseline, and over the course of IMPACT, based on medical and other factors. Across single IMPACT sessions, despite prior apprehension and fatigue, patients recounted feeling better and more energized post-session. Conclusions: Results from this visual analysis highlight potential positive effects of IMPACT acutely and overtime, with variability in QoL responses. Findings offer early guidance for planned scaling efforts.

LAY SUMMARY

A 12-week physical activity program delivered during cancer treatment helped children build or maintain their strength and feel more confident, joyful, and socially connected. While all children benefited from the 12-week physical activity program delivered during cancer treatment, individual responses varied based on medical and personal factors. Each physical activity session provided an immediate mood boost, even when children felt tired or worried beforehand.

Altering CG2 levels by Pyrvinium reveals sensitivity to Navitoclax in Acute megakaryoblastic leukemia - Poster #565

Presenter: Caroline Capdevielle, Université de Montréal

Other Authors: Sarah St-Amand, Mathieu Neault, Sara Bouikni, Marilaine Fournier, Heather J. Melichar, Laura Hulea, Frédérick A. Mallette

ABSTRACT_ID#3163

ABSTRACT

Acute megakaryoblastic leukemia (AMKL) represents 10 % of pediatric acute myeloid leukemia. AMKL is frequently associated with mutations and the most frequent is the CBFA2T3::GLIS2 gene fusion (CG2). Unfortunately, CG2+ AMKL is associated with the poorest outcome and resistance to chemotherapy. In a mouse model of AMKL, CG2 expression is sufficient to induce leukemogenesis while altering the expression of BCL-2 family members. We therefore demonstrated the sensitivity of CG2+ AMKL to Navitoclax, a BCL-2 family inhibitor, in patient-derived xenograft (PDX) mouse model. Unfortunately, Navitoclax is not used clinically due to major side effects such as thrombocytopenia. Since CG2 plays an important role in leukemogenesis, affecting its function could create synergy with Navitoclax and lead to better therapeutic options. Pyrvinium is an FDA-approved anthelmintic drug described as a degrader of GLI family proteins via CK1 α -dependent mechanism. Given the similarities between CG2 and GLI proteins, Pyrvinium treatment decreases CG2 protein levels in AMKL cell line and is associated with decreased proliferation and induction of apoptosis (EC50 = 1.5 μ M). Pyrvinium-treated cells showed a reduction of CG2 transcriptional activity, as demonstrated by RNAseq. Navitoclax, used at lower doses in combination with Pyrvinium, synergistically induced apoptosis in vitro on CG2+ AMKL cell line and ex vivo in 2 different PDXs. SSTC3, a specific CK1 α agonist, also showed a decrease in CG2 at protein level and sensitizes cells to Navitoclax. Affecting CG2 levels in combination with Navitoclax could represent a promising therapeutic approach for pediatric AMKL patients who currently have limited treatment options.

LAY SUMMARY

Pyrvinium treatment reduces CG2 levels in a specific pediatric leukemia Navitoclax in combination with Pyrvinium increases the number of cancer cell deaths compared with treatment alone Regulation of CG2 is important to increase efficacy of combined treatment

Aberrant Inflammatory Response Associated with Chemotherapy-Resistance in T-Cell Acute Lymphoblastic Leukemia - Poster #566

Presenter: Oviní Amarasinghe, CancerCare Manitoba

Other Authors: Jesslyn Saw, Jacqueline A Boyle, Shokoufeh Abdollahi, David J Curtis, Cedric S Tremblay

ABSTRACT_ID#2326

ABSTRACT

Introduction: T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive subtype of leukemia, which is the most common childhood cancer. Past research comparing bone marrow samples from diagnosis and after treatment identified a population of therapy-resistant Relapse Inducing Cells (RICs) that drive T-ALL relapse. Understanding mechanisms that enable RICs to withstand chemotherapy will inform new and better treatments for T-ALL. Methods: More than 40% of T-ALL aberrantly express the oncogenic factor LMO2. The Lmo2Tg transgenic mouse model enables to study RICs, which emerge from the DN3 T-cell progenitor population during T-ALL development. To assess the effect of chemotherapy on RICs, we performed transplantation assays, RNA-seq and ATAC-seq following Chemotherapy (VXL) treatment or Vehicle. Results: Our preliminary data show that RICs found in preleukemic Lmo2Tg DN3 cells are enriched following VXL treatment. Transcriptomics data from Lmo2Tg DN3 cells showed that gene expression changes induced by VXL were transient. Pathway analysis revealed an aberrant Interferon (IFN)-gamma signature in Lmo2Tg DN3 cells at 24 hours after VXL treatment. ATAC-seq revealed increased chromatin accessibility to IFN Regulatory Factors (IRF) in therapy-resistant cells. To determine whether aberrant IFN-mediated signaling occurs early after induction chemotherapy, we assessed changes in Lmo2Tg DN3 cells at 24 hours after a single dose of VXL treatment. Conclusion: Changes induced by chemotherapy are transient, with VXL treatment selecting for chemoresistant RICs. Transcriptomic analysis of preleukemic Lmo2Tg DN3 cells revealed chemoresistance is associated with aberrant IFN-mediated signaling, suggesting IFN might promote chemotherapy-resistance in T-ALL.

LAY SUMMARY

Acute lymphoblastic leukemia (ALL) is the most common cancer in children, with the T-cell subtype (T-ALL) being particularly aggressive. Patients with T-ALL are treated with chemotherapy, that is quite toxic and induces lifelong side effects. Although most patients respond to treatment, some patients develop resistance to chemotherapy and T-ALL comes back within 2 years after diagnosis. My research project aims to understand why some T-ALL cases resist chemotherapy. Our discoveries will help to develop new ways to treat T-ALL for improving survival and quality of life of patients.

Delineating the roles of integrin $\alpha 6$ in central nervous system involved acute lymphoblastic leukemia - Poster #567

Presenter: Ami (T.T.Chau) Truong, University of British Columbia

Other Authors: Pascal Leclair, Ling Vicky Li, Gregor Reid, Chinten James Lim

ABSTRACT_ID#3072

ABSTRACT

Acute Lymphoblastic Leukemia (ALL) is the most common pediatric cancer, marked by the uncontrolled growth of immature lymphoblasts in the bone marrow. Despite advances in treatment, the 5-year survival drops from >85% to below 50% when leukemic cells invade the central nervous system (CNS). CNS-directed prophylactic chemotherapy administered to newly diagnosed ALL patients is effective at reducing rates of CNS-ALL, but often results in neurotoxicity, affecting quality of life. Integrins, particularly integrin $\alpha 6$ (CD49f), were shown to play crucial roles in cancer by regulating tumor cell trafficking and promoting drug resistance. While the correlation of $\alpha 6$ with leukemia persistence to chemotherapy is known, its specific roles in leukemia progression and CNS invasion are not fully characterized. We investigated the roles of $\alpha 6$ -integrins in CNS trafficking of NALM6, a neurotropic B-precursor ALL cell line. CRISPR-mediated ablation of $\alpha 6$ reduced broad-spectrum cell adhesion and SDF-1-directed chemotactic migration. In vivo studies in NSG mice revealed that loss of $\alpha 6$ significantly delayed systemic leukemia progression and CNS burden involving various CNS entry pathways. Furthermore, longitudinal monitoring and postmortem analysis suggested that CNS invasion by NALM6 ALL is initiated by early stage leukemic accumulation in the spinal and calvarial bone marrow. These findings elucidate mechanisms driving CNS invasion by leukemic cells, highlighting the importance of integrin $\alpha 6$ in promoting this phenomenon and the potential of utilizing integrin $\alpha 6$ as a target for diagnostic and prognostic strategies in CNS-ALL.

LAY SUMMARY

Acute Lymphoblastic Leukemia (ALL) is the most common cancer diagnosed in children. It arises from uncontrolled growth of immature white blood cells in the bone marrow. When leukemic cells are found to invade the central nervous system (CNS), patients' overall survival rate is reduced from 85-90% to below 50%. It is still unknown what properties of a leukemic cell predispose it to invade the CNS, and treatment options are limited and prone to brain damage. $\alpha 6$ -integrins are specialized adhesion proteins that facilitate cell binding to laminin, a matrix component that is abundant in CNS tissues. It is known to be correlated with leukemia chemotherapy persistence, and it is suspected that high levels of $\alpha 6$ expression promote leukemic cells to invade the CNS. However, direct evidence linking $\alpha 6$ -integrins to CNS-ALL is still lacking. My study found that: Integrin $\alpha 6$ plays crucial roles in facilitating cell adhesion and cell migration of CNS-invasive leukemic cells. Animals bearing cells that lack integrin $\alpha 6$ developed leukemia at a slower pace and had fewer leukemic cells in the brain and spinal cord. The CNS can be breached at early disease stages of leukemia, long before it can be detected in the patient's cerebral spinal fluid.

Integrative Transcriptomic Profiling of Metastatic Medulloblastoma using Patient-Derived Xenograft Models - Poster #568

Presenter: Ana Isabel Castillo Orozco, McGill University | Research Institute of the McGill University of Health Center (RI-MUHC)

Other Authors: Geoffroy Danieau, Livia Garzia

ABSTRACT_ID#3080

ABSTRACT

Medulloblastoma (MB) is a highly aggressive and the most common brain tumor in childhood. MB presents a high intertumoral heterogeneity, with at least four molecular subgroups identified (SHH, WNT, Group 3, and Group 4). Metastatic MB, or Leptomeningeal Disease (LMD), is predominantly found in the MB Group 3 type. Although LMD represents a main clinical challenge, its molecular mechanisms remain poorly characterized. Recent research has shown that primary and MB metastasis diverge dramatically. Our work has focused on establishing therapy naïve Group 3 Patient-Derived Xenografts models of primary and metastatic Medulloblastoma to conduct transcriptomic profiling at the bulk and single-nuclei RNAseq levels to identify genetic drivers/pathways that sustain leptomeningeal disease compartment. Our results show various signaling pathways enriched across LMD models, such as MYC targets, unfolded protein response, and fatty acid metabolism. Using single-sample GSEA analysis (ssGSEA) and deconvolution approaches, we have also identified that our PDXes models retain neoplastic subpopulations previously identified in MB single-cell sequencing studies. Similarly, we have identified slight differences in cell subpopulation proportions between primary and leptomeningeal compartments. Our single-nuclei studies have confirmed these results and differentially expressed genes previously found in bulk RNAseq analyses. These results suggest the presence of cell populations enriched in the metastatic compartment with an aberrant transcription phenotype and adaptations in metabolism to survive the leptomeningeal space. Our recent findings suggest that LMD should be treated differently from primary brain tumors and that identified metabolic pathways may be potential targets for targeted therapeutics to treat or prevent this devastating disease.

LAY SUMMARY

Medulloblastoma is an aggressive pediatric brain tumor that originates mainly from the cerebellum. Unfortunately, when it metastasizes, cancer cells disseminate along the brain coverings (meninges). These tumors spread much like butter on bread. Accordingly, these tumors cannot be surgically removed, which makes it very hard to obtain samples for research. Samples from autopsies are equally complex due to ethical and logistical challenges. Unfortunately, metastatic medulloblastoma is fatal, and no progress has been made in the last 30 years. In our lab, we have developed mice models that can faithfully recapitulate the behavior of metastatic medulloblastoma, allowing us to study it and compare it against primary brain tumors (before it metastasizes). We have demonstrated that primary and metastatic medulloblastoma exhibit significant differences in gene expression, which can provide insights into how to treat metastasis more effectively and design targeted therapies to prevent it. We have utilized state-of-the-art technologies, such as single-cell transcriptomics, to make robust comparisons between primary and metastatic tumors and reveal molecular differences at the cellular level. We have identified molecular patterns and populations enriched in the metastatic compartment, suggesting an aggressive behaviour characterized by aberrant hyper-transcription and exacerbated protein production.

Targeting Non-Genetic Kinase-Dependent Signaling Pathways to Prevent Medulloblastoma Tumor Relapse - Poster #569

Presenter: Ujala Chawla, University of Manitoba

Other Authors: Helgi Kuzmychova, Emma Martell, Akaljot Gerwal, Charul Jain, Tanveer Sharif

ABSTRACT_ID#2440

ABSTRACT

Medulloblastoma (MB) is the most common malignant pediatric brain tumour, classified into four molecular subgroups: WNT, SHH, G3, and G4. Among these, G3-MB is associated with the worst prognosis, with 5-year survival rate of approximately 60%. Despite intensive treatment, including surgery and chemoradiotherapy (CRT), ~30% of patients relapse with treatment-resistant tumours. Notably, relapsed tumours often retain their original molecular identity, suggesting non-genomic mechanisms, such as dysregulated cell signaling, may drive relapse and therapy resistance. We hypothesize that aberrant kinase signaling adaptations contribute to CRT resistance in G3-MB. To model this, we treated G3-MB cell lines and orthotopic patient-derived xenograft (PDX) mouse models with a clinically relevant CRT regimen. Phospho-kinase arrays and immunohistochemistry revealed increased activation of phosphorylated SRC (p-SRC) and its downstream targets in CRT-resistant (CRT-Res) G3-MB cells, but not in SHH-MB or normal human brain cells, indicating a subgroup and tumour-specific response. Functional studies using genetic knockdown and FDA-approved SRC inhibitors demonstrated that blocking p-SRC significantly reduced cell viability, stemness, and self-renewal. Mechanistically, G3-MB cells exhibited dual response to loss of p-SRC, where a subset of cells underwent apoptosis, while others shifted towards more differentiated phenotype. Importantly, therapeutic relevance of these findings are demonstrated by significantly reduced tumour burden and prolonged survival in PDX mouse model treated with combination of CRT and SRC inhibitor *in vivo*. These findings identify SRC signaling as a critical driver of therapy resistance and recurrence in G3-MB. Targeting SRC may represent a promising therapeutic strategy for improving outcomes in children with recurrent, treatment-resistant G3-MB.

LAY SUMMARY

Group 3 medulloblastoma (G3-MB) is a severe form of childhood brain cancer that often returns even after surgery, chemotherapy and radiation. Our research shows that relapses may not be due to new genetic mutations but instead to changes in how tumor cells respond to treatment through altered signaling pathways. We discovered that a protein called SRC becomes much more active in G3-MB tumors that survive treatment, but not in normal brain tissue or other types of medulloblastoma. This protein acts like a survival switch, helping cancer cells avoid death and maintain their ability to regrow and resist therapy, ultimately making harder to eliminate the disease. Using both lab-grown cells and animal models, we blocked SRC using drugs already approved for other diseases. When combined with standard treatment, these drugs reduced tumor growth, triggered cancer cell death, and extended animal survival. This suggests that adding SRC inhibitors to current therapies could be a promising strategy for treating relapse in children with G3-MB.

Pediatric Cancer

Monday, November 3, 2025

Exploring Dissemination of the international Pediatric Oncology Exercise Guidelines (iPOEG) Toolkit - Poster #570

Presenter: Emma McLaughlin, University of Calgary

Other Authors: Colleen Cuthbert, Amanda Wurz, S. Nicole Culos-Reed

ABSTRACT_ID#2512

ABSTRACT

Background: The international Pediatric Oncology Exercise Guidelines (iPOEG) provide evidence-based information to help pediatric cancer patients and survivors move more. To translate this evidence to pediatric cancer patients and survivors, caregivers, exercise professionals, healthcare providers, and support organizations, we created the iPOEG Toolkit comprised of resources (posters, infographics). To explore dissemination of the iPOEG Toolkit, reach and intended implementation were assessed. **Methods:** Dissemination is assessed by tracking the number of emails sent, presentations delivered, and downloads from the website and social media engagement (reach) and gathering demographic and intended use information via an optional post-download survey (intended implementation). Data from (08/2022-05/2025) were reviewed and analyzed descriptively. **Results:** Twelve emails were sent to >250 experts, 15 presentations have been conducted (n=4 lay, 9 academic), and 4 social media posts were made which were shared (n=7), liked (n=35), and/or reposted (n=3). Posters and infographics were downloaded 53 and 87 times, respectively. Eight individuals responded to the post-download survey (n=4 exercise professionals, n=2 healthcare providers, n=2 researchers) and indicated hearing about the Toolkit via emails (n=4), colleagues (n=2), publication (n=2), presentations (n=1), or social media (n=1). Respondents intended to use and share the Toolkit by printing and posting within their workplace, sharing with patients and families, reposting on social media, and translating to other languages. **Discussion:** Limited reach highlights a need for more concerted dissemination efforts. Identifying and contacting relevant pediatric oncology research and clinical groups and support organizations may be warranted. Further, better understanding of intended and actual implementation is still required.

LAY SUMMARY

The international Pediatric Oncology Exercise Guidelines Toolkit was developed to promote exercise guidelines and recommendations to end-users (e.g., pediatric cancer patients and survivors, caregivers, exercise professionals, healthcare providers, support organizations). Informative posters and infographics have been downloaded 53 and 87 times, respectively. Professionals intend to use the Toolkit in their context by printing the resources and posting them at their workplace, sharing with patients and families, reposting on social media, and translating to other languages.

Canadian Landscape of Treatment Approaches for Children with Relapsed or Refractory High-Risk Neuroblastoma - Poster #571

Presenter: Gabrielle Herzenberg, University of British Columbia

Other Authors: Daniel Morgenstern, Rebecca Deyell

ABSTRACT_ID#2344

ABSTRACT

Background: Despite intensified upfront therapy and routine use of immunotherapy for high-risk neuroblastoma (HRNB), children with refractory or relapsed (R/R) disease continue to face dismal outcomes. There is no standardized approach to R/R neuroblastoma, and treatment strategies vary widely, including conventional chemotherapy, chemoimmunotherapy, high-dose ^{131}I -MIBG, and enrollment in early-phase clinical trials, which are limited. Aims: We aimed to assess treatment approaches and drug access issues for R/R HRNB across Canada. Methods: In preparation for a national meeting on HRNB, we surveyed pediatric oncologists across Canada to identify practice patterns. Results: Of 39 oncologists approached, 30 (77%) responded, representing all 16 Canadian pediatric oncology programs. For patients with poor response to induction therapy, 97% (n=29) would consider chemoimmunotherapy with dinutuximab/irinotecan/temozolomide, and 33% (n=10) would consider high-dose ^{131}I -MIBG prior to consolidation. Chemoimmunotherapy was the preferred initial salvage regimen for 90% of respondents. Two oncologists (7%), both in Ontario, reported difficulty accessing dinutuximab for multiply relapsed disease. The number of chemoimmunotherapy cycles offered varied significantly, with a median of 3 cycles (range 2–17) without, and 16 cycles (range 5–18) with radiographic response. Allogeneic stem cell transplant was routinely offered by 10% and considered by an additional 20%. Conclusions: This survey highlights variability in the management of R/R HRNB across Canadian treatment centers. Despite widespread chemoimmunotherapy use as a preferred salvage regimen, significant variability remains in the number of cycles of this costly therapy. In our single payer healthcare system, there is a need to establish evidence-based treatment guidelines for patients with R/R neuroblastoma.

LAY SUMMARY

Children with relapsed or treatment-resistant high-risk neuroblastoma still face very poor outcomes, even though treatments have improved for newly diagnosed patients. There is no single, standard approach that doctors follow when the cancer comes back or doesn't respond. Most pediatric oncology doctors in Canada use a combination of chemotherapy and immunotherapy (called chemoimmunotherapy) as their first choice when treating relapsed disease. However, this is a costly therapy, requires inpatient hospital stays and there are big differences in how many treatment cycles patients receive, ranging from just a few to many. Access to certain treatments is mostly good across the country, but some doctors—particularly in Ontario—reported challenges getting the targeted antibody drug, dinutuximab, for children whose cancer had returned multiple times.

Pediatric Cancer

Monday, November 3, 2025

Training and Education Program Implementation at The Hospital for Sick Children's Clinical Trial Support Unit (CTSU) - Poster #572

Presenter: Jennifer Kim, The Hospital for Sick Children

Other Authors: Saudia Khan

ABSTRACT_ID#2730

ABSTRACT

Background: The pandemic led to many changes within the CTSU at SickKids, including reduced on-site staff support and introducing remote processes for work-from-home support. Amidst transitioning to a hybrid work model, the lack of streamlined processes for training current and newly hired staff fell upon each disease groups' Senior CRA. This led to increased staff turnover and feedback about insufficient support. In response, the Training and Education (T&E) program was born. **Purpose:** The T&E program was created in June 2023 to have a nominated T&E lead(s) streamline processes, answer questions, and provide consistent training for CRAs across all disease groups. **Methods:** Staff training was tracked in 2023/2024 using a Master Staff Training Record created for CTSU staff, which encompassed training topics that new and current staff required to complete their daily tasks. Training topics were further organized and offered based upon length of time in their role (0-3, 6 and 12+ months). After 6 months, we sought anonymous feedback to gauge the program's benefits and areas of improvement. **Results:** Since June 2023, 15 new staff were trained on 38 topics, including 20+ COG-related topics, with relevant resources offered. Of the 13 anonymous survey responses, 69% found the training sessions very helpful, while 31% found it somewhat helpful. Overall, 92% found the content of the sessions relevant to their daily workload. **Conclusions:** Implementing a T&E program with designated Lead(s) focused on topics relevant to CRA needs has successfully streamlined processes, improved work quality and provided overall support to CTSU staff.

LAY SUMMARY

Problem: The pandemic caused work changes and confusion in training new and current staff at SickKids' Clinical Trial Support Unit, leading to staff feeling unsupported and more people leaving. **Solution:** In June 2023, a new Training and Education (T&E) program was started with special leads to organize training, answer questions, and provide consistent support for all research staff. **Outcome:** Since starting, the program trained 15 new staff on many key topics, with most staff saying the training was helpful and relevant, improving overall support and work quality.

Developing and evaluating novel otoprotective agents to mitigate cisplatin-induced ototoxicity - Poster #573

Presenter: Lissandra Tuzi, Children's Hospital of Eastern Ontario/University of Ottawa

Other Authors: Nadine Azzam, Asna Latif, Aaron Fox, Erin M. Fitch, Ted Allison, Frederick G. West, Amit P. Bhavsar, Jason N. Berman

ABSTRACT_ID#2520

ABSTRACT

Cisplatin is an invaluable and effective chemotherapeutic treatment for many pediatric solid tumours that contribute to the overall ~80% five-year survival rate for childhood cancer patients. The major caveat of cisplatin is its various adverse effects, including nephrotoxicity and ototoxicity, which may result in lower cisplatin dosing or discontinuation. Cisplatin-induced ototoxicity (CIO) occurs in >50% of patients. Therapeutic prevention of cisplatin-induced hearing loss by 'otoprotectants' would reduce toxicity, while enabling ongoing cisplatin administration for therapeutic benefit. Sodium thiosulfate has recently been FDA approved as a CIO otoprotectant, but with restricted eligibility. Therefore, developing novel effective treatments to prevent or mitigate CIO remains critical. We have identified activation of Toll-like receptor 4 (TLR4) by cisplatin as a pro-inflammatory process, which contributes to ototoxicity, leading to the development of a selective inhibitor of the metal-induced activation of TLR4, without affecting other TLR4 inducers. This selective inhibitor reduced cisplatin damage of murine inner ear hair cells in vitro, suggesting potential otoprotective properties. We then evaluated if this compound negatively impacted cisplatin effectiveness in larval zebrafish xenografts, employing A549 lung cancer cells, SF188 glioblastoma cells, SKNAS neuroblastoma cells, and a primary neuroblastoma patient sample. Importantly, the addition of the specific inhibitor did not interfere with cisplatin cancer cytotoxicity in vivo. Thus far, this study indicates that targeting TLR4 using a selective inhibitor can reduce cisplatin toxicity, while retaining anti-cancer therapeutic effectiveness. These findings provide compelling preclinical data, suggesting an innovative potential strategy to balance cisplatin's anti-tumour effects and hearing loss, meriting further investigation.

LAY SUMMARY

Cisplatin is a highly effective chemotherapy for many pediatric solid tumours, contributing to an ~80% five-year survival rate in childhood cancer. However, its use is limited by side effects like hearing loss (cisplatin-induced ototoxicity, CIO), which affects over 50% of patients and may require dose reduction or treatment discontinuation. While sodium thiosulfate was recently FDA-approved to prevent CIO, its use is restricted, highlighting the need for new protective strategies. We identified that cisplatin activates Toll-like receptor 4 (TLR4), triggering inflammation that contributes to CIO. A novel, selective inhibitor targeting only the metal-induced activation of TLR4—without affecting other inducers of TLR4—was developed. This compound reduced damage to inner ear hair cells in vitro and did not compromise cisplatin's cancer-killing effects in larval zebrafish models bearing lung cancer, glioblastoma, and neuroblastoma xenografts. These findings suggest that selective TLR4 inhibition may protect hearing without reducing cisplatin's anti-tumour activity, offering a promising new approach to enhance cancer treatment safety and effectiveness.

Incidence and Mortality trends in Childhood Cancer in British Columbia, 1971-2020 - Poster #577

Presenter: Muntasir Rahman, BC Cancer Research Institute

Other Authors: Zhiran Tong, Rachel Altman, Mary De Vera, Karen Goddard, Fuchsia Howard, Joan Hu, Agnes Lai, Andrea Lo, Jeenat Mehareen, Helen McTaggart-Cowan, Justin Oh, Rod Rassekh, Caron Strahlendorf, Meera Rayar, Stuart Peacock

ABSTRACT_ID#3359

ABSTRACT

Introduction: Childhood cancers are one of the leading causes of disease-related deaths in Canadian children aged 0 to 14 years. Despite the severity, research into long-term trends of childhood cancers in British Columbia (BC) remains limited. This study aims to provide a demographic overview and analyze trends in childhood cancer incidence and mortality in BC. **Data and method:** Data were obtained from the BC Cancer Registry (1971–2020) to construct the Childhood, Adolescent, and Young Adult Cancer Survivor (CAYACS) database. Age-standardized incidence rates (ASIR) and mortality rates (ASMR) per million Canadian population for 12 major cancer groups were calculated. Joinpoint trends analyses were conducted to estimate Average Annual Percentage Change (AAPC) and Annual Percentage Change (APC). **Results:** Between 1971 and 2020, 5,067 childhood cancer cases were recorded in BC, with higher incidence in males (54.4%). Most diagnoses (45.4%) occurred among children aged 0–4 years. The most common cancers were leukemias (average ASIR=48.1), lymphomas (ASIR=14.7), and central nervous system (CNS) neoplasms (ASIR=33.1). A significant upward trend in overall incidence was observed (APC 0.42; $p<0.05$), with significant ($p<0.05$) increases in leukemias between 1971-2020, CNS neoplasms between 1971 to 2015, and malignant bone tumors between 1971-1989. There were 1,033 deaths, with the highest mortality rates from leukemias (ASMR=10.2), CNS neoplasms (ASMR=9.4), and neuroblastoma (ASMR=3.6). Overall mortality showed a significant decline (APC -1.94; $p<0.05$). **Conclusion:** Rising cancer incidence among BC children emphasizes the need for further research into the causes. Enhanced understanding may support earlier diagnoses and more targeted treatments to improve outcomes.

LAY SUMMARY

Research on long-term trends in childhood cancer in British Columbia (BC), Canada, remains limited, potentially obscuring important patterns and disparities in incidence and outcomes. Our analysis reveals a rising incidence of childhood cancers in BC over the study period (1971–2020), with notable increases observed in leukemias, central nervous system (CNS) neoplasms, and malignant bone tumors. In contrast, mortality rates have declined steadily during the same timeframe. Given that childhood cancer survivors typically face long-term late effects over a longer lifespan than other age groups, the need for ongoing cancer care is expected to grow significantly. Therefore, understanding these incidence and mortality trends is critical for anticipating future healthcare demands and informing policy decisions aimed at addressing the evolving burden of childhood cancer care in BC.

Pediatric Cancer

Monday, November 3, 2025

ACCESS: Advancing Childhood Cancer Experience, Science and Survivorship in Canada - Poster #578

Presenter: Stephanie Grover, ACCESS / The Hospital for Sick Children

Other Authors: Christine Williams, Adrienne Co-Dyre, James Whitlock, David Malkin

ABSTRACT_ID#2315

ABSTRACT

Advocacy for enhanced childhood cancer research and care in Canada by patients, families and the academic community led to federal funding to establish a national pediatric cancer consortium ('ACCESS'). ACCESS' mission is to ensure every child with cancer in Canada has access to the latest scientific advances, diagnostic tools, therapies, and supportive care leading to better outcomes and quality of life. ACCESS is a unique collective of diverse, multi-experiential and multi-expertise stakeholders (scientists, healthcare providers, industry leaders, partner organizations, advocates, patients, and their families), who are conducting research, changing policy, and developing new tools together to ensure better lives for children who experience cancer in Canada and for their loved ones. The goal is to implement transformative change in the delivery of fair, fast, effective, and safe cancer care for all children. ACCESS is driven and led by people with lived experience (PWLE) of pediatric cancer and framed by the patient journey. To date, ACCESS has unified a national community. It has: connected a community of >900 people and stimulated a culture of open communication in which diverse viewpoints, including those of PWLE, are sought and valued; built productive and effective partnerships with other organizations; initiated innovative national research studies that impact all aspects of the patient journey; produced accessible communications materials. Future priorities include achieving organizational sustainability, supporting collaborations and inclusivity, overcoming barriers that affect the access of children to diagnostics and treatment, enhancing data connectivity and sharing, and developing and implementing a national strategy for pediatric cancer.

LAY SUMMARY

National childhood cancer network that is a unique collective of scientists, healthcare providers, industry leaders, partner organizations, advocates, patients, and their families who are conducting research, changing policy, and developing new tools to ensure better lives for children who experience cancer in Canada and for their loved ones. The mission of ACCESS is to ensure every child with cancer in Canada has access to the latest scientific advances, diagnostic tools, therapies, and supportive care leading to better outcomes and quality of life. ACCESS is working to develop national best practices standards and tools for research and care suited to address the complex policy, regulatory, and practical barriers that prevent equitable access to cancer diagnosis, treatment and care for all children with cancer in Canada.

Preclinical Safety and Activity of ATRA and Bafilomycin-A1 in Juvenile Zebrafish: Toward Differentiation and Autophagy-Targeting in ARMS - Poster #579

Presenter: Roham Saberi, University of Manitoba

Other Authors: Bhavya Bhushan, Amirmohammad Eshtiaghi, Kharisma Del Mundo, Benjamin Lindsey, Saeid Ghavami

ABSTRACT_ID#2882

ABSTRACT

Background: Alveolar rhabdomyosarcoma (ARMS) is malignant pediatric cancer characterized by impaired myogenic differentiation and chemotherapy resistance. Beyond its homeostatic role, autophagy modulates tumor cell differentiation and survival under treatment stress. Simultaneously promoting differentiation and inhibiting autophagy may reduce tumor aggressiveness and improve therapeutic outcomes. **Rationale:** All-trans retinoic acid (ATRA) promotes myogenic differentiation, while Bafilomycin A1 (Baf-A1) inhibits autophagic flux by blocking lysosome-autophagosome fusion. We hypothesized that in vivo modulation of these pathways could define a safe and effective therapeutic window. Juvenile zebrafish provide a scalable model to assess drug efficacy and systemic tolerability. **Objective:** To evaluate the biological effects and safety of ATRA and Baf-A1 on muscle differentiation, autophagy inhibition, proliferation, and juvenile zebrafish behavior. **Methods:** Zebrafish at 21 days post-fertilization were treated every 72 hours for 14 days with ATRA (5, 10, 20 μ M) or Baf-A1 (2.5, 10 nM). Group size ($n = 5$) was based on power analysis ($\geq 20\%$ effect, 80% power, $\alpha = 0.05$). Behavioral (velocity, distance moved) and morphological (length, eye size) endpoints assessed toxicity. MyoD immunofluorescence measured differentiation, LC3/SQSTM1 puncta quantified autophagy via confocal microscopy, and EdU labeling assessed proliferation. ANOVA with Tukey's test assessed significance. **Results:** ATRA (20 μ M) increased MyoD expression and nuclei density ($p < 0.01$). Baf-A1 (10 nM) elevated LC3/SQSTM1 puncta ($p < 0.001$) and EdU+ cells ($p < 0.05$). No toxicity observed ($p > 0.05$). **Conclusion and Next Steps:** ATRA and Baf-A1 are well-tolerated and active in vivo. These results establish dosing parameters for future combination studies in ARMS models.

LAY SUMMARY

1. Improving muscle cell development may help treat aggressive childhood cancers like alveolar rhabdomyosarcoma (ARMS). This cancer resists treatment partly because the tumor cells don't develop into mature muscle cells properly.
2. We tested two drugs in young zebrafish to see if they could help. One drug (ATRA) encourages muscle cell development, and the other (Baf-A1) blocks a process called autophagy, which cancer cells often use to survive stress.
3. Both drugs worked safely in zebrafish. ATRA increased markers of muscle development, and Baf-A1 blocked autophagy and increased cell growth without impacting Juvenile Zebrafish behaviour and development. These findings support future testing of the drug combination to improve treatment options for ARMS.

Exploring the prenatal genetic determinants of tumour risk in Li-Fraumeni syndrome: sequencing of newborn screening dried blood spots - Poster #580

Presenter: Tanvi Anandampillai, The Hospital for Sick Children

Other Authors: Laura Raiti, Anita Villani, Adam Shlien, David Malkin, Yiming Wang

ABSTRACT_ID#3139

ABSTRACT

Li-Fraumeni Syndrome (LFS), caused by germline pathogenic variants in TP53, is a heritable cancer predisposition syndrome with a lifetime cancer risk approaching 100%. LFS is linked to a broad spectrum of early-onset cancers, including but not limited to brain cancer, breast cancer, osteosarcomas and soft-tissue sarcomas. There is broad phenotypic heterogeneity in the age of onset and tumour type, even in affected family members with identical TP53 variants. We have shown that this heterogeneity is associated with other germline genomic/epigenomic variants and somatic variants. Surprisingly, loss of heterozygosity (LOH) of TP53, a key driver of tumorigenesis, is predicted to arise years before tumour diagnosis. Considering the early onset of tumours in LFS, it is likely that the LOH of TP53 and other somatic variants arise prenatally with further clonal evolution after birth, which may dictate postnatal cancer risks in these patients. To explore the origin of these prenatal cancer driver mutations, we will perform genome sequencing and comprehensive cancer panel sequencing (864 cancer-associated genes) of newborn screening dried blood spots (NBS DBS) from 20 LFS patients and 5 healthy controls. We will focus on TP53 LOH and somatic cancer driver mutations, accompanied by global mutational landscape analyses. These findings will be compared to sequencing data from matched postnatal germline and tumour samples, readily accessible for >7 LFS patients in our cohort. Our study has the potential to impact surveillance practices and uncover new links between prenatally acquired mutations and pediatric cancers, revealing novel approaches for early diagnosis, prevention, and interception.

LAY SUMMARY

The developmental origin of pediatric cancers has captivated the scientific community for decades. Studies in newborn screening dried blood spots (NBS DBS, taken by heel prick at 24-48h after birth) from children later diagnosed with leukemia showed that some leukemia genetic changes emerge prenatally. However, this has never been studied in solid tumors or cancer predisposition syndromes (CPS) such as Li Fraumeni Syndrome (LFS). With the recent advancements in sequencing technologies, we can study the NBS DBS from LFS patients on a genomic scale. We have previously characterized the genetic changes present in the tumor and other normal tissues of patients with LFS followed at The Hospital for Sick Children. Using this unique patient cohort and bio/databank, we are studying the NBS DBS to explore whether the genetic changes driving tumour development later in life can also be found at the time of birth. Correlated with clinical data, we aim to define the tumor risks during childhood and adulthood with the landscape of genetic changes in neonates. This study has the potential to revolutionize the current timeline of surveillance and intervention for pediatric cancer predisposition syndromes such as LFS.

Identifying new metabolic vulnerabilities in CBFA2T3::GLIS2+ acute megakaryoblastic leukemia - Poster #581

Presenter: Sarah St-Amand, Université de Montréal

Other Authors: Caroline Capdevielle, Mathieu Neault, Alejandro Schcolnik-Cabrera, Zaynab Nouhi, Laura Hulea, Frédérick A. Mallette

ABSTRACT_ID#2875

ABSTRACT

The CBFA2T3::GLIS2 (CG2) chimera is seen in aggressive pediatric acute megakaryoblastic leukemia (AMKL). CBFA2T3 is normally expressed in hematopoietic cells and plays a role in stem cell maintenance and self-renewal. On the other hand, GLIS2, a transcription factor, is not expressed in normal hematopoietic cells. Recently, metabolic adaptations have gathered attention for their therapeutic potential. However, the metabolic reprogramming caused by CG2 and the role of CG2 and its interactors in this rewiring remains unexplored. RNA sequencing of CG2-driven tumors in a mouse model of AMKL revealed a dysregulation of several genes related to pyruvate metabolism. Steady-state analysis of metabolite levels by GC-MS showed an accumulation of pyruvate, aspartate and GABA. Deeper investigations in AMKL human cell lines showed that mitochondrial respiration and glycolysis were affected by the CRISPR-Cas9-mediated knockdown of GLIS2. Based on the transcriptomic data, we investigate the role of PDK2 in AMKL. Using PDK2 shRNA or AZD7545, a PDK inhibitor, we observed a decrease in cell proliferation 7 days post-infection or treatment. To understand how CG2 affects metabolism, we characterized the CG2 interactome by BioID. We discovered numerous transcription factors and chromatin modifiers, including lysine demethylase KDM4A. Targeting by shRNA or QC6352, a KDM4 inhibitor, has a marked negative effect on cell proliferation and synergizes with Navitoclax, a drug already validated as effective in our AMKL models. We propose that CG2 induces metabolic rewiring of hematopoietic cells by a transcriptional role to promote leukemogenesis. These vulnerabilities, undescribed in AMKL, represent potential therapeutic targets to treat this dreadful disease.

LAY SUMMARY

Leukemic cells adapt their metabolism to sustain rapid growth. Directly targeting some of these changes can be a potential therapy. Targeting the metabolic changes makes cells more sensitive to other drugs.

Defining CBFA2T3-GLIS2 transcriptional complexes in Acute MegaKaryoblastic Leukemia - Poster #582

Presenter: Veronica Gueye, Université de Montréal

Other Authors: Mathieu Neault, Caroline Capdevielle, Frédérick Antoine Mallette

ABSTRACT_ID#2923

ABSTRACT

Context: Acute MegaKaryoblastic Leukemia (AMKL) is a rare blood cancer mainly affecting children. It is caused by genetic mutations affecting bone marrow cells, the most common of which is the fusion of the CBFA2T3 and GLIS2 genes, two transcriptional regulators. This fusion results in the formation of CBFA2T3-GLIS2 (CG2) chimeric oncogene protein, which maintains cells in an undifferentiated state. However, due to a still insufficient knowledge of the mechanisms by which CG2 promotes leukemia, current therapies fail to eradicate the disease or ensure medium- and long-term survival. **Hypothesis / objectives:** We therefore propose that unravelling the CG2 interactome will help us to advance our understanding of these molecular mechanisms consequently open therapeutic(s) avenue(s). **Methodology:** We performed BioID experiments followed by gene ontology analysis. These analyses revealed a strong association of CG2 with proteins involved in transcriptional regulation and SUMOylation. **Results and discussion:** Interestingly, proteins involved in SUMOylation account for more than a third of the putative partners of CG2 either as enzymes involved in SUMOylation of target proteins or as target proteins for the various SUMO isoforms. We have also shown that inhibition of SUMOylation reduces cell proliferation and partially facilitates differentiation of leukemia cells. **Conclusion:** Together, this work will enable us to evaluate SUMOylation inhibition as a targeted therapy for patients suffering from CG2 positive AMKL. It will also provide us with a better molecular understanding of the mechanisms involved in promoting and maintaining an undifferentiated state in leukemia.

LAY SUMMARY

CG2's interactions point to SUMOylation: By tagging all the proteins that are in vicinity with the leukemia-driving CG2 fusion protein, we found that about one-third are linked to SUMOylation a process that adds SUMO to proteins and helps control their molecular mechanism. Inhibiting SUMOylation slows leukemia cells: When we used drugs to inhibit SUMOylation, the leukemia cells stopped multiplying and show a decrease of stemness marker c-Kit, suggesting that SUMOylation might be essential fuel for CG2-positive leukemia. A new treatment angle for children with AMKL: Because CG2-positive leukemia respond to SUMOylation inhibition in vitro, targeting SUMOylation related pathway could become a focused therapy that spares healthy cells while undermining the leukemia's growth engine.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Polymorphisms in the GSTA1 and UGT2B17 Genes, Estrogen Levels, and Menopause-Related Side Effects in Post-Menopausal Women in the MAP.3 Trial - Poster #583

Presenter: Harriet Richardson, Queen's University

Other Authors: Marisa Deodat, Vikki Ho, Philip Lazarus, Shaman Luo

ABSTRACT_ID#2673

ABSTRACT

Background: Exemestane (EXE), an aromatase inhibitor, lowers breast cancer risk in postmenopausal women but may lead to menopause-related toxicities, including fatigue, hot flashes, and joint pain. Genetic polymorphisms in GSTA1 and UGT2B17 may influence EXE/estrogen metabolism and toxicity severity. Understanding these influences could help personalize breast cancer prevention. Objectives: Among women randomized to EXE in the MAP.3 trial, this study aims to: Assess associations between GSTA1 polymorphisms and estrone (E1) and estradiol (E2) suppression at 1-year. Assess the main effect of GSTA1, and the joint effect of GSTA1 and UGT2B17 polymorphisms on menopause-related toxicities up to 1- and 5-years. Methods: This nested cohort study used data from MAP.3, a double-blind, placebo-controlled phase III trial. Estrogen levels were measured via liquid chromatography-mass spectrometry. Toxicities were assessed using the Common Terminology Criteria for Adverse Events v3.0 criteria. Log-binomial regression examined genotype associations with estrogen suppression and toxicities. Confounders were identified a priori, and models were stratified by race. Results: Compared to GSTA1*A/*A (wildtype), those with GSTA1*B/*B had higher risk of E2 suppression (RR1yr=1.51, 95% CI: 1.11–2.06) and severe joint pain (RR1yr=3.94, 95% CI: 1.22–12.71), while GSTA1*A/*B had an increased risk of fatigue (RR1yr=1.36, 95% CI: 1.04–1.78; RR5yr=1.29, 95% CI: 1.04–1.61) and severe joint pain risk (RR1yr=3.81, 95% CI: 1.32–10.96). No associations were found for hot flashes. Interaction analyses are ongoing. Conclusion: GSTA1 polymorphisms are associated with estrogen suppression, fatigue, and severe joint pain in EXE-treated women, highlighting potential for personalized breast cancer prevention.

LAY SUMMARY

Not all women respond the same way to breast cancer prevention medication. Exemestane is a medication used to lower estrogen levels and reduce the risk of breast cancer in postmenopausal women. While it can be very effective, it can also cause side effects like fatigue, hot flashes, and joint pain in some women. Our research explores why certain women experience more severe side effects than others. Genes involved in drug metabolism may influence estrogen levels and side effects. We studied women who took exemestane as part of a large trial. Those with specific versions of a gene called GSTA1 had bigger decreases in estrogen after one year. These same women were also more likely to report fatigue and severe joint pain during treatment. Another gene, UGT2B17, also plays a role, and we are exploring how these genes interact. This research could help tailor breast cancer prevention to the individual. By understanding how a woman's genetic makeup affects her response to treatment, we can move toward more personalized care. In the future, genetic screening could help identify women who are more likely to benefit from exemestane with fewer side effects, or guide alternative options to reduce risk more safely.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Precision cancer prevention: unraveling heterogeneity in dietary intake and metabolic health - Poster #584

Presenter: Alyssa D. Milano, BC Cancer

Other Authors: Sharon Kirkpatrick, Jennifer Vena, Parveen Bhatti, Ace Chan, Benoît Lamarche, Sarah Neil-Sztramko, Erin Bussin, Giulia Menichetti, Michael Wallace, Dylan Spicker, Ellen Sweeney, Silvia Stringhini, Rachel Murphy

ABSTRACT_ID#2859

ABSTRACT

Background: Poor quality diets are among the leading causes of cancer in Canada. Cancer prevention recommendations provide general dietary guidance without considering the range of factors that influence dietary intake and metabolism of foods and nutrients. To develop more effective interventions and policies, we need population-based studies that integrate multidimensional data from diverse populations. Objective: The purpose of the CHARacterizing heterogeneity in dietary intake among structurally excluded populations using Multidimensional data (CHARM) study is to understand how structural, environmental, social, and biological factors contribute to population-level differences in diet quality and metabolic health. Methods: This study will use dietary, socio-demographic, and geospatial environment data from ~100,000 participants in the HEALthy eating and supportive environments (HEAL) study, which is nested in the Canadian Partnership for Tomorrow's Health. Blood and stool samples will be collected in a subset of participants (N=5,000, 75% structurally excluded). Structurally excluded groups are defined as participants with lived/living experience of discrimination, marginalization, and/or oppression. Metabolomics and measures of metabolic health (adiposity, triglycerides, serum lipids, HgbA1C) will be assessed. Data-driven methods will be used to identify factors associated with dietary intake. Intersectional analyses will be applied to determine how associations are modified by social positions and identities. Results: Data and biospecimen collection are ongoing and expected to be completed in 2027. Conclusions: The CHARM study will provide foundational evidence on how a range of factors interact to shape dietary intake and metabolic health. Our findings can inform more precise dietary recommendations tailored to population needs and cancer prevention.

LAY SUMMARY

Cancer prevention recommendations largely use a one-size-fits all approach for dietary guidance that overlooks other factors that influence dietary intake and metabolic health. CHARM will utilize multi-dimensional data, including unique biospecimen data, from a large cohort of Canadians to apply an intersectional analysis, identifying how factors across environmental, social, structural, and biological systems influence differences in dietary intake, diet, and metabolic health. The findings from CHARM may help inform more precise dietary recommendations for cancer prevention, including for structurally excluded populations who have a higher burden of diet-related disease, including cancer.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Multi-omic Dissection of Lymph Node Metastasis in Oral Squamous Cell Carcinoma - Poster #585

Presenter: Ayan Chanda, University of Calgary

Other Authors: Daniel Young, Steven C. Nakoneshny, Thomas Wayne Matthews, Shamir Chandarana, Robert D Hart, Joseph C Dort, Antoine Dufour, Martin D Hycza, Pinaki Bose

ABSTRACT_ID#2793

ABSTRACT

Lymph node metastasis (LNM) is a critical prognostic factor in oral squamous cell carcinoma (OSCC), yet the molecular events underpinning this process remain insufficiently understood. We performed a comprehensive multi-omic analysis of 60 primary OSCC tumors with matched adjacent normal tissues, stratified by nodal involvement. This study is a part of the Marathon of Hope Cancer Centres Network (MOHCCN) Gold Cohort. Each sample was profiled using whole genome sequencing (WGS), whole transcriptome sequencing (WTSeq), Assay for Transposase Accessible Chromatin by sequencing (ATAC-seq), mass spectrometry-based proteomics, and metabolomics screen. This dataset enabled the identification of coordinated alterations linked to metastatic progression. Leveraging multi-omic factor analysis (MOFA2), we identified latent factors that distinguish tumors with LNM from those without. These factors revealed underlying regulatory programs and variation patterns not apparent within individual data modalities, highlighting the value of unsupervised integrative approaches in revealing hidden biological structure. In parallel, recurrent non-coding somatic mutations were identified in LNM-positive tumors. These variants are enriched in regions outside canonical coding sequences, overlapping with known transcription factor binding sites and regions of altered chromatin accessibility. By combining large-scale data across molecular domains, our study aims to define robust, biologically informed signatures of LNM in OSCC. This integrative framework not only supports mechanistic hypothesis generation but also holds promise for the identification of biomarkers relevant to risk stratification and therapeutic targeting. This work underscores the importance of systems-level approaches in oncology, where multifactorial processes like metastasis are best understood through a composite lens of multi-omics.

LAY SUMMARY

We studied how oral cancers spread to nearby lymph nodes. In oral squamous cell carcinoma (OSCC), cancer that spreads to lymph nodes (called lymph node metastasis, or LNM) is linked to worse outcomes. However, the exact biological reasons behind this spread are not well understood. We used a multi-layered approach to study 60 patient tumors. By combining different types of data—including DNA, RNA, protein, and chromatin (which helps regulate genes)—we examined tumors with and without lymph node involvement. This allowed us to see how different molecular changes work together to influence cancer progression. We discovered patterns that may explain why some tumors spread. Using different computational analyses, we found hidden molecular signatures that distinguish tumors with LNM. We also identified non-coding DNA mutations (changes outside of genes) that may affect how genes are turned on or off. These findings help us better understand how cancer spreads and may lead to better tools for predicting risk or developing new treatments. Our goal is to use this research to improve how we identify, monitor, and treat aggressive forms of oral cancer.

Precision Medicine and Biomarkers

Monday, November 3, 2025

UGT2B17 Cytoplasmic Expression in Primary Tumors Predicts Lethal Prostate Cancer: Multi-Cohort Insights - Poster #586

Presenter: Ashwini Uchil, Université Laval

Other Authors: Louis Lacombe, H       Hovington, Herv            , David Simoyan, Patrick Caron, V            , Mathieu Latour, Armen Aprikian, Alain Bergeron, Simone Chevalier, Ginette McKercher, Fadi Brimo, Ladan Fazli, Neil Fleshner, Martin Gleave, Pierre I. Karakiew

ABSTRACT_ID#2436

ABSTRACT

Background: Prostate cancer (PCa) is hormone-dependent, with UGT2B17 playing a central role in androgen inactivation. This study aimed to evaluate whether UGT2B17 expression in prostatectomy specimens can serve as a prognostic marker for lethal PCa. Methods: A prespecified hypothesis posited that UGT2B17 expression (>25%) in primary tumors is associated with an aggressive disease phenotype, leading to metastasis, castration-resistance (CRPC), and mortality in men initially diagnosed with localized disease. Two high-density prostate tumor tissue microarray (TMA) datasets were analyzed: the first from the Canadian Prostate Cancer Biomarker Network biobank (CPCBN; n=1454) and the second from the PROCURE cohort (n=1562). Kaplan-Meier and Cox proportional hazard ratio analysis were used to evaluate metastasis-free survival, CRPC and prostate cancer specific mortality (PCSM). Steroid levels were measured in plasma samples by mass spectrometry, and a linear regression model was used to evaluate variations in hormone levels based on tumoral UGT2B17 expression. Results: UGT2B17 was associated with prognostic factors and linked to elevated androsterone glucuronide levels (AST-G; 60%), the major circulating androgen-inactive metabolite, which is inactivated by UGT2B17. Kaplan-Meier and multivariable Cox analyses revealed that higher tumoral UGT2B17 is associated with an increased risk of progression to metastatic/CRPC stages and with PCSM. Conclusion: UGT2B17 expression influences hormone levels and identifies a subset of patients at increased risk of progression to an incurable disease stage. Findings support the notion that enhanced UGT2B17, likely through increased androgen inactivation, creates a low-androgen tumor environment that drives tumor progression to a more aggressive phenotype.

LAY SUMMARY

Prostate cancer is hormone dependent cancer, and an aggressive subtype evolves from a low androgen environment. Our analysis in over 3000 patients identified a subset of patients with high expression of UGT2B17, a protein that inactivates androgens. We found that higher UGT2B17 levels to be linked to; Increased risk of disease progression, including metastasis and castration resistant prostate cancer. Shorter time to progression and higher rates of mortality from prostate cancer. Changes in hormone levels, suggesting a shift to aggressive cancer phenotype. We suggest UGT2B17 measure at the early stage, could be a useful biomarker that can be used to identify the high-risk patients of lethal prostate cancer and help guide further treatment decisions.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Slow and Steady Wins: Prognostic Implications of Response Kinetics in Multiple Myeloma - Poster #587

Presenter: Arvind Mer, University of Ottawa

Other Authors: Ben Patrick, Amirhosein Ebrahimi, Alissa Visram

ABSTRACT_ID#3053

ABSTRACT

Introduction: In multiple myeloma (MM), emerging evidence suggests that differential treatment response kinetics (DTRK)-the speed of achieving a clinical response to initial therapy-may influence patient outcomes. However, systematic evaluation of DTRK and its genomic associations remains limited. This study addresses these gaps. **Methods:** Patients were classified as fast or slow responders based on achieving at least a partial response within 4 months of first-line therapy. Overall survival (OS) hazard ratios (HRs) were calculated using Cox proportional-hazard regression. Genomic associations with DTRK were identified via bioinformatics analysis. Tumor microenvironment composition was evaluated using single-cell data analysis. A robust machine-learning approach was employed to develop a DTRK gene signature. **Results:** In the CoMMpass cohort (n=709), slow responders had significantly improved outcomes (OS HR=0.66, p<0.001). This finding remained robust even after adjusting for clinical covariates and was validated in an independent cohort (The Ottawa Hospital cohort, n=754). Multi-omics analyses showed enrichment of genomic alterations in critical MM-related (TP53) or stemness-related (AURKB, TAX1BP3) genes and upregulation of cell-cycle, stemness, and differentiation pathways in fast responders. Slow responders had increased NK-cells and T-lymphocytes, whereas fast responders exhibited elevated plasma cells and monocyte precursors. The machine-learning-derived DTRK gene signature was successfully validated in three independent cohorts. **Conclusion:** Our analysis across multiple cohorts robustly establishes DTRK as a significant prognostic marker in multiple myeloma. We also found that distinct genomic and immune profiles are associated with treatment response kinetics. Our DTRK gene signature offers promising clinical utility for personalized multiple myeloma treatment strategies.

LAY SUMMARY

Multiple myeloma patients who responded more slowly to treatment had better long-term outcomes. Fast responders had more harmful genetic mutations and more aggressive disease, while slow responders showed stronger immune activity. A gene signature was developed to predict response speed and support personalized treatment.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Identifying Clinically-Relevant Genetic Predictors of Morphine Response in Children with Cancer - Poster #588

Presenter: Catrina Loucks, University of British Columbia

Other Authors: Erika Scott, Jia He Zhang, Rena Daniel, Graeme Ernest-Hoar, Julia Charlton, Alice van Zanten, Jessica Lovnicki, Colin Ross, Rod Rassekh, Bruce Carleton, Ruth Grunau

ABSTRACT_ID#2480

ABSTRACT

Opioids (e.g., morphine) are commonly prescribed for pain relief in children with cancer; however, responses vary from ineffectiveness to adverse effects. Variation in several genes has been associated with opioid response in the literature; however, insufficient/inconclusive results have hindered the development of pharmacogenetic tests to predict/prevent undesirable responses. To identify strong predictors of opioid responses, we recruited 152 opioid-treated patients from across Canada and identified children with extreme responses to morphine (i.e., little to no pain relief) who are most likely to carry genetic variants with large effects. We then performed pharmacogenomic analyses (adjusting by age/ancestry) focused on genetic variants previously associated with morphine response in children identified through a systematic review. These analyses validated a previous association with rs4680 G, which increases the activity of the COMT enzyme mechanistically involved in opioid-based pain relief (odds ratio [OR]=2.44, 95% confidence interval [CI]=1.25-4.76, $p=0.009$) as well as an association with rs17863783 T, which increases the expression of the drug metabolizing enzyme UGT1A6 (OR=15.07, 95% CI=1.64-138.3, $p=0.02$). Few studies have assessed genetic associations with opioid response in children. Our findings, in a large pediatric patient population, represent an exciting validation of the association between higher COMT activity (increased COMT-mediated neurotransmitter metabolism) and poor opioid response and suggest that patients with UGT1A6 rs17863783 might experience poor opioid responses due to increased opioid metabolism. Validation of published genetic associations will help to facilitate development of predictive pharmacogenetic tests to guide dosing/analgesic choice in children with cancer to limit their burden of pain.

LAY SUMMARY

Children with cancer frequently experience highly-distressing pain requiring opioids (e.g., morphine), yet many children experience inadequate pain relief that may be attributable to individual genetic factors. A genetic variant that increases the activity of the COMT enzyme mechanistically involved in opioid-based pain relief more than doubles the chance of inadequate morphine-based pain relief in children with cancer, while a genetic variant that increases the expression of the drug metabolizing enzyme UGT1A6 increases the risk of morphine ineffectiveness by 15 times. Few studies have assessed genetic associations with opioid response in children, and our findings, in a large pediatric patient population of 152 opioid-treated children recruited from across Canada, will help to facilitate development of predictive genetic tests to guide dosing/analgesic choice in children with cancer to limit their burden of pain.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Use of Automated Ki67 Analysis and Machine Learning Models to Predict Oncotype DX Risk of Recurrence Categories in Early-Stage Breast Cancer - Poster #589

Presenter: Angela MY Chan, Alberta Health Services

Other Authors: Angela MY Chan, Md Mahsin, Satbir Singh Thakur, Mie Konno, Kata Osz, Holly Wilson, Tasnima Abedin, Haocheng Li, Roxana Tudor, Gilbert Bigras, Don Morris, Emeka Enwere, Hua Yang, Etienne Mahe, Paola Neri

ABSTRACT_ID#2758

ABSTRACT

Breast cancer treatment is mainly determined by its molecular subtype classified based on key driver genes. Additional assays, such as Ki67 proliferation index and gene expression analysis, including Oncotype DX, may be used to refine the prognostication and prediction of chemotherapy benefit. Despite strong evidence supporting its use, Oncotype DX is a costly assay. Hence, there is a strong motivation to develop alternative inexpensive strategies to minimize its clinical use. Ki67 positivity is a major contributor to the calculation of the Recurrence Score (RS) for Oncotype DX, and the Ki67 proliferation index itself is a predictive biomarker for the use of CDK4/6 inhibitor in lymph node-positive breast cancer. However, the reliable and reproducible scoring of Ki67-positive cells is challenged by the high inter- and intra-observer variability observed in the manual scoring method. To overcome this limitation, we employed unbiased Ki67 digital image analysis in conjunction with clinical and pathological variables and developed a random forest machine learning (ML) model to predict the Oncotype DX RS with high accuracy, sensitivity, and specificity. Since there is no unique ML model that outperforms others in this type of prediction circumstance, this study also compares various machine learning models, including Support Vector Machine, Extreme Gradient Boosting, and Elastic Net Regression, to identify the most effective model for predicting the Oncotype DX RS. This study features one of the largest patient cohorts of this type (n=1381), and the model developed can significantly reduce health system costs while maintaining accuracy in breast cancer testing.

LAY SUMMARY

Reduce health system cost: The development of a robust, reproducible, and cost-effective machine learning model to predict the Oncotype DX Recurrence Score offers an inexpensive alternative to the costly Oncotype DX assay while maintaining high accuracy. **Use of readily available resources:** The machine learning model utilizes information available in the patient charts and laboratory tests that are already part of the breast cancer testing workflow. **Comparison of Machine Learning Models:** Different machine learning models are compared to find the best prediction model to use in a large population of early-stage breast cancer patients.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Validation of Biobanked Sample Quality following an Extended Longitudinal Study - Poster #590

Presenter: Danielle Simonot, Alberta Cancer Research Biobank

Other Authors: Christine Friedenreich, Lin Yang, Farit Vakehtov, Pamela Round, Kerry Courneya, Darren Brenner, Yibing Ruan, Leanne Dickau, Dylan O'Sullivan, Teena Makowichuk, Shannon Brown, Tracy Xu

ABSTRACT_ID#2831

ABSTRACT

Alberta Cancer Research Biobank (ACRB) is a provincial biobank that collects, processes and stores biospecimens for cancer and health research. Between 2012 and 2023, ACRB assisted the Alberta Moving Beyond Breast Cancer (AMBER) study to collect samples from 1528 breast cancer participants from Calgary and Edmonton at 3 timepoints: baseline (diagnosis), 1- and 3-years of follow-up. While all samples were collected, processed and stored using standardized ACRB protocols, variability of sample characteristics was expected due to the length of the study, age of the samples, length of time from blood draw to processing and freezing and sample handling by different provincial laboratories. Analysis of serum samples was conducted for 3 biomarkers (C-reactive protein, insulin and glucose) by Alberta Precision Laboratories. To minimize variation during analysis, samples were organized such that duplicate participant samples for all timepoints were included on one plate, alongside control and replicate QC samples. Samples were randomly assigned to boxes to disperse variation in sample age, location and processing characteristics. Including all participant and control samples, 11,922 data points were received. Despite anticipated variation due to sample characteristics, most notably sample age, initial analysis demonstrated remarkably little variation in the intra-and inter-CV results for all analytes (<0.05). The largest inter-batch variation amongst the 3 biomarkers (3%) is likely due to testing variation by the lab. ACRB and AMBER study have demonstrated that with proper collection, processing and biostorage protocols, biobanked samples can maintain high quality biomarker results even if stored for more than 10 years.

LAY SUMMARY

The Alberta Cancer Research Biobank assists researchers to collect, process and store biospecimens for cancer and health research. Standardized biobank protocols for sample collection, processing and storage have been demonstrated to maintain exceptionally high sample quality following an extended longitudinal study, with sample age ranging up to 12 years.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Androgenic Effects of 11-oxyandrogens in Castration-Resistant Prostate Cancer - Poster #591

Presenter: Ariane Lapointe-Belleau, Faculty of pharmacy Université Laval; CHU de Québec - Université Laval

Other Authors: Michèle Rouleau, Lyne Villeneuve, Joseph Descarreaux, Patrick Caron, Caroline Beaudoin, Ashwini Uchil, David Simonyan, Cylia Dahmani, Charles Joly-Beauparlant, Vincent Gélinas, Arnaud Droit, Nicolas Marcoux, Vincent Castonguay, Louis Lacombe, Éric Lévesqu

ABSTRACT_ID#2450

ABSTRACT

Background and Objective: Adrenal-derived 11-oxygenated androgens (11-oxyandrogens) emerged as potential key contributors to prostate cancer (PCa) progression through their biotransformation into potent androgen receptor (AR) agonists. The objective was to obtain clinical and mechanistic evidence of 11-oxyandrogens contribution in patients with metastatic castration-resistant prostate cancer (mCRPC) initiating AR signaling pathway inhibitors (ARPI). **Methods:** A pilot study of 35 mCRPC evaluated serum steroids via mass spectrometry and their association with survival using Kaplan-Meier curves. The capacity of 11-oxyandrogens in driving PCa cell proliferation and its reversal by enzalutamide was assessed, supported by comprehensive transcriptomic and proteomic analyses in CRPC cells. **Key Findings and Limitations:** We highlight 11-ketotestosterone (11KT) and its hydroxylated derivative, 11-hydroxytestosterone (11OHT) as the predominant potent androgens, accounting for 81% of circulating total androgens. Despite the limited sample size, higher baseline levels of 11KT, 11OHT, the abundant precursor 11 β -hydroxyandrostenedione (11OHA4) and the downstream metabolite 11-hydroxyandrosterone (11OHA5), are associated with prolonged progression-free survival (PFS) (hazard ratios (HRs) ranging from 0.56 to 0.69; $P < 0.05$), suggesting an enhanced treatment response. 11KT and 11OHT drive robust AR-mediated proliferative responses of CRPC cells and modulate expression profiles, paralleling canonical androgens. The AR antagonist effectively inhibits their androgenic actions, suggesting they are primarily AR-mediated. Additionally, 11-oxyandrogens display distinct effects, including the activation of potential AR-independent pathways. **Conclusions and Clinical Implications:** These findings show that 11-oxyandrogens are drivers of AR activation and support the clinical relevance of 11-oxyandrogen profiling to help predict response to ARPI in mCRPC, underlining the need for further investigation.

LAY SUMMARY

We studied the contribution of non-canonical androgens originating from the adrenal gland, named 11-oxygenated androgens, in patients with metastatic castration-resistant prostate cancer (mCRPC) initiating treatment. We found that circulating non-canonical 11-oxygenated androgens predominate in mCRPC patients and that patients with higher circulating levels of 11-oxygenated androgens exhibit a better response to treatment and demonstrate improved survival outcomes. We also observed that 11-oxygenated androgens display a strong androgenic influence on cancer cells, by stimulating proliferation and by inducing a shift in gene and protein expression, with some distinctive effects from classical androgens. Profiling androgens including 11-oxygenated androgens may help predict how patients respond to therapies, highlighting their clinical relevance and the need for further study.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Multi-omics analysis suggests that the long non-coding RNA NRAD1 dysregulates RNA splicing and mitochondrial function in triple-negative breast cancer - Poster #592

Presenter: Hannah Cahill, Dalhousie University

Other Authors: Jaganathan Venkatesh, Raj Prenap Arun, Marie-Claire Wasson, Meghan McLean, Justin Brown, Mukhayyo Sultonova, Dan Vidovic, Patrick Murphy, Paola Marcato

ABSTRACT_ID#3199

ABSTRACT

Introduction: Triple negative breast cancer (TNBC) is an aggressive breast cancer subtype associated with distinct gene expression profiles, worse patient outcomes, and needs new therapies. Increased understanding of the factors that lead to aberrant gene expression in TNBC will lead to novel treatment solutions. Long non-coding RNAs (lncRNAs) regulate gene expression in a variety of ways. Our work investigates a TNBC-enriched lncRNA, non-coding RNA in the aldehyde dehydrogenase 1A pathway (NRAD1) and its mechanisms of gene regulation that lead to TNBC progression. **Materials and Methods:** We performed multi-omics analyses, including proteomics, RNA sequencing (RNA-seq), chromatin Isolation by RNA purification (CHIRP)-seq, assay for transposase-accessible chromatin using sequencing (ATAC-seq) and RNA-pulldown assays on TNBC MDA-MB-468 and SUM149 cells, with or without NRAD1 knockdown. Gene ontology (GO) enrichment was performed on the NRAD1 regulated and bound proteins. Motif enrichment was performed on ATAC-seq and CHIRP-seq data, as well as overlaying with the RNA-seq data to determine RNA-splice sites and transcript variants. **Results:** Knockdown of NRAD1 altered the expression of hundreds of genes and proteins connected to cancer promoting pathways, concordant with altered chromatin structure and chromatin binding of NRAD1. GO enrichment of the proteomics and RNA-pull down data analysis suggested that NRAD1 both regulates and is bound to proteins that function in non-coding RNA metabolic processes, mitochondrial processes, and RNA splicing. **Conclusion:** Together, this work reveals new mechanistic information into how dysregulated lncRNA-chromatin networks result in the aberrant gene expression of TNBC leading to disease progression.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Association of NMR-based metabolomics and lung cancer risk in the UK Biobank - Poster #593

Presenter: Beiwen Wu, University of Toronto Dalla Lana School of Public Health

Other Authors: Joanne Kotsopoulos, Jennifer Brooks, Rayjean Hung

ABSTRACT_ID#3148

ABSTRACT

Background Lung cancer remains a global public health challenge. Advances in metabolomic profiling allow for the identification of biomarkers for early detection but evidence remains scarce. **Methods** We included 267,099 participants (2,089 lung cancers) from the UK Biobank, with 251 metabolites (170 absolute and 81 ratio measures) measured at baseline. Multivariable Cox proportional hazards models were used to estimate the hazard ratios (HRs) between each metabolite and lung cancer risk, adjusting for demographics and established confounders. P-values were corrected for multiple testing. Principal component analyses (PCA) were performed separately on absolute and ratio measures. PCs explaining 90% of the variance in each set were incorporated into Cox models to assess their combined effects on lung cancer risk. **Results** A total of 169 metabolites (102 absolute and 67 ratios measures) were found to be significant. Among absolute measures, glycoprotein acetyls [HR per SD=1.19, 95%CI=1.14-1.25], pyruvate, lactate, components of extremely large VLDL (excluding cholesteryl esters), and triglycerides in very large VLDL and HDL subclasses were associated with increased risk [HRs ranging from 1.05-1.09]. PCs driven by VLDL, HDL, and ketone bodies (PC1-PC3, PC6) showed positive associations; PC8 and PC9 (glycolysis and fatty acid-driven) showed inverse associations. For ratio measures, phospholipid and triglyceride percentages across various lipoprotein subclasses showed increased risk [HRs ranging from 1.06-1.18]. PCs driven by LDL/VLDL/IDL and fatty acids (PC1, PC5, PC7-PC9) showed positive associations; PC4 (HDL -driven) showed inverse associations. **Conclusion** These findings highlight complex metabolic patterns associated with lung cancer risk, warranting further investigation through pathway analyses.

LAY SUMMARY

Blood metabolites can help detect lung cancer early. In a large UK Biobank study of over 267,000 participants, we found that certain blood metabolites - particularly those related to inflammation (e.g., glycoprotein acetyls), energy metabolism (e.g., pyruvate and lactate), and lipid compositions across various lipoprotein subclasses (e.g., triglycerides in extremely large VLDL, very large VLDL, medium HDL, and small HDL) - were associated with a higher risk of developing lung cancer. The development of lung cancer involves more complex metabolic changes than individual markers. Using principal component analysis (PCA), we identified broader metabolic signatures associated with lung cancer risk. These findings highlighted the complexity of metabolic patterns and supported the use of pathway-based approaches for more accurate biomarker discovery and early detection.

Precision Medicine and Biomarkers

Monday, November 3, 2025

FTSJ3 changes epigenetic and spatial nuclear architecture in telomerase overexpressing cells - Poster #594

Presenter: Jared Price, University of Saskatchewan

Other Authors: Yue Zhang, Frederick Vizeacoumar, Vincent Maranda, Mary Lazell Wright, Liliia Kyrlenko, Andrew Freywald, Franco Vizeacoumar

ABSTRACT_ID#3236

ABSTRACT

The holoenzyme, telomerase, is frequently overexpressed in most cancer types and is generally associated with tumor survival. As a highly overexpressed biomarker, it implicates itself as a potential therapeutic target. Despite this potential, its direct inhibition has yet to produce a clinically relevant therapy against cancer. Here, we propose a novel strategy called synthetic dosage lethality (SDL) to overcome the deficiencies of previous efforts and apply it to models of telomerase over-expressing cancers. The identification of SDL interactions is of therapeutic interest because if a particular gene exhibits SDL with a tumor-driving molecular alteration, then targeting this SDL gene will cause lethality selectively in cancer cells harboring this alteration. The Vizeacoumar lab has utilized a pooled lentiviral CRISPR and shRNA screen against the entirety of the human genome to identify gene knockouts which was cross-referenced with cell line specific gene essentiality data from DepMap to create a short list of potential targets. These targets were then stratified according to canonical function, drugability, loss-of-fitness specificity, and essentiality. Here, I validate our top candidate gene, FTSJ3, as a telomerase-overexpression SDL partner. I identify key novel phenotypes dependent on FTSJ3 expression in telomerase positive and negative cell lines which likely contribute to the telomerase-specific loss of fitness. This work is exceptionally exciting as this target may have tumor-agnostic applicability due to the near-ubiquitous nature of telomerase expression in malignant solid tissue and key absence in normal somatic tissue.

LAY SUMMARY

Telomerase is a cancer-specific biomarker found in 85% of solid tumors. A key protein, FTSJ3, may be inhibited and cause cell death only in cells that express telomerase, therefore sparing normal cells. FTSJ3 loss is toxic in telomerase over-expressing cells by causing genome disorganization and overall genetic instability.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Expanding Access to Precision Oncology Through Quality-Controlled Whole Genome and Transcriptome Analysis from Routine FFPE Samples - Poster #595

Presenter: Connor Frey, BC Cancer

Other Authors: Jing Xu, Erin Pleasance, Melissa McConechy, Stephen Yip, Steven Jones, Marco Marra, Janessa Laskin

ABSTRACT_ID#3132

ABSTRACT

Background: Formalin-fixed paraffin-embedded (FFPE) tissue presents technical challenges for whole genome and transcriptome analysis (WGTA). We assessed the feasibility of WGTA using FFPE within the Personalized OncoGenomics (POG) program. Leveraging FFPE would promote equity in precision oncology. Methods: We analyzed 190 FFPE specimens across 26 tumour types and 8 hospitals using WGTA; benchmarking performance by comparing a subset of 44 matched FFPE and fresh-frozen (FF) biopsies to assess alignment of clinically relevant variants. Whole genome and RNA-Seq libraries were constructed using Illumina paired-end sequencing to 80X WGS coverage and 200 million RNA-Seq reads. Using these matched datasets, we developed quality control metrics to identify poor quality libraries, particularly within gene expression analysis. We refined nucleic acid extraction protocols and bioinformatic workflows. Results: In 44 matched FF/FFPE samples, FFPE libraries detected 90% of therapeutically relevant single nucleotide variants, 87% copy number variants, structural variants and transcriptomic profiling. Optimized FFPE specific analysis enabled 93.2% of total reports, including 110 cases (57.9%) reporting both DNA and RNA alterations. Partial analysis for 77 cases (35.3%) including 66% DNA only, and 33% with RNA expression disclaimers. Only 4.7% samples completely fail. DNA and RNA demonstrate differences in quality, RNA shows a marked decline, especially with increased storage time. Factors including tumour type, biopsy site and hospital of origin are needed to optimize FFPE quality. Discussion Our data support the use of FFPE for WGTA. High concordance with FF samples highlights the potential of FFPE to broaden access to precision oncology.

LAY SUMMARY

Plain Language: Archived cancer tissue, commonly stored as formalin-fixed paraffin-embedded (FFPE) tissue, can be used for precision cancer next-generation sequencing methods. This study found that FFPE samples closely matched fresh-frozen tissue in detecting selected clinically important mutations, copy number changes, structural variants, and gene expression patterns. Whole-genome and transcriptome analysis (WGTA) results from FFPE samples were successfully integrated into clinical workflows via molecular tumour boards, enabling real-time decision-making in patient care. Notably, the median number of clinically relevant variants identified from FFPE tissue was equivalent to that derived from fresh-frozen samples across both genomic and transcriptomic analyses. While DNA from FFPE samples is usually high quality for genomics, RNA quality drops over time. Careful quality checks and attention to tumour type, biopsy location, and storage conditions are needed to ensure accurate transcriptomic data.

Precision Medicine and Biomarkers

Monday, November 3, 2025

A qualitative exploration of patient and clinician perspectives for homologous recombination deficient (HRD) biomarker testing to guide precision medicine for advanced ovarian cancer treatment - Poster #596

Presenter: Julia McKenna, BC Cancer

Other Authors: Yelena Aizenberg, Melica Brodeur, Mark Carey, Sarah Ferguson, Laura Hopkins, Iwa Kong, Janice Kwon, Susie Lau, Donna Pepin, Karla Willows, Christa Wood, Helen McTaggart-Cowan

ABSTRACT_ID#2634

ABSTRACT

Background: Homologous recombination deficiency (HRD) is the result of a dysfunctional DNA repair process, leading to apoptosis of cancer cells. HRD testing can provide diagnostic information for people with advanced ovarian cancer and their medical team to guide the management of their disease. Currently, HRD testing is not publicly funded for all Canadians due to concerns regarding the impact on decision-making that key participants have about HRD testing. Objective: To explore the knowledge, attitudes, and experiences of advanced ovarian cancer patients and clinicians regarding HRD testing. Methods: We conducted pan-Canadian one-to-one, semi-structured interviews with patients and clinicians. Guided by our earlier literature review and the Health Belief Model, the topic guide was co-developed with patient research partners. Interview topics included current knowledge of HRD testing, barriers and facilitators, experience with test result interpretation, and suggestions for future rollout. Interviews were transcribed and thematically analyzed. Results: Data collection and analysis are underway. Preliminary findings revealed positive perceptions towards HRD testing. Facilitators to HRD testing included patients' value for information about their cancer and patients' desire for strong relationships between patient and providers. Perceived barriers led patients to feeling uninformed about their decision to take the HRD test. Patients desired more resources (e.g., pamphlets, websites) and more formal test result interpretation to help inform decision-making processes. Conclusion: Findings will inform future research action plans, including the improvement towards standardizing the nationwide HRD testing process. By increasing awareness and equitable access to HRD testing across Canada, this will improve informed patient decision-making.

LAY SUMMARY

HRD testing is a valued biomarker test for advanced ovarian cancer patients in informing future treatments, but its rollout process requires standardization and better inclusion of informative resources to improve patient-informed decision making processes.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Differential predictive roles of chemotherapy response score for Olaparib and Niraparib in treatment of tubo-ovarian high-grade serous carcinomas - Poster #597

Presenter: Christa Aubrey, University of Alberta

Other Authors: Preety Nijjar, Elenna LaPlante, Carol Ewanowich, Martin Koebel, Sophia Pin, Cheng-Han Lee

ABSTRACT_ID#3133

ABSTRACT

Introduction: PARPi has revolutionized the treatment of high-grade serous tubo-ovarian carcinomas (HGSC) with Olaparib used as adjuvant maintenance therapy for BRCA1/2 mutated HGSC and Niraparib primarily for BRCA1/2 wild-type (wt) HGSC. We aim to evaluate the association between chemotherapy response score (CRS) and clinical response to Olaparib and Niraparib. Methods: The study included a consecutive series of 94 HGSC patients who received PARPi in Alberta with institutional research board approval. CRS, BRCA1/2 and treatment information were collected and statistical analyses were performed using SPSS. Results: 46 HGSCs harbored BRCA1/2 mutations (42 receiving Olaparib in the primary adjuvant setting) and 48 HGSCs were BRCA1/2 wt (36 receiving Niraparib in the primary adjuvant setting). For Olaparib in the primary adjuvant setting, 4 of 6 (67%) with CRS 1 progressed while on treatment (median time to progression of 407 days) compared to 1 of 10 (10%) with CRS 2 and 0 of 7 (0%) with CRS 3 (combined median treatment period of 832 days except one who progressed after 98 day). For Niraparib in the primary adjuvant setting, 1 of 6 (17%) with CRS 1 progressed while on treatment (median treatment period of 722 days except one who progressed after 128 days), compared to 7 of 11 (64%) with CRS 2 and 7 of 10 (70%) with CRS 3 (combined median time to progression of 241 days). Conclusion: Higher CRS score appears to be predictive for Olaparib treatment response among BRCA1/2 mutated HGSC but not for Niraparib treatment response among BRCA1/2 wt HGSC.

LAY SUMMARY

- In the real-world setting (Alberta, Canada), Olaparib is primarily used in the primary adjuvant maintenance setting for BRCA1/2 mutated high-grade serous tubo-ovarian carcinomas whereas Niraparib is used both in the primary adjuvant maintenance setting and recurrent setting for BRCA1/2 wild-type high-grade serous tubo-ovarian carcinomas.
- Higher CRS score appears to be predictive for Olaparib treatment response among BRCA1/2 mutated high-grade serous tubo-ovarian carcinomas but does not appear to be predictive for Niraparib treatment response among BRCA1/2 wild-type high-grade serous tubo-ovarian carcinomas.
- An unexpected association between low CRS score (CRS 1) and apparent treatment response to Niraparib among BRCA1/2 wild-type high-grade serous tubo-ovarian carcinomas warrants further investigation.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Clinical and Metabolomic Characterization of Chemotherapy-Induced Fatigue in Metastatic Colorectal Cancer - Poster #598

Presenter: Jodi Rattner, University of Calgary

ABSTRACT_ID#3164

ABSTRACT

PURPOSE: Chemotherapy-induced fatigue (CIF) is a common toxicity in cancer treatment, affecting quality of life and potentially survival. Its mechanisms remain unclear. This study examined the relationship between CIF and survival, along with associated blood chemistry and metabolomic changes. **PATIENTS AND METHODS:** Data were analyzed from a randomized trial of cetuximab (N=374) vs. cetuximab+brivanib (N=376) in metastatic colorectal cancer (NCIC-CTG CO.20). Fatigue grades were assigned using standard criteria. Serial blood samples (baseline, weeks 1, 4, and 12) underwent metabolomic profiling via gas chromatography-mass spectrometry. O-PLS-DA was used to model CIF-related changes. **RESULTS** Fatigue correlated with reduced magnesium levels and elevated AST/ALT, without liver dysfunction. CIF did not impact treatment response or early cessation but was linked to shorter progression-free and overall survival. A metabolomic model for severe CIF (26 metabolites; $R^2Y = 0.908$, $Q^2Y = 0.74$, $CV\text{-ANOVA} = 7e-11$) suggested muscle breakdown. **CONCLUSION** CIF-related metabolic and electrolyte disturbances emerge early, before symptom onset. The strong association with hypomagnesemia warrants further study to determine if magnesium supplementation could mitigate fatigue and improve survival outcomes. Muscle catabolism may also contribute to CIF.

LAY SUMMARY

1. Fatigue has a significant association with shorter OS in patients receiving palliative treatment with TKIs for metastatic CRC.
2. There is a strong association between hypomagnesemia with CIF. Hypomagnesemia is easily managed, and may have benefit that can lessen the burden for patients undergoing strenuous therapy regimens.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Male pattern baldness and prostate cancer risk: results from the BIOmarkers of Prostate Cancer/Prevention and Environment (BIOCaPPE) prospective study - Poster #599

Presenter: Clément Larose, chu Québec - université Laval

Other Authors: Roxane Tourigny, Karine Robitaille, Fred Saad, Michel Carmel, Armen Aprikian, Yves Fradet, BIOCaPPE Network, Vincent Fradet

ABSTRACT_ID#2579

ABSTRACT

Background: The association between male pattern baldness and prostate cancer (PCa) remains controversial. This study aimed to investigate the relationship between baldness and the risk of PCa in the BIOmarkers of Prostate Cancer/Prevention and Environment (BIOCaPPE) study, a prospective multicentre observational cohort designed to identify biomarkers of PCa risk. **Methods:** Between 2013 and 2020, 1,470 men at high risk of PCa enrolled in the BIOCaPPE study were not receiving 5-alpha reductase inhibitors and then eligible for our analysis. Hair loss history was assessed using the modified Norwood-Hamilton scale at ages 20, 30, and 40, as well as at study baseline. Multivariate Cox logistic regression models were used to evaluate associations between PCa risk and various baldness characteristics, including pattern (frontal or vertex), age of onset, and severity. **Results:** Over a median follow-up of 6.4 years (Q1–Q3: 4.8–8.0), 237 cases of PCa were diagnosed. No significant association was found between baldness and overall PCa risk (HR 1.06; 95%CI 0.74–1.50; $p=0.768$). However, baldness was significantly associated with an increased risk of aggressive PCa (Grade Group ≥ 3) (HR 3.03; 95%CI 1.03–8.92; $p=0.043$). The association was stronger among individuals with frontal baldness (HR 3.46; 95%CI 1.11–10.76; $p=0.032$) and those who began balding at the age of 30 (HR 3.74; 95%CI 1.20–11.65; $p=0.023$). **Conclusion:** Baldness may be associated with an increased risk of aggressive PCa. These findings suggest that baldness could serve as a potential biomarker for identifying individuals at higher risk of developing aggressive forms of PCa.

LAY SUMMARY

Many men experience hair loss as they get older. Baldness is influenced by male hormones, which also play a role in the development of prostate cancer. Using a large pan-Canadian observational cohort of men at high risk of prostate cancer, baldness was not associated with an increased risk of developing prostate cancer. However, men with baldness were more likely to develop a more aggressive form of the disease. Baldness, particularly in men under the age of 30, may serve as an indicator of an increased risk of developing aggressive prostate cancer. If this association is validated in other studies, it could become a clinical risk factor.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Ontario Biobanks: Where research meets resources of oncology samples and data - Poster #602

Presenter: Katarina Maksimovic, Ontario Institute for Cancer Research

Other Authors: Riley Cox, Larry Phouthavongsy, Ilinca Lungu, Terry Hawrysh, Dianne Chadwick, Lincoln Stein

ABSTRACT_ID#2666

ABSTRACT

Statement of Problem. Access to high-quality oncology samples and data is essential for cancer research. Ontario is home to over 20 independent Canadian Tissue Repository Network-certified oncology biobanks, each with deep inventories of research-ready samples, data, and research services. Significant challenges include lack of visibility to the research community and the need for researcher to contract with multiple biobanks for rare tumours and large cohorts. **Proposed Solution.** To enhance research access to oncology samples and data, the Ontario Tumour Bank, supported by the Ontario Institute for Cancer Research, is coordinating the formation of a provincial network called Ontario Biobanks. The goal of the network is to connect researchers to sample and data collections from adult, pediatric, and companion animal participants with solid cancers and hematologic malignancies, as well as the Ontario Health Study and cancer clinical trial cohorts. Interest and engagement in Ontario Biobanks have been overwhelming. Annual meetings are well-attended and a charter outlining the network's structure, function, and goals has been jointly developed and was recently approved for establishing formal membership. Working groups have compiled biobank inventories of samples, data and services, and are creating the governance model. Next steps include development of the federated catalogue, unified research request process, access-fee guidelines and research access website. **Conclusions.** Ontario Biobanks is improving collaboration between biobanks. By enhancing visibility and streamlining access to oncology samples, data, and services, the network has the potential to accelerate research and cancer care in Ontario, across Canada and beyond.

LAY SUMMARY

Access to samples and data from cancer patients is essential for cancer research. The samples and patient data from biobanks are available to researchers to study cancer and find better treatments. However, researchers struggle finding samples and data needed because they are unaware of the many biobanks in Ontario, and the process of getting samples is slow and different for each biobank. This is especially a problem for researchers who need a lot of samples or samples from rare cancer types which may take years or decades for one institution alone to collect. The Ontario Tumour Bank is creating a network called Ontario Biobanks, which plans to connect biobanks and researchers. This will be done by creating a website with a list of member biobanks and the available samples and data. Ontario Biobanks will develop a consistent process of getting samples from member biobanks. This will make it easier and faster for researchers to get samples and data from multiple biobanks at a time. By making it easier for researchers to access biobanks' samples and data, Ontario Biobanks has the potential to fast-track cancer research, improving patient healthcare in Ontario and beyond.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Uncovering metastasis risk-associated genes: A Transcriptomic Study of the B2B Cohort - Poster #603

Presenter: Glenda Romero-Hernández, University of Calgary

Other Authors: David Yang, Nigel Brockton, Qingrun Zhang, Karen Kopciuk, Carrie Simone Shemanko

ABSTRACT_ID#2394

ABSTRACT

Breast cancer is one of the most common cancers worldwide and one of the leading causes of death among women. Unfortunately, most breast cancer-related deaths are due to its ability to spread to other parts of the body. The Breast Cancer to Bone (B2B) Metastases Research Program is a multidisciplinary program consisting of a prospective cohort of 478 breast cancer patients from Southern Alberta, with a follow-up for more than 10 years. In this study, we leveraged transcriptomic data obtained from the formalin-fixed, paraffin-embedded patient primary tumours with the goal of identifying a gene signature associated with metastatic progression. Using this dataset, we identified differences in gene expression between patients who developed metastasis and those who did not, reflecting diversity in expression profiles. Functional enrichment analysis of the differentially expressed genes revealed pathways related to metabolism and cancer progression, suggesting the specific molecular mechanisms driving metastasis. Survival analyses using Cox's proportional hazard model revealed genes associated with a higher risk of metastasis recurrence. We found two genes involved in cell migration, proliferation, and cell survival that resulted in being unfavorable biomarkers of cancer progression and recurrence. These findings were validated in the independent TCGA cohort. These genes showed hazard ratios greater than 1, supporting their prognostic potential. Together, these results offer a new insight into the risk prediction of developing metastasis and provide new potential targets for new therapeutic strategies. Therefore, it will be an important step toward more personalized breast cancer care.

LAY SUMMARY

We analyzed primary tumor samples from 478 breast cancer patients enrolled in a long-term study to identify new molecular markers associated with metastatic progression. The samples were collected at diagnosis and linked to clinical outcomes over more than 10 years of follow-up. We discovered a set of genes that were significantly expressed in patients who later developed metastasis. These genes are involved in key biological processes that may facilitate the spread of cancer to other parts of the body. Some genes were associated with a higher risk of metastasis recurrence. These findings suggest that elevated expression of these genes could serve as prognostic markers for recurrence risk. This knowledge may help inform treatment decisions and pave the way for more personalized and targeted therapeutic strategies in breast cancer care.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Building a cancer trajectory biobank: Research opportunities using Alberta's Tomorrow Project and the Alberta Cancer Research Biobank - Poster #604

Presenter: Laura Grant, Alberta's Tomorrow Project

Other Authors: Danielle Simonot, Ashley Brown, Adrian Driga, Shandra Harman, Mie Konno, Gillian MacNevin, Teena Makowichuk, Cara McGinley, Kerry Munro, Sara Nejatinamini, Will Rosner, Grace Shen-Tu, Bud Skiffington, Farit Vakehtov, Jennifer Vena

ABSTRACT_ID#2991

ABSTRACT

Background: Alberta's Tomorrow Project (ATP) is a prospective cohort study established in 2000 aimed at understanding causes of cancer. The Alberta Cancer Research Biobank (ACRB) is an open-access source of biosamples collected from people diagnosed with cancer and operating since 2002. Both groups are rich repositories with a common interest in supporting cancer research. **Methods:** Between 2000 and 2015, ATP recruited 55,000 Albertans with no previous cancer diagnosis, collecting blood, urine, and saliva alongside physical measurements and health and lifestyle data. Some ATP participants have subsequently developed cancer. Since 2002, ACRB has collected blood, urine, and tissue samples from patients at the time of cancer diagnosis plus some health and lifestyle data. In 2017, ATP and ACRB established joint infrastructure to identify participants in both repositories with available biosamples from before (in ATP) and at time of cancer diagnosis (ACRB). **Results:** As of 2025, 412 participants have data and biosamples available from both repositories. Serum, plasma, buffy coat, urine, DNA, normal tissue, and cancer tissue, as well as health and lifestyle data, are available for researcher request. Researchers can request access to these data and biosamples using an established access process. Linkage with additional administrative health data (including cancer registry) is also supported. **Conclusion:** This collaboration addresses a critical gap in cancer research by providing access to rare prospectively collected biosamples spanning before and at time of diagnosis from the same individuals. For more information, please email atp.research@ahs.ca and bio.bank@ahs.ca.

LAY SUMMARY

Alberta's Tomorrow Project has been following healthy Albertans since 2000, while the Alberta Cancer Research Biobank has been collecting samples from cancer patients since 2002. By combining forces, researchers can now access samples and data from both collections through a single request, making cancer research more efficient and comprehensive. Most cancer research can only study people either before they get cancer OR at the time they are diagnosed. Together, ATP and ACRB have blood and tissue samples collected from over 400 participants both before and after cancer diagnosis. This "before and after" view is extremely valuable for understanding how cancer develops.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

tRNA Fragments Extracted From Patient Plasma Extra Cellular Vesicles Display Potential As Biomarkers for High-Grade Glioma - Poster #605

Presenter: Jeremy Roy, Atlantic Cancer Research Institute

Other Authors: Thomson Phinney, Ali Alwadei, Jae Han, Kathleen Attwood, Mary MacNeil, Gabriel Wajnberg, Adrienne Weeks

ABSTRACT_ID#3158

ABSTRACT

Transfer RNA (tRNA) are non-coding RNAs that bring amino acids to ribosomes during translation. Recent studies have shown that cleaved fragments derived from tRNA (tRFs) serve regulatory roles in epigenetics, signaling, transcription and translation. tRFs can be released from cells and can be found in extracellular vesicles to perform some aspects of paracrine signaling. Utilizing high throughput small RNA sequencing, we have identified over 750 uniquely or multi-mapped (6.9% of total sRNA mapped reads) tRFs in extracellular vesicles of high-grade glioma (IDH-WT) patient plasma. By performing differential expression analysis we identified 6 upregulated tRFs and 36 downregulated tRFs between high-grade glioma plasma EVs and non cancer controls (n = 14, FDR<0.05). Differentially expressed tRFs (all 42) were further analyzed using bioinformatic techniques (RNAhybrid and IntaRNA) to predict potential gene targets. By performing gene ontology analysis (ShinyGO) we found that 70 of 75 genes related to glioma formation were identified as potential gene targets, indicating a possible role of tRFs in the regulation and formation of gliomas. A number of differentially expressed tRFs were predicted to target cancer-related genes. This analysis demonstrates that tRFs may serve as important biomarkers and regulators with therapeutic potential in the future.

LAY SUMMARY

Glioblastoma (GBM) is a devastating disease with no known cure. Identifying non-invasive biomarkers of disease would allow clinicians to better diagnosis GBM and predict prognosis. RNA molecules released from cancer cells that are detected in plasma are a promising novel avenue to help with GBM.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Synthetic Lethal Interaction Network of Chromosome 4p Loss in Basal Breast Cancer - Poster #606

Presenter: Lynn Karam, Concordia University

Other Authors: Alain Pacis, Rohan Dandage, Michael Schwartz, Anastasia Gherghi, Gabriel Alzial, Alysh Orr, Mayerly Castrillon, Belen Hernandez, Paria Asadi, Traver Hart, Genevieve Deblois, Andreas Bergdahl, Morag Park, Guillaume Bourque, Elena Kuzmin

ABSTRACT_ID#2427

ABSTRACT

Chromosome 4p (chr4p) loss is a recurrent large chromosomal deletion in basal breast cancer, the predominant molecular subtype of triple-negative breast cancer (TNBC), associated with the worst prognosis. To characterize chr4p synthetic lethal network in basal breast cancer and reveal mechanisms underlying chr4p loss buffering, basal breast cancer samples with copy-neutral and deletion status of chr4p were identified within our Primary Tumor / Patient-Derived Xenograft panel and Cancer Cell Line Encyclopedia data. Differential gene expression analysis revealed that chr4p loss is associated with global transcriptomic changes involving oxidative phosphorylation and reduction/oxidation (redox) balance pathways. Analysis of publicly available DepMap pooled genome-wide CRISPR-Cas9 screening data identified chr4p loss genetic interactions enriched for similar pathways. Functional validations of the genetic interactions resulted in reduced viability in chr4p loss samples. To follow up on the synthetic lethal interactions with oxidative metabolism, high-resolution respirometry revealed that chr4p loss cells are less respiratory and have high proton leak. Consistent with this observation, chr4p loss samples showed reduced mitochondrial respiratory complexes protein abundance and elevated reactive oxygen species levels. Seahorse Cell Mito Stress assay further revealed that chr4p loss was associated with reduced oxygen consumption and glycolytic rates suggesting metabolic quiescence as a selective advantage conferred by this aneuploidy. Mitochondrial morphology analysis revealed an increased mitochondrial network indicative of a compensatory mechanism. Drug sensitivity analysis identified that compounds disrupting redox balance selectively target chr4p loss cells. This study highlights large chromosomal deletions as potential targetable biomarkers in TNBC, offering novel therapeutic strategies for precision oncology.

LAY SUMMARY

Breast cancer is a heterogeneous disease classified into three major subtypes: the ER/PR-positive subtype, the HER2-amplified subtype, and the basal subtype, which is enriched for Triple Negative Breast Cancer (TNBC). TNBC lacks estrogen, progesterone, and HER2 receptors, and therefore lacks targeted therapies. However, it frequently displays large chromosomal deletions, including loss of the short arm of chromosome 4 (chr4p), which occurs in approximately 65% of TNBC cases. Chr4p loss is an early event in tumor evolution, associated with a proliferative advantage and poor clinical outcomes. We identified and validated the genetic vulnerabilities of TNBC by analyzing publicly available data. Specifically, we identified genetic interactions that result in synthetic lethality, meaning decreased viability in cells with chr4p loss while not affecting cells with chr4p copy neutrality. We identified a perturbed respiratory function associated with chr4p loss through a high-resolution respirometry assay and identified higher levels of reactive oxygen species levels further indicating perturbed mitochondria function. Furthermore, chr4p loss was associated with reduced oxygen consumption and glycolytic rates suggesting metabolic quiescence which has been linked to treatment resistance. This study provides insight into the functional role of large chromosomal deletions in cancer, thereby revealing potential therapeutic avenues for precision oncology.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Mutational Analysis of Primary and Advanced Chordoma Tissue using Next-Generation Sequencing - Poster #607

Presenter: Josh Chan, University of Calgary

Other Authors: Joseph Kendal, Zhenfeng Duan, Al Ferreira, Alireza Samiei, Scott Nelson, Arun Singh, Elizabeth Lord, Brooke Crawford, Nicholas Bernthal, Francis Hornicek

ABSTRACT_ID#2296

ABSTRACT

Background: Chordomas are rare ectodermal bone malignancies derived from transformed notochordal remnants. Histologic variants include conventional (80-90%), chondroid (5-15%) and de-differentiated (2-8%). Since chordomas are relatively chemotherapy and radiotherapy-resistant, novel targeted agents are needed to expand treatment approaches and improve outcomes. **Objective:** To analyze the genomic landscape of chordoma and identify potential pathogenic and druggable targets. **Methods:** Eighty-six tumor samples derived from chordoma patients treated at Massachusetts General Hospital, UCLA and the University of Miami were included. Tumor specimens were sent for comprehensive molecular profiling using next-generation sequencing. The most frequently mutated genes were identified and categorized by subtype, and microsatellite instability and PD-L1 staining were assessed. **Results:** Histologic subtypes included 70 conventional chordomas (81.4%), 9 chondroid chordomas (10.5%) and 7 de-differentiated chordomas (8.1%). The most common mutations were CDKN2A/B (28/86, 33%), LRP1B (10/86, 12%), PBRM1 (9/86, 11%) and EGFR (8/86, 9%). By subtype, CDKN2A/B mutation was most common in conventional chordoma (24/70, 34%) and chondroid chordoma (3/9, 33%). CDKN2A/B and EGFR mutations were most common in de-differentiated chordoma (2/7, 29%). Microsatellite instability was not detected in 7/69 (10.1%) samples. PD-L1 staining of tumor and immune cells was scarce, with scores <1 in 38/41 (92.7%) and 22/25 (88%) patients, respectively. **Conclusions:** This study provides a robust, high-dimensional sequencing assessment from 86 chordoma tissue samples and a descriptive overview of the genomic landscape of this rare, difficult to treat malignancy. Future studies should include in vitro assessment of gain and loss of function of frequently altered pathways to validate these findings.

LAY SUMMARY

This study provides a comprehensive genomic analysis of 86 chordoma samples, identifying CDKN2A/B, LRP1B, PBRM1 and EGFR as the most frequently mutated genes. Microsatellite instability and PD-L1 staining of tumor and immune cells were scarce.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Is PRAME a good prognostic indicator for detecting the development of metastasis in uveal melanoma patients - Poster #608

Presenter: Manjot Jhinjar, University of Calgary

Other Authors: Ezekiel Weis, Trafford Crump

ABSTRACT_ID#2856

ABSTRACT

Background Previous research studies have suggested that PReferentially expressed Antigen in Melanoma (PRAME) expression has been associated with increased metastatic development in uveal melanoma patients. The purpose of this study is to correlate PRAME expression with metastatic status in uveal melanoma patients undergoing plaque brachytherapy. **Methods** This retrospective study analyzed data from the Alberta Ocular Brachytherapy Program. This program maintains registry of all patients it diagnoses with uveal melanoma for their lifetime. All patients diagnosed from 2017 to 2022 were included in this study. Patients were excluded if they were not residents of Alberta (or moved out of province during our follow-up window), if they had died, or were otherwise lost to follow up. Metastatic disease was tracked through Netcare, Alberta's provincial health record, until March 2025. Tumor samples obtained via fine needle aspirate biopsy (FNAB) were analyzed for PRAME expression. A 2×2 table and Chi-Square test was used to test for an association between PRAME and the development of metastasis. **Results** There were 360 patients included in this study. Of those, 21 (5.8%) were diagnosed with metastatic disease. Among the metastatic cases, 18 (86%) presented PRAME- expression and 3 (14%) presented PRAME+ expression. For the non-metastatic, 274 (81%) had PRAME- expression and 65 (19%) had PRAME+ expression. The Chi-Square value of 0.31 was not statistically significant ($p=0.578$). **Conclusion** The results from this study suggest there is no significant association between PRAME expression and metastatic status in uveal melanoma, countering existing research on PRAME and its association with metastasis.

LAY SUMMARY

Our study focuses on correlating the association between PRAME and the development of metastasis in uveal melanoma patients. Statistical analysis of clinical data included in this study indicate that there are no significant associations between PRAME expression and metastatic status in uveal melanoma, drawing into question whether PRAME is a viable biomarker in prognosing metastatic risk in uveal melanoma patients. These findings provide a counter-narrative to existing research and thus suggest that further research is warranted regarding PRAME being a viable prognostic biomarker in predicting metastatic risk in uveal melanoma patients. Prognostic markers are important and useful tools in assisting clinicians to plan and guide treatment strategies for their patients. Prognostic markers enable clinicians to offer personalized treatments that are tailored to unique clinical profiles and based on the respective needs of each their individual patients.

Monday, November 3, 2025

Preclinical evaluation demonstrates therapeutic potential for MEK inhibition in NF1-deficient high-grade serous tubo-ovarian carcinomas - Poster #610

Presenter: Isabelle Nechala, University of Alberta

Other Authors: Rui Zhe (Rachel) Yang, James Key, Pooja Praveen Kumar, Lauren Chen, Guihua Zhang, Yangxin Fu, Cheng-Han Lee

ABSTRACT_ID#2473

ABSTRACT

Introduction: High-grade serous carcinoma (HGSC) is the most common and lethal form of tubo-ovarian cancer. Molecularly, it can be classified as homologous recombination proficient (HRP) or deficient (HRD). HRD tumors often harbor BRCA1/2 mutations and are more sensitive for current stand-of-care clinical therapies. In contrast, HRP tumors lack effective systemic treatment. A subset is characterized by NF1-inactivation, which is expected to enhance RAS-MEK-ERK pathway signaling. We hypothesize that targeting this altered signaling may benefit patients with NF1-deficient HGSC, particularly in the HRP setting. **Methods:** CRISPR-engineered and naturally occurring NF1-deficient and NF1-intact HGSC cell lines were treated with increasing doses of the MEK inhibitors trametinib or avutometinib. Western blots assessed RAS-MEK-ERK pathway activity. In vivo studies used immunocompromised mice implanted with NF1-deficient or proficient HRP HGSC (OVCAR3) xenografts and treated with trametinib (0.5 mg/kg, I.P. daily). **Results:** NF1-deficient HGSC cells exhibited increased sensitivity to both MEK inhibitors, with significantly lower IC50 values compared to NF1-intact cells. These cells showed reduced levels of phospho-ERK1/2 following treatment with trametinib or avutometinib. In vivo, trametinib significantly reduced tumor weight in NF1-deficient xenografts (CRISPR NF1 knock-out OVCAR3) compared to vehicle control ($p=0.0057$). In contrast, the same treatment had no significant effect on tumour weight in NF1-proficient OVCAR3 xenografts compared to vehicle control ($p=0.0732$). **Conclusion:** NF1-deficient HRP HGSCs display enhanced sensitivity to MEK inhibition. These findings support MEK inhibition as a promising therapeutic strategy for NF1-deficient HGSC subtype. Studies evaluating the in vivo efficacy of trametinib and avutometinib in naturally occurring NF1-deficient models are ongoing.

LAY SUMMARY

1. High-grade serous ovarian carcinomas may respond better to targeted treatment: High-grade serous ovarian carcinomas (HGSC) with NF1 gene inactivation shows overactive signaling in a pathway called RAS-MEK-ERK, important for regulating cell growth, making these tumors potentially more vulnerable to inhibitors of the pathway.
2. MEK inhibitors work better on tumors that are NF1-deficient: HGSC cell lines lacking NF1 were more sensitive to MEK inhibitors (trametinib and avutometinib) compared to cells with normally expressed NF1, meaning these drugs are more effective at reducing the growth of NF1-deficient cell lines.
3. In vivo studies support the in vitro findings: In vivo experiment with NF1-deficient tumors, daily treatment with trametinib significantly slowed tumor growth, while it had minimal effect on tumors with normal NF1 expression, suggesting MEK inhibitors may be a potential treatment for NF1-deficient HGSC.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Marathon of Hope Quebec: Building a Regional Framework for a Pan-Canadian Initiative - Poster #611

Presenter: Luisa Izzi, McGill University

Other Authors: Marathon of Hope Quebec Consortium

ABSTRACT_ID#3174

ABSTRACT

The Marathon of Hope Cancer Centres Network (MOHCCN) is a federally funded initiative aiming to accelerate precision oncology by integrating genomic, clinical and outcome data from 15,000 Gold Cohort Cases across Canada. The Marathon of Hope Quebec (MoHQ) brings together clinician-scientists, researchers, bioinformaticians, and data scientists from hospitals, cancer research institutes and sequencing centres across Québec to contribute to this effort. The initiative focuses on the whole genome and transcriptome sequencing of retrospective and prospective cases across multiple cohorts and tumour types, addressing four priorities, including treatment resistance to immunotherapy and precision medicine, tumour heterogeneity, clinical validity and utility of genomics in real-time decision-making and the biology of rare cancer subtypes. Participating sites contribute biospecimens from certified biobanks and standardized clinical data. Sequencing is performed at the McGill Genome Centre and other partner sequencing platforms in Quebec. Clinical data are extracted from the Système d'archivage des données en oncologie (SARDO) and local data repositories, then integrated into local instances of Annotated Tissue Management (ATiM) at each hospital. Clinical data are mapped to the MOHCCN clinical data model within ATiM, and once linked to genomics, are deposited in the CanDIG platform for secure data sharing. To date, the MoHQ has sequenced samples from over 2300 patients from 7 institutions. We have established pipelines for genomic analysis and clinical data ingestion, enabling collaborative research across our institutions and the MOHCCN. The infrastructure developed provides a framework for future initiatives in oncology, demonstrating the power of regional coordination in building a national network.

LAY SUMMARY

The Marathon of Hope Cancer Centres Network (MOHCCN) is a Canada-wide initiative funded by the federal government with the goal of profiling and analyzing 15,000 cancer patients across the country. The Marathon of Hope Quebec (MoHQ) brings together doctors, scientists and data experts from hospitals, cancer research institutes and sequencing centres from across Quebec to profile the genetic code of thousands of tumours to understand why certain tumours resist to therapy, how they change over time and how to better treat rare types of cancer. Thus far, the MoHQ has profiled tumours from over 2300 patients from 7 hospitals across Quebec and built the infrastructure to securely collect and share the genetic and clinical data with researchers across the MOHCCN.

Monday, November 3, 2025

Circulating tumour DNA (ctDNA) based detection of genetic immune escape in advanced prostate cancer (aPCa) - Poster #612

Presenter: Nicolette M. Fonseca, University of Victoria

Other Authors: Asli D. Munzur, Jack V.W. Bacon, Gillian Vandekerkhove, Grainne Donnellan, Cecily Q. Bernales, Jenny N. Tran, Franz Fenninger, Karen R. Sherwood, Alexander W. Wyatt, Nicolette M. Fonseca

ABSTRACT_ID#2445

ABSTRACT

T cell recognition of neoantigens presented by the Major Histocompatibility Complex Class I (MHC Class I) molecules is essential for anti-tumour immunity. Loss of MHC Class I expression therefore represents a mechanism of immune evasion in aPCa, with implications for the efficacy of immunotherapies. However, the genomic events contributing to MHC Class I loss, particularly loss of heterozygosity (LOH) of the polymorphic HLA-A, HLA-B and HLA-C genes, remain poorly defined. Furthermore, ctDNA has not been investigated as an analyte for tracking tumour-specific HLA alterations, as current approaches rely on whole genome or exome sequencing of tissue. We developed a targeted DNA sequencing panel (ImmGen) to capture 6597 unique HLA-A, HLA-B and HLA-C alleles. Probe design was optimised using machine learning applied to the IMGT-HLA database (v3.10). HLA genotyping and HLA-LOH detection was performed using published tools and validated in select cases with clinical grade HLA genotyping (Holotype HLA, v2 Omixon). We assembled a cohort of 40 aPC patients with cell-free DNA (n=28), tumour tissue (n=17) and matched germline DNA (n=40). ImmGen achieved 90% HLA allele concordance (216/240) across 3 HLA genotyping tools, versus 79% (90/114) via exome sequencing (where available). In 19 patients with available Holotype HLA results, ImmGen HLA types were 100% concordant. Germline HLA homozygosity was detected in 39% of patients (16/40). HLA-LOH was detected in ctDNA (>30% purity) and tissue from 33% (8/24) and 35% (6/17) of patients respectively. Our results demonstrate that HLA-LOH can be detected non-invasively and in real-time using cost-effective targeted sequencing of ctDNA.

LAY SUMMARY

T cells are part of the immune system and help fight cancer by recognizing abnormal proteins (called neoantigens) presented on the surface of cancer cells via an exquisitely specific identification system called MHC Class I. However, certain prostate cancers, especially when metastatic, escape detection by losing MHC Class I, effectively hiding from immune detection. We do not fully understand the genetic changes that cause this loss, specifically those involving the key genes encoding MHC Class I (HLA-A, HLA-B and HLA-C). Current methods to track these changes require invasively collected tumour tissue and a costly type of DNA sequencing. Our research group developed a new blood test called ImmGen that can detect changes in the HLA genes using a simple blood draw. This test used machine learning to optimize the capture of thousands natural HLA gene variants. In a study of 40 men with advanced prostate cancer, the test showed high accuracy to identify HLA genes compared with standard methods, and could detect important genetic changes in blood at a similar rate compared to tumour tissue. This new test offers a less invasive, real-time way to track how prostate cancer may be evading the immune system.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Evaluating the prognostic utility of circulating tumour DNA in metastatic urothelial cancer - Poster #615

Presenter: Maria Stephenson, Vancouver Prostate Centre

Other Authors: John Pham, Andrew Murtha, Cecily Bernales, Grainne Donnellan, Karan Parekh, Jack Bacon, Matti Annala, Sarah Ng, David Müller, Bernhard Eigl, Guliz Ozgun, Peter Black, Corinne Maurice-Dror, Kim Chi, Gillian Vandekerckhove, Alexander Wyatt

ABSTRACT_ID#2809

ABSTRACT

Metastatic urothelial cancer (mUC) is an aggressive disease with diverse clinical outcomes. Recent studies have suggested that the fraction of circulating DNA in the blood plasma that is tumour-derived (i.e. ctDNA%) carries prognostic information in mUC. However, there is ambiguity as to the relation between precise ctDNA levels and patient prognosis, and the relationship between ctDNA% and clinical disease characteristics is under-characterized. We evaluated the prognostic relevance of pretreatment ctDNA%, and the relationship between ctDNA% and baseline patient clinical characteristics. For this interim analysis (accrual is ongoing), our dataset consists of 119 mUC patients with pretreatment blood taken within 90 days of metastatic diagnosis. Samples underwent deep-targeted DNA sequencing and ctDNA% was determined via somatic mutations and copy number alterations. ctDNA% was significantly higher in patients with liver metastases (20.9% vs 3.44%, $p=0.00047$). Higher ctDNA% may associate with worse ECOG performance status (2+ vs 0-1; 18.0% vs 3.92%, $p=0.098$) and lymph node metastases (12.5% vs 3.15%; $p=0.082$). ctDNA% was not correlated with age at metastatic diagnosis ($R=-0.17$, $p=0.062$), sex ($p=0.38$), nor other metastatic sites (lung $p=0.56$, bone $p=0.24$). Higher ctDNA% was associated with shorter overall survival (OS). Patients with no detected ctDNA had the highest median OS (20.1 months), in contrast to patients with detected ctDNA below the median (<8%, 14.9 months), within the 3rd quartile (8-25%, 10.9 months), and above the third quartile (11.4 months). These results support ctDNA% as a robust, independent prognostic factor in mUC.

LAY SUMMARY

In metastatic urothelial cancer, the fraction of circulating DNA in the blood that is tumour-derived is a prognostic biomarker. Detection of circulating tumour DNA is associated with worse overall survival. Elevated circulating tumour DNA abundance is associated with presence of liver metastases, and independent of other clinical characteristics such as age and sex.

Precision Medicine and Biomarkers

Monday, November 3, 2025

The identification of HER2+ breast cancer recurrent signatures from primary tumours - Poster #616

Presenter: Paola A. Marignani, Dalhousie University

Other Authors: Jinhong Kim

ABSTRACT_ID#2931

ABSTRACT

Precision medicine aims to provide more effective treatments, detect disease earlier and improve patient outcomes. HER2 positive (HER2+) breast cancer accounts for 20-30% of all breast cancer cases and is highly aggressive. The mortality rates for HER2+ are higher than other breast cancer subtypes. Our question is whether HER2+ breast cancers express transcriptomic signatures that are distinguishable between non-recurrence and recurrence. Methods: We analyzed tumours from 32 patients identified with HER2+ breast cancer that were treated with Herceptin adjuvant therapy. From these patients, 16 did not experience recurrence, while 16 did experience recurrence within 5 years of Herceptin treatment. Tumours were prepared for scRNAseq analysis, followed by computational analysis of data. Results: From the 192,000 single nuclei analyzed, a total of 27,303 genes were identified and annotated. We found 62.8% and 66.8% DEGs were specific to recurred and non-recurred data, respectively. We applied GSEA for transcriptomic characterization using multiple reference databases such as transcription factor (TF) collection, biological pathways, hallmark gene sets and gene ontology (GO) terms. We identified 203 unique and 379 unique TFs were enriched in recurred and non-recurred tumours, respectively. The number of enriched biological pathways revealed that 50 of 62 pathways were activated in non-recurred tumour cells compared with recurred tumour cells where 38 of 62 pathways were activated. Summary: The outcome of our study reveals significant molecular distinction between recurrent and non-recurrent HER2+ breast cancers, suggesting potential biomarkers for predicting recurrence. PM is supported by Breast Cancer Canada.

LAY SUMMARY

20-30% of HER2+ breast cancer patients experience recurrence despite Herceptin treatment. The mortality rates for HER2+ are higher than other breast cancer subtypes. We identified a gene signature that is unique for patients that experience HER2+ breast cancer recurrence. The gene signatures we discovered predict which women with HER2+ breast cancer will likely experience recurrence.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

InterpretKB: An Integrated Knowledgebase Platform for Interpretation of Oncogenomic Data to Determine Actionability and Pathogenicity of Sequence Variants - Poster #617

Presenter: Nazanin Vaziri, University of Calgary

Other Authors: Rehan Faridi, Faisal Khan

ABSTRACT_ID#3097

ABSTRACT

With the increasing integration of cancer genomic sequencing into clinical and research workflows, there is a growing demand for efficient, accurate, and cost-effective methods for interpreting sequence variants. Existing public and commercial knowledgebases—such as CIViC, OncoKB, and JAX-CKB—support variant classification but require manual curation and are associated with high per-use costs. To address these challenges, we introduce InterpretKB, a Microsoft SQL-based knowledgebase developed using over 5,000 tumor samples manually curated by the Hematology Translational Lab since its clinical accreditation in 2017. InterpretKB will serve as a centralized platform for rapid querying and classification of oncogenic variants based on pre-existing definitions and curated data. Accompanying this knowledgebase is Interpretor, a custom decision-support application integrating APIs from key genomic resources such as ClinVar, COSMIC, and gnomAD. Together, these tools will support the automated and curated interpretation of whole-genome and transcriptome datasets. Our goals include reducing per-analysis costs by 50–60%, ensuring consistency in clinical reporting, and minimizing redundancy across bioinformatics tools. The system will offer batch annotation capabilities, enabling high-throughput analysis of variants with insights into mutation function, gene/protein localization, and prevalence across tumor types. Additionally, Interpretor will interface with public resources such as OncoKB, PMKB, and PharmGKB for enriched annotation. InterpretKB represents a scalable, cost-efficient solution for oncogenomic interpretation, poised to accelerate reporting workflows and enhance clinical decision-making in precision oncology. The platform will ultimately contribute to improved patient outcomes by streamlining variant annotation and supporting evidence-based therapeutic targeting.

LAY SUMMARY

Streamlined and Cost-Effective Variant Analysis: InterpretKB platform is designed to speed up and simplify the interpretation of cancer-related genetic mutations. This system is expected to cut analysis costs by 50–60% and reduce repetitive work.

Reliable and Scalable Clinical Tool: Built on data from over 5,000 tumor samples, InterpretKB ensures accurate classification of genetic variants, helping physicians make better-informed decisions in cancer treatment.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Identification of Novel Single-Nucleotide Variants Specific to Lung Adenocarcinoma - Poster #618

Presenter: Paola A. Marignani, Dalhousie University

Other Authors: Jinhong Kim

ABSTRACT_ID#2938

ABSTRACT

In Canada, 32,100 Canadians were diagnosed with lung cancer in 2024, and 20,600 died from the disease. The five year survival rate for late-stage lung cancer is 22%, compared with 55% for early-stage diagnosis. Notwithstanding the difference in the etiology of lung cancer in never-smokers and smokers, lung cancer is the leading cause of cancer-related deaths amongst Canadians. Objective: The objective of our research is to discover tumour-specific single-nucleotide variants (SNVs) in lung cancer patients by applying full-length (FL) scRNA-seq analyses can provide valuable insights into the genomic landscape and heterogeneity of lung adenocarcinomas (LUADs) at the single-cell level in both sexes with different smoking status. Methods and Results: We identified new and unique cell type-specific eSNVs through genotyping eSNVs in individual cells/cell population in early-and late-stage LUADs. Applying Machine Learning (ML) algorithms, we measured transcriptomic intratumour heterogeneity to indicate the extent of entropy-based distance for the ambiguity of protein-protein interaction networks and found an approximate 28X higher degree of network-based nJSD distance from patient-paired normal lung cells to late-stage LUADs compared with early-stage LUADs. Summary: We identified new tumour-specific SNVs unique amongst the sexes. These newly identified SNVs will form the foundation of biomarker panels that will be used for early detection of lung cancers. Our long-term goal is to change the lung cancer survivorship narrative. PM is supported by Canadian Cancer Society and Cancer Research Society

LAY SUMMARY

20,600 Canadians died from lung cancer in 2024 The five year survival rate is 22% We identified novel and unique gene variations between women and men, for early- and late- stage lung cancers

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Examining the effects of the inhibitor of mRNA translation zotatifin in models of uveal melanoma - Poster #619

Presenter: Noah Wirasinghe, Lady Davis Institute

Other Authors: Raul Flores Gonzalez, Wilson H Miller Jr, Sonia V del Rincon

ABSTRACT_ID#2361

ABSTRACT

Uveal melanoma (UM) is a high fatality cancer arising in the uveal tract of the eye. While local tumour control in patients diagnosed with UM has been achieved, more than half of UM patients will develop incurable metastasis (primarily to the liver). Due to the unique molecular characteristics of UM that render BRAF- and MEK-targeted therapies as well as immunotherapies ineffective, the inhibition of eIF4F-mediated mRNA translation serves as a promising treatment for metastatic UM. We take advantage of next generation inhibitors, namely the eIF4A inhibitor zotatifin, to test as novel treatments in metastatic UM. We have shown that zotatifin decreases proliferation, clonogenic outgrowth, and invasion in vitro. At the molecular level, we have verified that zotatifin represses FGFR1, a known target in triple-negative breast cancer. We have also identified SQLE as a novel target of zotatifin in UM. SQLE functions in cholesterol biosynthesis and its expression is associated with cancer progression in other tumour types. Additionally, clinical analysis of the TCGA UVM dataset reveals that patients with high SQLE expression have significantly poorer overall survival, highlighting SQLE as a potential prognostic biomarker and therapeutic target. We will use UM cells engineered to lack SQLE to further interrogate the role of cholesterol biosynthesis in UM. We have encouraging in vivo mouse model data showing that zotatifin blocks UM outgrowth and future studies will test the ability of the drug to decrease liver metastasis. Overall, our results suggest that eIF4A inhibition may serve as an effective treatment against metastatic UM.

LAY SUMMARY

Uveal melanoma (UM) is a high fatality cancer because it is highly metastatic and historically unresponsive to available therapies. Zotatifin is a first-in-class drug inhibitor that selectively reduces the synthesis of proteins supporting the spread of cancer (ie. metastasis) in the body. We have shown that zotatifin blocks UM metastatic potential using patient-derived UM cell lines, suggesting that zotatifin may serve as an effective treatment against metastatic UM. We have used state of the art technologies to understand at the cellular level how zotatifin elicits its anti-cancer effects.

Precision Medicine and Biomarkers

Monday, November 3, 2025

miRNA Biomarkers Predicting Response to Gemcitabine and Docetaxel in Leiomyosarcoma - Poster #620

Presenter: Pegah Rahimizadeh, McGill University

Other Authors: Philippe Jolivet, Paul Huang, Matt Van de Rijn, Joanna Przybyl

ABSTRACT_ID#2627

ABSTRACT

Introduction: Gemcitabine and docetaxel (Gem/Tax) is a common chemotherapy for leiomyosarcoma (LMS), but only 25–36% of patients respond. The biological basis for this variability is unknown, and no predictive biomarkers are available. Identifying such markers could guide personalized treatment and avoid unnecessary drug exposure. Here, we expanded our prior study that identified transcriptomic and epigenomic biomarkers predictive of Gem/Tax response by performing miRNA sequencing. **Methods:** miRNA expression was profiled in 35 archival, untreated primary LMS tumors from patients later treated with Gem/Tax. Sixteen patients developed recurrence after treatment (non-responders), while 19 did not (responders). Differential miRNA analysis was performed using SAMseq. Recurrence-free survival (RFS) was assessed with Kaplan–Meier analysis and log-rank test. Correlation between miRNAs and their target genes (from prior RNA-seq data) was examined using Spearman correlation. A multivariate Cox regression model was used to create a predictive miRNA signature. **Results:** Three miRNAs, hsa-miR-196a-5p, hsa-miR-625-3p, and hsa-miR-7977, were significantly upregulated in non-responders. Lower expression of these miRNAs was associated with better RFS. Significant correlations were found between these miRNAs and their target genes: hsa-miR-7977 was positively correlated with IL6R, POU2AF1, KCNE4 and negatively with PHF1, SENP5; hsa-miR-196a-5p was positively correlated with HOXB7; hsa-miR-625-3p was negatively correlated with MAP3K1. A prognostic risk score was calculated as: $\text{Risk score} = (0.0000314 \times \text{miR-196a-5p}) + (0.008816 \times \text{miR-625-3p}) + (0.000273 \times \text{miR-7977})$. This model accurately stratified patients into high- and low-risk groups with distinct survival outcomes. **Conclusion:** This is the first study to identify miRNAs as candidate biomarkers for Gem/Tax response in LMS. These miRNAs may contribute to chemoresistance, warranting further investigation.

LAY SUMMARY

Only a subset of LMS patients respond to chemotherapy: Leiomyosarcoma (LMS) patients treated with gemcitabine and docetaxel (Gem/Tax) have variable outcomes, and we currently cannot predict who will benefit from this treatment. Three microRNAs may predict treatment response: In our study, three specific microRNAs (miR-196a-5p, miR-625-3p, and miR-7977) were found at higher levels in patients whose tumors came back after treatment, suggesting these may be linked to poor response. A risk score may help guide treatment decisions: Using the expression levels of these microRNAs, we created a score that classified patients into high- or low-risk groups, which matched their chance of recurrence. In the future, this signature could help personalize treatment plans and avoid ineffective chemotherapy in patients who are unlikely to respond to this therapy.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Advancing Clinical Delivery of Personalized Care with Tailored Educational Materials (ADAPT-Care) - Poster #621

Presenter: Patil Mksyartinian, Colorectal Cancer Canada

Other Authors: Navdeep Dehar, Winson Cheung, Kaiden Jobin, Yogita Rao, Barry Stein, Ozioma Uzodinma, Vincent Raske, Nimira Alimohamed

ABSTRACT_ID#2486

ABSTRACT

Patients and healthcare professionals (HCPs) face significant challenges in understanding and communicating precision medicine (PM) concepts, including biomarker testing and targeted therapies. Prior research has highlighted the need to increase patient awareness of biomarker testing, its benefits, and its role in treatment decision-making. This pilot study addresses these barriers by examining the use of PM-tailored educational resources designed to support shared decision-making and improve communication between HCPs and patients. The study is underway at the Arthur Child Comprehensive Cancer Centre and University of Calgary. Oncologists will recruit eligible patients diagnosed with colorectal, pancreatic, cholangiocarcinoma, prostate, or bladder cancer at various stages. Patients who have undergone biomarker testing and are using it to inform treatment decisions will receive printed and digital educational materials explaining biomarker testing, targeted therapies, and common biomarkers. These materials were co-created by a multi-stakeholder group co-led by the patient community through FT3 (global coalition) and adapted to the Canadian context. Patients will complete a digital questionnaire assessing demographics, perceived knowledge, empowerment, communication, and capacity for shared decision-making. Treating physicians will also review the materials and complete a separate survey capturing their practices and perspectives on biomarker testing and impact of the resources. The study aims to include 200 patients and their physicians. Data will be analyzed using descriptive and bivariate statistics, with multivariable logistic regression applied where relevant. Early results show overwhelmingly positive responses. The findings will help demonstrate the potential of PM-tailored resources to enhance communication, empower patients, and improve shared decision-making in cancer care.

LAY SUMMARY

1. **Provider-Patient Discussions:** Precision medicine (PM) and target therapies can be difficult to understand. Prior research has highlighted the need to increase patient awareness of biomarker testing, its benefits, and its role in treatment decision-making. Both patients and healthcare providers often struggle to talk about complex topics like biomarker testing and targeted therapies, which are important for personalizing cancer treatment.
2. **Educational Materials:** Clear educational tools can make a difference. This study is testing printed and digital materials that explain biomarker testing and targeted therapies in a simple, accessible way. These resources were co-created with an expert multi-stakeholder group and adapted for use in Canada. These materials are being evaluated through patient and physician surveys to assess their impact on knowledge, communication, and shared decision-making.
3. **Shared-Decision Making:** Early results from the study are promising. To date, patients and physicians report that the PM-tailored educational materials improve communication, help patients feel more informed, and support shared decision-making about treatment. The study is still recruiting participants, and future analyses will explore how patient demographics relate to outcomes, and how the materials compare to current communication practices.

Precision Medicine and Biomarkers

Monday, November 3, 2025

Developing mouse lung cancer models of dysregulated Cyclin E/CDK2 axis to test treatments combining CDK2 inhibitors and immune checkpoint blockade - Poster #622

Presenter: Pierre-Olivier Gaudreau, Queen's University

Other Authors: Rachel Bishop, Cole Sandler, Eun Young Bae, Andrew Craig

ABSTRACT_ID#2918

ABSTRACT

Lung cancer response rates with immune checkpoint blockade (ICB) remains low, and causes of ICB resistance are poorly understood. Elevated Cyclin E/CDK2 signaling axis occurs in a subset of patients with lung cancer (CCNE1-amplified or FBXW7-deficient), and this has been linked to increased replication stress, ICB resistance and worse patient outcomes. Treatments with CDK2 inhibitors (CDK2i) have shown recent promise in eliminating cancer cells, but these treatments can also promote expression of the PD-L1 immune checkpoint. Here, we developed mouse syngeneic lung cancer models with inducible Cyclin E overexpression, or Fbxw7 knock-out (KO). The latter model will prevent targeting of Cyclin E, and several other proto-oncoproteins, for degradation via the ubiquitin-proteasome system. Compared to isogenic control cells, Cyclin E overexpression or Fbxw7 KO cells showed increased proliferation rates, and increased sensitivity to treatments with INX-315, a selective CDK2i. However, CDK2i treatments upregulated levels of PD-L1 on the surface of the lung cancer cells. These findings suggest potential synergy between CDK2i and PD-L1 ICB when tested in combination. Ongoing syngeneic lung cancer engraftment studies in immune competent mice are testing whether CDK2i and PD-L1 ICB are synergistic in vivo, and how these treatments alter the tumour immune microenvironment in these novel lung cancer models.

LAY SUMMARY

- Mouse lung cancer models engineered with inducible Cyclin E overexpression or Fbxw7 knock-out (KO) showed increased proliferation rates compared to control cells.
- Although Cyclin E overexpression and Fbxw7 KO cells were sensitive to treatments with a selective CDK2 inhibitor (INX-315), these treatments led to PD-L1 upregulation on these lung cancer cells.
- These findings suggest potential synergy between CDK2i and PD-L1 immune checkpoint blockade when tested in combination.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Exploring Cancer Patients' and Caregivers' Perspectives and Knowledge Regarding Biomarker Testing in Canada - Poster #623

Presenter: Patil Mksyartinian, Colorectal Cancer Canada

Other Authors: Selina Xu, Chrissa Barroma, Sandra Peláez, Barry Stein

ABSTRACT_ID#2872

ABSTRACT

While biomarker testing can provide various benefits for cancer patient outcomes, numerous challenges persist that cause inequities in access across Canada. An online survey consisting of 51 questions was disseminated to evaluate biomarker testing and precision medicine knowledge and experiences from Canadian patients and caregivers. Responses were recorded between June 2023 and January 2024 and assessed various aspects of the biomarker testing experience including the expectations and challenges of patients. Quantitative and qualitative analyses were conducted using Microsoft Excel and R for descriptive and correlative data analysis, respectively. Among the 74 responses, patients reported an overall moderate experience with positive outcomes for those who underwent biomarker testing, including changes to treatment plans and the shrinking of tumours. The main challenges identified included knowledge gaps, a lack of testing availability, turnaround time for results, and financial constraints, all of which contribute to the disparities in biomarker testing access. Qualitative analysis of responses further emphasized a strong patient desire for patient-centred care and collaborative decision-making for biomarker testing options and treatment planning. Addressing these challenges through increased education, policy advocacy, and advancing infrastructure can help to reduce interprovincial inequities in biomarker testing and contribute to improving cancer patient outcomes.

LAY SUMMARY

Not everyone has equal access to biomarker testing in Canada. Many patients face barriers to access to biomarker testing such as limited availability of biomarker testing, long wait times for test results, and financial challenges (e.g., paying out-of-pocket for testing), which can affect their ability to get the right cancer treatment at the right time. Patients value personalized and informed care. Those who received biomarker testing often experienced benefits, such as more tailored treatments and improved outcomes (e.g., shrink/control their cancer). Still, many patients expressed a need for better communication from their healthcare providers and involvement in their decision-making. There are gaps in biomarker testing awareness and access. The survey revealed that both patients and caregivers often don't know enough about biomarker testing and personalized, especially that biomarker testing can help determine the best cancer treatment for them. Improving education, strengthening policies, and investing in better infrastructure for biomarker testing could help reduce these gaps and improve care for all Canadians.

Precision Medicine and Biomarkers

Monday, November 3, 2025

The Achilles heel of metabolism that links normal breast development and breast cancer progression - Poster #624

Presenter: Carrie S. Shemanko, The University of Calgary

ABSTRACT_ID#2398

ABSTRACT

Cellular metabolism impacts multiple key cellular processes and as well as malignant transformation and the tumour microenvironment. We have found a signaling network within these hubs that is essential for cell fate processes in normal breast development and also in breast cancer. We discovered that a key molecular regulator, and the cholesterol biosynthesis pathway is essential for cell fate choice in breast development, and the production of milk producing alveoli from stem/progenitor cells. Multiple therapeutics target the cholesterol biosynthesis pathway that is also active in breast cancer cells and bone osteoclasts. We demonstrated the efficacy of one such drug in a preclinical animal model of bone metastasis, showing a reduction in osteolytic bone damage with a combination of preventative and therapeutic treatments. We identified a hormone receptor that drives sensitivity to the drug and can be used as a biomarker for personalized breast cancer therapy of women at high risk of bone metastasis or with bone metastasis. We identified pathways and key molecules that coordinate both normal development and breast cancer metastasis, finding an Achilles heel for breast cancer treatment.

LAY SUMMARY

Given the importance of cholesterol biosynthesis to breast development during pregnancy and lactation, the results may indicate that women should discontinue taking cholesterol-reducing drugs before pregnancy and lactation. Cholesterol-reducing drugs have efficacy in prevention and treatment of breast cancer bone metastasis. A key biomarker which sensitizes breast cancer cells to treatment can provide personalized medicine options.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Integration of personal whole genome sequencing data for the management of adult cancer patients: a breast cancer feasibility study - Poster #625

Presenter: Patricia N. Tonin, McGill University

Other Authors: Aidan Cruikshank, Rui Li, Corinne Serruya, Amanda Lovato, Nicole Palmour, Janick St-Cyr, David Fleiszer, Yann Joly, Sarkis Meterissian, Jiannis Ragoussis

ABSTRACT_ID#2306

ABSTRACT

We developed a new biobanking framework to rationalize the integration of germline whole genome sequencing (WGS) to improve the management of cancer patients during their life course. We report the findings of the first phase involving 100 adult participants with a personal history of invasive breast cancer (BC) recruited at a Montreal hospital-based breast clinic. High quality WGS data was derived at the McGill Genome Centre using the cost effective MGI DNBSEQ-T7 technology. Principal component analyses revealed the diversity of participants consistent with self-reported ancestry data. The Cancer Predisposition Sequencing Reporter (CPSR) computational workflow was used to: select cancer associated genes; classify variants according to American College of Medical Genetics (ACMG) guidelines; detect germline biomarkers for prognosis, diagnosis or drug sensitivity/resistance in cancer; report incidental findings genes according to ACMG recommendations; and provide a detailed output file comprised of variant information, gene annotation and external link to relevant databases. Preliminary data has revealed: all 6 known and 8 additional carriers of clinically relevant variants in BC predisposing genes; 12 carriers of other cancer risk genes. A targeted analysis identified 1-93 carriers of 20 rare/common variants in 15 BC-therapy induced cardiotoxicity genes significantly associated with anthracyclines, Herceptin and/or radiotherapy. Although data was limited to research, WGS can provide specific and novel information with potential to impact health care of BC patients. Moreover, we achieved a high recruitment rate (74%) given the diversity of our study cohort (including Indigenous peoples) suggesting broad receptivity to integrating WGS in health care.

LAY SUMMARY

With increasing knowledge of the human genome there is a need to develop strategies to effectively identify clinically relevant heritable genetic biomarkers that could improve the care of cancer patients across their life course. Towards this goal we report on the: development of a new research-based biobank aimed at rationalizing the integration of unique variations in each person's genome to improve the personalized care of each adult patient diagnosed with cancer. Initial genetic findings of 100 participants recruited at a Montreal hospital-based breast clinic (as an example) showing that through the use of state-of-the-art whole genome sequencing technology and data analyses pipelines we were able to identify carriers of clinically relevant variants associated not only with breast cancer risk but risk to other cancers, impacting management of care; and as well identified carriers of pharmaco-genetic variants which may inform about risks to heart health with standard of care breast cancer treatments. achievement of a high recruitment rate (74%) which, given the self-reported ethnic diversity of our study population including Indigenous peoples, suggests a broad receptivity to integrating whole genome sequencing in health care.

Precision Medicine and Biomarkers

Monday, November 3, 2025

XevaDB: A cloud-based platform for open sharing of large-scale pharmacogenomics profiles of patient-derived xenografts for precision oncology - Poster #626

Presenter: Sisira Kadambat Nair, University Health Network

Other Authors: Guanqiao Feng

ABSTRACT_ID#2381

ABSTRACT

Despite the abundance of in vivo patient-derived models across institutions, limited visibility, incompatible formats, and experimental variation often hinder collaboration and full resource utilization. We propose to integrate clinical data and PDX pharmacogenomic profiles from the Princess Margaret Living Biobank (Ontario) and McGill Goodman Cancer Institute MUHC-RI's live biobank (Quebec) into a unified infrastructure. Our goal is to streamline access and reduce barriers to using multi-institutional PDX data for both Canadian and international researchers. Aims Integrate public in vivo pharmacogenomic datasets into FAIR multimodal objects. Establish an infrastructure to facilitate the curation and inter-lab integration across institutions. Implement and release of XevaDB for scalable, data sharing, visualisation and analysis of PDX data. Results We have developed the Xeva R/Bioconductor package (<https://www.bioconductor.org/packages/release/bioc/html/Xeva.html>) enabling storage of clinical, omics and drug response data along with the power tools for modelling and visualizing PDX growth curves. We implemented a prototype of web-application, called XevaDB (xevaldb.ca), for the concurrent visualisation of various PDX drug response metrics and associated molecular profiles. Impact XevaDB currently hosts 370 patient tumour samples, 733 genomic profiles, and over 128,000 PDX tumour growth measurements across diverse chemotherapy and targeted drugs. Despite not being formally launched, it has already seen global uptake, with 279 unique users from over 20 countries in 2023–2024 led by the USA (38.4%), Canada (37.6%), and China (7.5%). These early results underscore the platform's potential for broad, international data sharing and analysis.

LAY SUMMARY

PDX Data is hard to share: Even though many labs across Canada generate patient-derived tumor models (PDXs), collaboration is difficult due to data being stored in different formats and locations. We built a shared Cloud platform: Currently, we are combining PDX data from two major Canadian biobanks into a single system called XevaDB to make it easier for Canadian as well as international researchers to access and use this valuable cancer research data. XevaDB is already making an impact: With over 370 tumor samples and data from 20+ countries, XevaDB is helping scientists explore how different cancers respond to treatments before translating to patients.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Pre-diagnostic serum metabolome and breast cancer risk: a nested case-control study - Poster #627

Presenter: Jaclyn Parks, BC Cancer Research Institute

Other Authors: Ly Trinh, Treena McDonald, Andrew Roth, Grace Shen-Tu, Jennifer Vena, Rachel Murphy, Parveen Bhatti

ABSTRACT_ID#2405

ABSTRACT

Background: Metabolomics offers a promising approach to identify biomarkers for timely intervention and enhanced screening of individuals at increased risk of developing breast cancer. **Methods:** We conducted a study of 593 female breast cancer cases and 593 matched controls nested in two prospective cohort studies. Mass spectrometry, without liquid chromatography, was used to conduct untargeted metabolomics profiling of serum samples collected before cancer diagnosis. Logistic regression was used to estimate odds ratios (OR) for a one standard deviation increase of metabolite intensities. Partial least squares discriminant analyses were applied to those metabolites significantly associated with breast cancer to develop risk prediction models. **Results:** Associations were evaluated with a total of 837 metabolites. Twenty-four metabolites were significantly associated with overall breast cancer risk, including 13 associated with decreased risk and 11 associated with increased risk. Putative identifies of the metabolites included various pollutants, dietary factors, amino acids, fatty acids, phospholipids, and sex hormone derivatives. For example, a metabolite identified as acetyl tributyl citrate, a plasticizer in food wrappings, was associated with an increased risk of breast cancer (OR=1.21; 95% CI: 1.07-1.37). Risk prediction models for overall breast cancer and the various subtypes were found to have modest levels of prediction accuracy (area under the curve ranged from 0.60-0.63). **Conclusions:** Additional studies are needed to confirm the identities of the metabolites and validate their associations with breast cancer risk. Metabolomics should be evaluated in conjunction with other 'omics technologies for creation of clinically useful risk prediction models.

Precision Medicine and Biomarkers

Monday, November 3, 2025

The Guy Lafleur Precision Oncology Program: Bridging Big Data and Clinical Innovation - Poster #628

Presenter: Véronique Ouellet, CRCHUM

Other Authors: Christine Caron, Maxime Caron, Arielle Elkrief, Dominique Trudel, Réjean Lapointe, Anne-Marie Mes-Masson, Antoine Désilets

ABSTRACT_ID#3297

ABSTRACT

The Guy Lafleur Precision Oncology Research Program is a transformative initiative to accelerate personalized cancer care at the CHUM. In its first phase, the program focuses on building an integrated pipeline from participant tumor specimens and clinical data to optimized treatment strategy. By leveraging CHUM's high-quality biobanks, tissue preparation and sequencing facilities, data science and bioinformatician expertise, and clinician engagement, the program is advancing of precision oncology in clinical care. To date, 41 fresh-frozen tissue samples from high-grade serous ovarian carcinoma have been histologically reviewed and processed for DNA/RNA extraction. Germline DNA was also obtained. Among these, 28 samples met the minimal quality control criteria and yielded high-quality whole genome sequencing (both germinal and somatic) as well as whole transcriptome data (somatic). Variant calling was performed using DRAGENTM (Illumina) and GenPipes (Canadian Centre for Computational Genomics). Data will be integrated into the cBioPortal visualisation platform. A harmonized research report is being developed in alignment with national initiatives such as the pan-Canadian Marathon of Hope Cancer Centres Network (MOHCCN). A molecular tumor board will be launched to bring together multidisciplinary clinical and research experts to guide data interpretation, optimize reporting, and align the program with evolving precision oncology standards. The Guy Lafleur Program will provide strategic support to oncology care and research teams in their decision-making. Collaboration between clinical and basic research teams will foster advances in therapeutic innovation and professionals will have access to high-quality data in an environment conducive to knowledge exchange and discovery.

LAY SUMMARY

The Guy Lafleur Precision Oncology Research Program is a transformative initiative aiming to improve cancer care through personalized medicine at the Centre hospitalier de l'Université de Montréal (CHUM). Collaboration between clinical and research teams will foster new discoveries and accelerate advances in cancer discovery and therapeutic innovation. Treatment efficacy will be enhanced by tailoring therapies to each patient's unique cancer profile. This project will pave the way for more personalized and comprehensive care, with the goal of reducing side-effects, improving quality of life, and increasing patients' chances of recovery.

Precision Medicine and Biomarkers

Sunday, November 2, 2025

Mast cells as biomarkers for capecitabine benefit in triple negative breast cancer - Poster #629

Presenter: Katherine Rich, University of British Columbia

Other Authors: Elahe Shenasa, Dongxia Gao, Ali Bashashati, Torsten Nielsen

ABSTRACT_ID#3054

ABSTRACT

Background: Triple negative breast cancer (TNBC), accounting for 10-15% of cases, carries the worst prognosis and does not benefit from endocrine or HER2 targeted therapies. Clinical trials such as FinXX and CIBOMA found some TNBC patients do benefit from adjuvant capecitabine. RNA expression profiling found mast cell genes predicted capecitabine benefit in these trials. We explore whether deep learning can detect mast cells on hematoxylin & eosin (H&E) stained pathology slides, as an inexpensive predictive biomarker. Design: Using a contrastive learning framework, we trained a deep learning model that can identify mast cells from H&E-stained tissue sections. The model was trained on an internal UBC breast cancer series (n=283) and rigorously evaluated against CD117 positive controls, as well as serial breast cancer tissue microarrays stained with CD117 and tryptase. The model was then applied to tissue sections from the FinXX trial and compared with gene expression and outcome data. Results: The model achieved a balanced accuracy of 89.6%. In the UBC series tissue microarray, model predictions significantly correlated with CD117 and tryptase-stained cores ($R=0.43$, 0.41 respectively, $P<0.0001$). Mast cell predictions were significantly correlated to the average gene expression of mast cell markers ($R=0.34$, $P<0.001$). Comparison with outcome data from the FinXX trial will be presented. Conclusion: We have developed and validated a robust algorithm to detect mast cells from H&E stained tissue to help determine if mast cells are a predictive biomarker for capecitabine benefit in TNBC.

LAY SUMMARY

We found that machine learning can reliably detect mast cell presence from standard tissue images. This gives us a quick and cost-effective way of detecting and evaluating potential biomarkers. Our mast cell classifier was thoroughly validated. It outperformed a pathologist's annotations when compared to gold-standard immunohistochemistry (IHC) stains, showing stronger correlation with the true presence of mast cells. Mast cells may help identify which triple negative breast cancer patients benefit from capecitabine. While past studies relied on gene expression data, our model brings us closer to confirming mast cells as a biomarker for guiding treatment decisions.

Precision Medicine and Biomarkers

Monday, November 3, 2025

D-Dimer as a Prognostic Marker in Active Cancer Patients with Ischemic Stroke – a Systemic Review and Meta-Analysis - Poster #630

Presenter: Yu Hao, University of Calgary

Other Authors: Danielle Roy, Claire Wells, Faizan Khan, Candice Poon, Michael Hill, Andrew Demchuk, Phil Wells, Ronda Lun

ABSTRACT_ID#3049

ABSTRACT

Background: Ischemic stroke in active cancer patients results in significantly higher morbidity and mortality compared to non-cancer patients. Multiple cohort studies have examined d-dimer as a surrogate marker for outcomes in cancer-associated ischemic stroke but are limited by small sample sizes. Our objective was to conduct a systematic review and meta-analysis of existing literature on the prognostic value of serum d-dimer measured at the time of ischemic stroke for active cancer patients. Methods: We searched for studies published before July 2024 that measured serum d-dimer in active cancer patients at the time of the ischemic stroke, which reported at least one outcome of interest: death, recurrent stroke, and poor functional outcomes (mRS 3-6). For primary analysis, we used a random-effect meta-analysis model to pool the median difference in d-dimer levels between those with the outcomes of interest versus those without. Results are reported as median differences. Due to studies having different cutoffs for effect measures, studies that could not be pooled were narratively summarized using forest plots. Results: Of the 5152 titles and abstracts screened, 23 eligible studies reported a measure of association between serum d-dimer and an outcome of interest. D-dimer was significantly higher in patients who died (median difference 11.18 [95%CI 9.67-12.70], $p < 0.001$), and also significantly higher in patients who experienced recurrent stroke (median difference 14.54 [95%CI 2.09-26.99], $p = 0.022$). Conclusion: Serum d-dimer measured at the time of ischemic stroke in patients with active cancer could help predict recurrent stroke and mortality but is not predictive of functional outcomes.

LAY SUMMARY

Patients with active cancer and ischemic stroke have significantly worse outcomes compared to stroke patients without cancer. Out of 5152 titles and abstracts screened, 23 eligible studies that reported an association between serum d-dimer levels and mortality, recurrent stroke or poor functional outcomes were included within the meta-analysis. D-dimer levels measured at the time of the stroke were significantly higher in active cancer patients who died, and active cancer patients who experienced recurrent stroke during the follow-up period, compared to patients with no outcomes of interest. D-dimer levels measured at the time of the stroke were not significantly different between active cancer patients with good vs poor functional outcomes. Serum D-dimer measured at the time of the ischemic stroke in active cancer patients could help predict mortality and recurrent stroke but does not predict the patient's functional outcomes.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Development of novel Hsp90 inhibitor MHR21 for Auger therapy - Poster #635

Presenter: Alexandra Kennedy, University of Alberta

Other Authors: Miguel Herrera Rueda, Valeria Mancinelli, Frank Wuest, Paul LaPointe

ABSTRACT_ID#3264

ABSTRACT

Radiotheranostics is a minimally invasive technique for diagnosing and treating cancer, however, it is limited by the availability of targetable proteins and high-affinity molecules. One potential target is the 90 kDa heat shock protein (Hsp90), a chaperone essential for protein folding and stabilization. Cancer cells rely heavily on Hsp90, and Hsp90 inhibitors have been found to have a tumor-retention property. Onalespib (AT13387), a potent Hsp90 inhibitor in phase III clinical trials, served as the basis for our Structure-Activity Relationship (SAR) study. We synthesized 10 Onalespib analogues, replacing the isopropyl group on the resorcinol moiety with bulky atoms such as bromine and iodine to mimic its steric effects. The piperazine ring was also substituted with chiral scaffolds, R/S quinuclidine and R/S piperidine, to assess the influence of the aliphatic moiety on biological activity. In vitro assays showed that non-chiral compounds MHR12 and MHR17 had the lowest affinity for Hsp90, while R-stereochemistry analogues MHR16 and MHR21 had the highest. Additionally, brominated analogues MHR17, MHR20, and MHR21 exhibited the strongest disruption of Hsp90 client signaling in cell-based assays. Lastly, we used microscopy techniques to show that Hsp90 inhibition does not alter Hsp90 localization. Our findings indicate that substituting the isopropyl group with iodine or bromine in the resorcinol framework of Onalespib preserves Hsp90 inhibitory activity. MHR21 emerged as a lead compound and will be radiolabeled with ⁷⁷Br for further evaluation as a potential radiotheranostic agent, specifically for Auger therapy, which involves the precise delivery of low-energy Auger electrons to DNA.

LAY SUMMARY

The activity of Hsp90 inhibitor Onalespib (AT13387) is conserved following the replacement of the isopropyl group for bromine or iodine. The stereochemistry of our compounds has an effect on their affinity; compounds with R-stereochemistry had a higher affinity for Hsp90. Our lead compound, MHR21, is a promising candidate for Auger therapy.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Advantages of the Clinical Alberta linac-MR system - Poster #636

Presenter: B. Gino Fallone, Cross Cancer Institute University of Alberta

ABSTRACT_ID#2414

ABSTRACT

The Alberta Linac-MR (LMR) is a novel inline magnetic resonance-guided radiotherapy (MRgRT) system developed by the University of Alberta and Cross Cancer Institute. It integrates a 6 MV linear accelerator with a bi-planar 0.5 T open-bore MRI, uniquely aligning the radiation beam parallel to the magnetic field (Bo). This parallel configuration significantly reduces the Lorentz force, minimizing dose perturbations at tissue-air interfaces, and improves dosimetric accuracy and geometric consistency compared to all other MR-linacs. The LMR's large open bore and ± 23 cm 3D couch shift capabilities enable isocentric treatment of peripheral tumors, reduce claustrophobia, and facilitate precise tumor positioning—features not possible with other systems. Its design simplifies mechanical integration, improves electromagnetic compatibility, and supports efficient adaptive workflows. Custom shielding minimizes interference between the linac's RF emissions and the MR system's signals. The platform supports real-time imaging at 4 frames per second, including T2-weighted and diffusion-weighted sequences, with direct beam's-eye-view imaging enabled by its rotational design. Dosimetric commissioning shows minimal Bo-related effects, with ion chamber measurements and Monte Carlo simulations aligning well within treatment planning systems, particularly significantly, minimizing dose-perturbations at tissue-air/lung interfaces compared to the other MR-linacs. Optimized for treating mobile and anatomically complex tumors (e.g., pancreas, liver, lung, pelvis), the LMR supports margin reduction, gating, dose escalation, and organ-at-risk sparing. Its open architecture fosters translational research with full access to MR sequences, beam control, and software—enabling development of biologically adaptive protocols and AI-driven workflows. The Alberta Linac-MR is a clinically and scientifically advanced MRgRT platform.

LAY SUMMARY

More Accurate, Safer Cancer Treatment: The Alberta Linac-MR (LMR) combines an MRI scanner with a radiation machine in a unique way—aligning the radiation beam with the magnetic field. This setup improves accuracy and reduces unwanted radiation effects, especially near air-filled spaces like lungs. **Patient-Friendly and Flexible Design:** Its open, wide-bore design reduces feelings of claustrophobia and allows treatment of hard-to-reach tumors. A movable treatment couch helps doctors precisely target tumors (isocentric) in various body areas, including the lungs, liver, and pelvis. **Advanced Real-Time Imaging:** The system provides clear, real-time MRI images (4 frames per second), helping doctors track tumors during treatment. It even offers a "beam's-eye view," letting clinicians see exactly where the radiation is going. **Supports Innovation and Research:** LMR is designed to be research-friendly, giving full access to imaging, software, and controls. This allows development of new, personalized treatment strategies, including the use of AI and biologically adaptive radiation plans. In short, LMR is a next-generation cancer treatment platform to treat all solid cancer tumors offering high precision, improved patient comfort, and strong support for clinical research and innovation.(iso

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Perioperative Pain Management for Breast Cancer Surgeries: A National Survey of Canadian Anesthesiologists - Poster #637

Presenter: Cindy Chang, University of Toronto

Other Authors: Gary Ko, May Lynn Quan, David Lim, Sharon Peacock, Stephen Choi, Hance Clarke, Sinziana Avramescu, Richard Brull, Karim Ladha, Allana Munro, James Khan

ABSTRACT_ID#2967

ABSTRACT

Chronic postsurgical pain affects up to 46% of patients after breast cancer surgery. Anesthetic techniques such as regional anesthesia and intravenous (IV) lidocaine infusions have been suggested to reduce this risk. We aimed to investigate perioperative pain management practices among Canadian anesthesiologists, focusing on these emerging modalities. An anonymous, cross-sectional survey was distributed in March 2025 to all English-speaking members of the Canadian Anesthesiologists' Society. The survey assessed preferences for analgesic techniques in lumpectomy and mastectomy. Eighty-three anesthesiologists completed the survey (6% response rate). Oral acetaminophen, NSAIDs and opioids were the most used agents. Regional anesthesia (29% vs. 13%) and intraoperative IV lidocaine (20% vs. 11%) were used more often in mastectomies than lumpectomies. Routine use of regional anesthesia (23%) and IV lidocaine (22%) was mainly driven by goals to reduce opioid use and chronic pain risk. Key barriers included institutional practices, time constraints, and perceived inefficacy. There is considerable variability in pain management strategies for breast cancer surgery. While acetaminophen, NSAIDs, and opioids remain the mainstay, regional anesthesia and IV lidocaine infusions are underutilized despite their potential to reduce persistent postsurgical pain. Given the significant burden of chronic pain following breast cancer surgery, these findings underscore the need for further research and targeted interventions to support evidence-based practices. This study highlights the need for standardized, evidence-based guidelines and system-level support to facilitate broader adoption of multimodal analgesic strategies. Addressing these gaps is essential to improving long-term pain outcomes and overall quality of life for breast cancer patients.

LAY SUMMARY

This national survey highlights considerable variability in perioperative pain management strategies among Canadian anesthesiologists caring for breast cancer patients. While systemic analgesics, particularly acetaminophen, NSAIDs, and opioids, remain the mainstay across all phases of care, emerging modalities like regional anesthesia and IV lidocaine infusions are underutilized despite their potential to reduce persistent postsurgical pain. Only 23% and 22% of respondents routinely used regional nerve blocks and intravenous lidocaine infusions, respectively. The primary rationale for adopting regional anesthesia and IV lidocaine was to minimize opioid use and mitigate chronic pain risk. However, barriers such as institutional norms, perceived inefficacy, and time constraints hinder broader implementation. Given the significant burden of chronic pain following breast cancer surgery, these findings underscore the need for further research and targeted interventions—such as updated clinical pathways, enhanced regional anesthesia training, and resource allocation—to support evidence-based practices. Future research should focus on overcoming institutional barriers and exploring long-term outcomes associated with broader adoption of these analgesic adjuncts. Addressing these gaps is essential to improving long-term pain outcomes and overall quality of life for breast cancer patients.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Evaluation of Efficacy in Prostate Cancer Contouring for MR-Guided Radiotherapy Using Contouring Training Modules - Poster #638

Presenter: Sangjune Lee, University of Calgary

Other Authors: Breanna Fang

ABSTRACT_ID#2304

ABSTRACT

Introduction: Prostate cancer is the second most common cancer in males and a leading cause of cancer-related death globally. In Canada, over 24,000 new cases and 4,500 deaths are expected annually. MR-guided radiotherapy offers precise contouring and reduced toxicity, with adaptive features allowing dose adjustments based on individual responses. However, this workflow is resource-intensive, with contouring being a time-consuming step. Methods: MRI scans from prostate cancer patients treated in Alberta and from The Cancer Imaging Archive (TCIA) were used. Radiation oncology (RO) residents in Alberta participated between March and July 2024. After contouring 5 baseline cases, they completed a Contour Training Module and contoured 5 additional cases. In the final phase, they received individualized feedback and a training video before contouring 5 final cases. Contours were compared to a gold standard set by Calgary radiation oncologists. Outcomes included: (1) volumetric conformity to assess accuracy, and (2) time spent per case to assess efficiency. Results: We have completed a literature search and developed training modules to teach prostate MR contouring. Automated tools using Boolean logic were implemented to provide visual and quantitative feedback for training and assessment. Conclusions: Findings of this study will inform future strategies to train RO residents to perform MR contours. With the two MR-Linac machines recently installed at the Arthur Child Cancer Centre, there's an increasing need for optimization of the skillset and responsibilities of the multidisciplinary team to maximize efficiency. This study lays the groundwork for future research in MR contour training.

LAY SUMMARY

- **Improving Contour Training:** We created automated tools using Boolean logic to provide visual and quantitative feedback to help radiation oncology residents improve their contouring skills to ultimately better treatment outcomes and reduce side effects for patients.
- **Assessing Tools for Learning:** We created educational modules and using automated tools provided individualized feedback to give residents feedback on how closely their work matches expert standards. We aim to assess the efficacy of these tools to guide future research in MR contour training.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Deoxymabs for Targeted Meitner-Auger Electron Therapy of Breast Cancer - Poster #639

Presenter: Chahat Suri, University of Alberta

Other Authors: Frank Wuest, Jenilee Woodfield, Samantha Laier, Cody Bergman

ABSTRACT_ID#2359

ABSTRACT

Conventional radiotherapy frequently lacks tumor specificity, leading to off-target toxicity in over 60% of breast cancer patients. This project aims to enhance therapeutic precision by employing Auger electron-emitting radioisotopes delivered directly to breast cancer cell nuclei using tumor-homing, DNA-binding antibodies DX3 and antibody fragment DX1. These deoxymabs target nucleoside transporters hENT1/2 to achieve selective uptake. Live-cell imaging with Alexa Fluor 488-labeled DX3 and DX1 confirmed nuclear internalization in MDA-MB-231 and MCF7 cells. Cytotoxicity assays indicated minimal intrinsic toxicity of DX3 and DX1 in MCF7 and MDA-MB-231 cells over 72 hours. For radiolabeling, DX3 and DX1 were functionalized with the chelator NOTA and labeled with ^{64}Cu . Cellular uptake studies showed ~80% internalization of ^{64}Cu -labeled DX3 and DX1 within 60 min. Competitive uptake assays with the hENT1/2 radioligand [^{18}F]FLT demonstrated effective nucleoside transporter engagement in breast cancer cell lines MDA-MB-231 and MCF-7, with 50% and 40% inhibition at 25 μM (DX3) and 46 μM (DX1), respectively. Nuclear localization and hENT1/2-mediated uptake validate DX3 and DX1 as promising DNA-targeting vectors for Auger electron therapy. Ongoing work focuses on radiolabeling DX3 with the novel Meitner-Auger emitter ^{135}La and evaluating its therapeutic efficacy in preclinical breast cancer models.

LAY SUMMARY

Conventional radiotherapy often harms healthy tissues because it doesn't specifically target cancer cells. Our research uses special antibodies, DX3 and DX1, that can enter breast cancer cells and go directly to their nuclei. These antibodies are designed to recognize and bind to nucleoside transporters (hENT1/2), which are commonly overexpressed on breast cancer cells. Live-cell imaging confirmed that both DX3 and DX1 successfully reached the nuclei of breast cancer cells, making them an ideal model to deliver auger electrons to breast cancer cells. We found that DX3 and DX1 antibodies by themselves do not harm healthy cells, making them safe carriers for radiation. Also, when tagged with the radioactive isotope copper-64 (^{64}Cu), about 80% of the dose was internalized into the nucleus of breast cancer cells within 60 minutes. To ensure these antibodies are using hENT1/2 to enter cells, we ran tests using a known radioligand called [^{18}F]FLT that goes through hENT1/2. Our antibodies were able to compete effectively, reducing FLT uptake by 40–50%, confirming that they are actively engaging the right transporters. This selective uptake is a promising sign for using these antibodies to deliver next-generation Meitner-Auger electron emitters like ^{135}La for treating breast cancer more precisely and effectively.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Real-Time 3D Treatment Volume Tracking to Mitigate MR-LINAC Latencies - Poster #640

Presenter: Elodie Lugez, Toronto Metropolitan University

ABSTRACT_ID#2390

ABSTRACT

Magnetic Resonance-guided Linear Accelerators (MR-LINACs) offer real-time imaging during radiation therapy using 2D cine images to monitor patient anatomy. However, MR-LINACs experience system latency—approximately 200 ms each for image acquisition and processing—posing a challenge for real-time target tracking. To address this shortcoming, a predictive strategy using LightGBM, a machine learning model known for efficiency and strong performance with limited data, was explored. Target displacements were computed by registering each incoming cine image to the 3D reference image. Each patient-specific LightGBM model was initially trained on the first 60 displacements and continuously updated with new data. The models then predicted the next three displacements to compensate for future latency. The proposed method was evaluated on nine datasets, each containing a 3D reference image, 2D cine series, and corresponding ground truth displacements. Predictions were assessed at 400 ms and 600 ms ahead. Without prediction, latency led to average tracking errors of 2.62 mm and 3.46 mm, respectively. LightGBM significantly reduced these errors to 1.37 mm and 1.83 mm ($p < 0.05$), correcting over 47% of the latency-induced inaccuracy. These results highlight the potential of adaptive, patient-specific models to enhance real-time tracking accuracy in MR-guided radiotherapy, potentially allowing for tighter treatment margins and improved clinical outcomes.

LAY SUMMARY

- New MRI-guided linear accelerators (radiation machines) can acquire real-time images during treatment. They are used to track the motion of the treatment volume. However, delays in capturing and processing images can lead to less accurate targeting of the tumor.
- Machine learning helps predict tumor movement. This work presents a personalized prediction model using a fast and efficient machine learning method called LightGBM. This model learns how a tumor moves and predicts where it will be next.
- The proposed method reduced the average tracking error caused by system delays by over 47%. Improved accuracy in computing the treatment volume position may lead to better treatment.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Surgical margin assessment of bone tumours: A systematic review of current and emerging technologies - Poster #641

Presenter: Haitham Shoman, Concordia University

Other Authors: Jawad Al-Kassmy, Maryam Ejaz, Kalin Kahla, Mario Dacunto

ABSTRACT_ID#2593

ABSTRACT

Osteosarcoma is the most common malignant bone tumour. Complete surgical excision is critical to achieve optimal outcomes and lower recurrence rates. However, accurate assessment of tumour margins remains a challenge and multiple technologies are employed for this purpose. The aim of this study is to highlight current and emerging technologies and their efficacy in detecting clear bone margins intraoperatively. Databases searched include: Medline, Embase, Global Health and Google Scholar. Studies were screened using predetermined eligibility criteria. Data was extracted based on study and patient characteristics, modes of detection, and commercial availability, followed by quality assessment. A total of 17 studies were included. The primary diagnosis varied, with osteosarcoma being reported by 9 studies. Three studies reported relapse, ranging between 17.6%–48%. Twelve studies reported non-invasive imaging as the mode of detection used, while 4 studies reported the use of frozen section. MRI and CT were found to have an accuracy of up to 93 %. Raman spectroscopy was reported to have an accuracy, sensitivity, and specificity of 69%, 58.8% and 83.3% respectively. CT had a sensitivity and specificity up to 83% and 100%, respectively. In conclusion, there seems to be high potential for the use of multimodal technologies to increase the accuracy of intraoperative margin assessment. Although imaging modalities possess a fair level of accuracy, they carry the risk of radiation exposure, are expensive, and cannot be used in-situ. Future clinical trials are needed to test the effectiveness of these technologies to measure the diagnostic accuracy and overall patient survival.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Population-based experience of neoadjuvant treatment for patients with gastrointestinal stromal tumors (GIST) in Alberta - Poster #642

Presenter: Emily Heath, Alberta Health Services

Other Authors: Omar Khan, Lloyd Mack

ABSTRACT_ID#3312

ABSTRACT

Introduction GISTs are the most common mesenchymal tumour in the gastrointestinal tract. Surgical resection remains the mainstay of treatment for localized GIST. When R0 resection is not feasible or is morbid, neoadjuvant therapy is indicated. We aim to describe the population, tumour characteristics, and outcomes for GIST patients treated with neoadjuvant imatinib in Alberta. **Methods** A retrospective cohort study was conducted of GIST patients diagnosed between 2008-2022. Data were abstracted from the provincial cancer registry. Chart review confirmed imatinib dosing, tumour characteristics, surgical outcomes, and recurrence. Tumour response was evaluated using RECIST criteria. **Results** 693 GIST cases were identified from 2008-2022. Of these, 42 had primary non-metastatic disease and received neoadjuvant imatinib. 54.8% had primary gastric tumours, 61.9% were 5-10cm and 73.8% were high risk by NIH criteria. Median duration of imatinib therapy was 283.5 days, with median cumulative dose of 113g. Five patients had dose escalation above 400mg/day, 16 required dose reduction. At time of surgery 21(50%) had partial response, 18(42.9%) had stable disease, 3(7.1%) had progressive disease. 1/23 with gastric GIST underwent total gastrectomy, 6/8 with rectal GIST underwent APR. No patients required a Whipple. R0 resection was achieved in 85.7%. For patients treated with neoadjuvant imatinib, median follow up was 55.7 months. Five(11.9%) patients died, six(14.3%) had local recurrence, seven(16.7%) had distant metastases on imaging, and 64.2% remain disease-free. **Conclusion** Dosing and duration of neoadjuvant imatinib for non-metastatic GIST patients in Alberta is individualized. Imatinib facilitates R0 resection in most, with favourable surgical and long-term outcomes.

LAY SUMMARY

1. Pre-operative imatinib therapy is helpful to shrink GIST tumours and improve surgical outcomes.
2. Dosing and duration of pre-operative imatinib therapy for GISTs is individualized for each patient.
3. Overall survival and recurrence outcomes for patients with GISTs treated with pre-operative imatinib therapy are favourable.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Advancing the most accurate radiotherapy for mobile tumours with hybrid Linac-MRI Systems - Poster #643

Presenter: Jihyun Yun, Cross Cancer Institute

Other Authors: Keith Wachowicz, Eugene Yip, Satyapal Rathee, B. Gino Fallone

ABSTRACT_ID#2314

ABSTRACT

Accurate radiation delivery to mobile tumours (e.g., lung or liver) remains an unresolved challenge in modern radiotherapy, primarily due to their motion and deformation caused by respiratory and cardiac activity. Unlike stationary tumours (e.g., brain), which allow sub-millimetre targeting, mobile tumours introduce localization uncertainties of up to several centimetres. Invasive approaches using surgically implanted markers exist but carry risks such as misalignment and medical complications. Targeting uncertainty stems largely from the lack of real-time tumour imaging during irradiation, often requiring expanded beam margins to ensure full tumour coverage—at the cost of increased radiation exposure to nearby critical organs. Minimizing these margins requires continuous visualization of tumour position and shape during irradiation, currently achievable only with hybrid linear accelerator–MRI systems. Our lab developed one such system—the Alberta Linac-MR—and launched clinical trials in 2023. With the Alberta Linac-MR, our vision is to deliver the most accurate, non-invasive radiotherapy for mobile tumours. To achieve this, we developed non-invasive intra-fractional tumour-tracked radiotherapy (nifteRT)—a technique that integrates real-time MRI, tumour auto-contouring, motion prediction, and dynamic beam modulation during irradiation. In 2013, we achieved the first-ever demonstration of nifteRT in a phantom study, delivering radiation with sub-1.7 mm accuracy to a moving lung tumour target. Recently, we enhanced nifteRT with advanced AI: CNNs for accelerated MRI, U-Net for tumour auto-contouring, and LSTM for motion prediction. Our work has been published and recognized with prestigious awards at international scientific conferences. Successful nifteRT will provide a novel, non-invasive radiotherapy solution for treating mobile tumours in the thoracoabdominal regions.

LAY SUMMARY

Mobile tumours are difficult to treat with radiotherapy. Radiotherapy uses high-energy radiation to destroy cancer cells. When tumours are stationary—like those in the brain—they can be targeted with sub-millimetre accuracy, minimizing damage to healthy tissue. In contrast, mobile tumours—such as those in the lungs or liver—move and deform with each breath and heartbeat, making accurate targeting very difficult. To compensate, doctors often enlarge the irradiation area, increasing the risk to nearby organs. We developed a new system to see tumours in real-time. Our lab invented the Alberta Linac-MR, a machine that combines MRI with radiation delivery, enabling real-time tumour tracking during treatment. Clinical trials began in 2023. We created an AI-powered, non-invasive treatment approach. Using the Alberta Linac-MR, we are developing nifteRT—a novel technique that tracks tumour motion in real-time without requiring invasive procedures. Unlike conventional methods relying on surgically implanted markers or enlarged margins, nifteRT dynamically adjusts the radiation beam as the tumour moves—like a smart spotlight—delivering highly accurate and safer radiotherapy. We recently enhanced nifteRT with advanced AI technology to accelerate MRI, automatically contour tumours, and predict motion. With these upgrades, successful nifteRT will provide a novel, non-invasive radiotherapy solution for treating mobile tumours in the thoracoabdominal regions.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Evaluating the Impact of a 0.5T Magnetic Field on Radiation-Induced DNA Damage Repair in Normal Fibroblasts Using a biplanar Linac-MR - Poster #644

Presenter: Armin Gamper, University of Alberta

Other Authors: Michael Zhang, Alireza Gazor, B. Gino Fallone, Tania Wood

ABSTRACT_ID#2981

ABSTRACT

Over half of cancer patients get radiation therapy (RT) during treatment, an estimated 40% of cures are attributed to RT. Recently, MR-Linacs have entered the clinic, allowing 4-dimensional RT using MRI, tracking tumours in real-time during treatment. Using a magnetic field during RT introduces a new factor that could impact side effects such as radiation dermatitis (RD). Given the high prevalence and profound impact of RD on patients, we need to understand how the magnetic field of MR-LINACs impacts patient skin to prevent or mitigate RD. The Alberta MR-LINAC is unique for its use of a biplanar MRI with a magnetic field parallel to the radiation beam. In megavoltage photon RT, the deposited dose rapidly rises beyond the patient surface, reaches a maximum at depth d_{max} , and then slowly decreases. The buildup region creates a 'skin-sparing' effect. To study the magnetic field influence on a) dose deposition and b) biological consequences of the dose deposited, we investigated skin DNA damage and its repair using skin fibroblasts. Our data show that the effects on the surface dose due to contaminant electron particles in the air column upstream of the patient match the prediction by our Monte Carlo simulations, showing our ability to model and investigate this effect. Our findings indicate that the presence of the magnetic field does not significantly alter the DNA damage induction in fibroblasts once corrected for the increased dose at the surface in this device. Next steps involve characterizing potential changes in DNA repair kinetics and signaling.

LAY SUMMARY

MR-Linacs (MRI integrated with a linear accelerator) have entered the clinic, allowing tumour tracking in real-time during radiation treatment and reducing doses to organs surrounding the tumour target. Using a magnetic field for tumour tracking during radiation therapy (RT) introduces a new factor that could impact side effects such as radiation dermatitis. Radiation dermatitis (skin damage) is a common side effect of RT. Preventing or mitigating radiation dermatitis would greatly improve patient outcomes and quality of life during and after RT. The Alberta MR-LINAC is unique for its use of a biplanar MRI with a magnetic field parallel to the radiation beam. While avoiding potential more serious side effects expected in other configurations, the magnetic field design increases the doses deposited on the skin surface. Our research shows that the DNA damage in skin fibroblasts correlates with the expected values based on simulations to predict changes in dose deposition. Furthermore, once corrected for the dose deposited, the magnetic field does not have a significant effect on the initial DNA damage. Ongoing studies examine whether the magnetic field can influence the DNA damage response, such as DNA damage repair and signalling, both of which could affect the induction of radiation dermatitis.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

A narrative review of evidence-based practices to enhance recovery from uveal melanoma surgery - Poster #645

Presenter: Michelle Pham, University of Calgary

Other Authors: Paige Campbell, Ezekiel Weis, Trafford Crump

ABSTRACT_ID#3099

ABSTRACT

Background: Uveal melanoma is the most common intraocular cancer in adults. Surgical management achieves high rates of local tumour control but can be associated with peri- and post-operative morbidity. A well-coordinated perioperative care pathway may address these risks. Objective: To review and synthesize state-of-the-art evidence for optimizing patient recovery following uveal melanoma surgery. Methods: A targeted literature search was conducted across PubMed, Embase, and relevant ophthalmology guidelines. Original research, expert consensus statements, and clinical practice recommendations addressing perioperative optimization in ocular oncology were identified. Relevant findings were categorized into preoperative, intraoperative, or postoperative management to create a framework for recommended practices. Results: Preoperative care should include comprehensive patient education regarding the surgical process, expected recovery timeline, and warning signs of complications. Nutritional support, appropriate use of eye drops, and management of ocular surface conditions can facilitate healing and help prevent complications. During surgery, employing techniques that minimize tissue trauma, maintain ocular pressure stability, and prioritize pain management is recommended. Multimodal analgesia protocols can decrease opioid requirements and enhance recovery. Postoperatively, providing structured recovery plans is recommended. Patients should avoid activities that increase intraocular pressure, limit exposure to waterborne pathogens, and use protective eyewear. Clear guidance on medication management and hygiene practices can further improve outcomes. Conclusions: A coordinated perioperative care pathway can enhance recovery, reduce complication rates, and improve patient satisfaction following uveal melanoma surgery. Prospective studies are warranted to validate these recommendations and establish standardized clinical guidelines to enhance recovery after ocular oncologic surgery.

LAY SUMMARY

Patients undergoing ocular oncologic surgery can face peri- and post-operative morbidity. The development of a standardized, evidenced-based perioperative care pathway may minimize these risks. Recommendations to enhance recovery after uveal melanoma surgery include preoperative (patient education, nutritional support, and addressing pre-existing ocular surface conditions), intraoperative (optimizing surgical techniques and analgesia), and postoperative (providing structured recovery plans) practices.

Radiotherapy and Surgical Interventions

Monday, November 3, 2025

Evaluating Radiation Techniques: Optimizing Protocols for Lung Cancer Treatment Planning – Preliminary Work - Poster #646

Presenter: Mackenzie Moring, University of Calgary

Other Authors: Charles Kirkby

ABSTRACT_ID#3204

ABSTRACT

Background: The presence of very low-density (VLD) media and physiological motion complicate radiation therapy treatment planning for lung cancer. Treatment planning systems increase the fluence through VLD media to achieve the prescribed dose to the treatment volume, but can inaccurately calculate the dose to VLD media. **Purpose:** To characterize dose as a function of Hounsfield Unit (HU) for different CT simulators and treatment planning algorithms — including Anisotropic Analytical Algorithm (AAA), AcurosXB (AXB), and, in the future, Monte Carlo — as preliminary work to improve the dosimetry of lung stereotactic body radiation therapy (SBRT). **Methods:** We created a water-equivalent phantom ($40 \times 20 \times 40 \text{ cm}^3$) containing a central cavity ($32 \times 4 \times 40 \text{ cm}^3$) in Research ARIA, delivered 100 Monitor Units, and calculated the dose to isocenter using both AAA and AXB. The cavity's HU varied from -650 to -1000. This process was performed for the CT simulator at the Jack Ady Cancer Centre, and the CT and the PET/CT simulators at the Arthur J.E. Child Comprehensive Cancer Centre. Monte Carlo will be used as a ground truth for dose deposition in VLD media. **Results:** AAA overestimates the dose in VLD media, while AXB exhibits the expected decrease in dose deposition as HU decreases. Below approximately -950 HU, each curve plateaus, due to the calibration curves defining air above -1000 HU. Monte Carlo is expected to align more closely to the AXB results, but without the VLD plateau. **Conclusion:** This characterized relationship between VLD media and dosimetry will enable further work to improve lung SBRT planning.

LAY SUMMARY

Common treatment planning algorithms experience limitations when calculating the dose to very low-density media such as air. Air is commonly included in the planning target volume for lung cancer radiation treatments. Treatment planning systems will apply a higher fluence of radiation through very low-density media to achieve the prescribed dose to the treatment volume, but they can also inaccurately calculate the dose to very low-density media. When a patient breathes during treatment, their lung tumor can move into a region where the treatment planning system has planned for a very low-density media, causing it to receive too much dose. Each CT simulator has its own unique calibration curve that assigns a Hounsfield Unit (HU) to a voxel in the CT scan based on the density of the tissue being imaged. Characterizing the relationship between dose and HU can help us identify the HU threshold where the very low-density regime begins and further improve lung cancer radiation therapy treatments.

Radiotherapy and Surgical Interventions

Sunday, November 2, 2025

Development of a Magnetically Actuated Capsule for Targeted Drug Delivery, Biopsy, and In Situ Monitoring in Gastrointestinal Cancer Applications - Poster #647

Presenter: Onaizah Onaizah, McMaster University

Other Authors: Shivam Gupta

ABSTRACT_ID#2312

ABSTRACT

Early detection, localized treatment, and continuous monitoring are critical to improving outcomes in gastrointestinal (GI) cancers. Conventional approaches often rely on tethered endoscopes, which can be uncomfortable, invasive, and limited in their reach. In this work, we present the development of a magnetically actuated, untethered capsule designed for targeted drug delivery, tissue biopsy, and in situ monitoring within the GI tract. Leveraging external magnetic fields, the capsule achieves wireless navigation and activation, enabling it to access challenging anatomical regions with high precision and minimal discomfort to the patient. The capsule integrates three core functionalities: (1) a magnetically triggered drug reservoir for localized therapeutic release, (2) a minimally invasive biopsy mechanism for tissue sampling, and (3) embedded sensing capabilities for environmental monitoring. Phantom model experiments demonstrate the capsule's ability to navigate complex pathways, deploy therapeutic payloads, and retrieve tissue samples with sufficient force and minimal trauma. Control of the capsule is achieved through a custom magnetic actuation platform and real-time tracking system, allowing for closed-loop manipulation in dynamic environments. This multifunctional platform addresses key limitations in current GI cancer diagnostics and therapeutics by offering a non-invasive, precise, and modular alternative. Future work will focus on preclinical validation and optimizing the magnetic control algorithms to improve usability in clinical settings. This technology represents a significant step toward more accessible and patient-friendly cancer care.

LAY SUMMARY

We are developing a small, pill-sized robot that can move through the digestive system without any wires. Using magnets outside the body, we can guide this capsule to specific areas of the gastrointestinal (GI) tract. This makes it easier to reach difficult-to-access regions without the discomfort or limitations of traditional endoscopy. This capsule can deliver cancer drugs exactly where they are needed. By carrying a small reservoir of medication, the capsule can release treatments directly at a tumour site, reducing side effects and improving the effectiveness of the therapy. It can also take tiny tissue samples and monitor the local environment. The capsule includes a tool for collecting biopsies and sensors to check conditions like temperature or pH, helping doctors diagnose and track disease without repeated invasive procedures.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Circulating microRNA-375 enables blood-based detection and staging of lung neuroendocrine neoplasms - Poster #648

Presenter: Simona Damiani, Queen's University at Kingston

Other Authors: Dong Yao Wang, Zoe Manuel-Epstein, Yousif Al-Aboosi, Xiaojing Yang, Genevieve Digby, Neil Renwick, Kathrin Tyryshkin

ABSTRACT_ID#2616

ABSTRACT

Introduction: Lung neuroendocrine neoplasms (NENs) are a heterogeneous group of increasingly common tumors that are often diagnosed in late-stage disease. microRNAs (miRs) are small regulatory RNA molecules that can be excellent biomarkers due to their abundance and stability in tissues and biofluids. Recent work identified that miR-375 is higher in lung NEN compared to non-NEN tissues (PMID: 32743554). In this study, we evaluated the clinical utility of plasma miR-375 as a marker of lung NEN diagnosis and progression. **Methods:** Platelet-depleted plasma samples were collected from 135 pre-treatment patients [41 lung NEN; 30 lung squamous cell carcinoma (LUSC); 30 lung adenocarcinoma (LUAD); and 34 non-neoplastic lung disease (NNLD)]. We quantitated miR-375 relative abundance using real-time PCR. Results are expressed as mean relative cycle threshold value $\pm$ standard deviation. miR-375 expression was investigated in 20 formalin-fixed paraffin-embedded sections derived from the resection tissues of a subset of these patients using chromogenic in situ hybridization. **Results:** Plasma miR-375 levels were higher in lung NEN patients (13.89 ± 2.88) compared to patients with LUSC (8.77 ± 2.15 , $p < 0.001$), LUAD (9.31 ± 1.95 , $p < 0.001$), or NNLD (9.31 ± 1.67 , $p < 0.001$). Additionally, plasma miR-375 was elevated in lung NEN patients with distant metastases (14.51 ± 2.87) relative to lung NEN patients without distant metastases (12.32 ± 2.01 , $p = 0.03$). Analysis of resection tissues revealed miR-375 positive cells in all samples derived from patients with lung NENs. **Conclusions:** Plasma miR-375 is an emerging biomarker of lung NEN detection and staging. Further understanding of the role of miR-375 in metastasis may reveal novel therapeutic targets for lung NENs.

LAY SUMMARY

Lung neuroendocrine neoplasms (NENs) are a diverse group of cancers that often go undetected until they have spread, making treatment more difficult and outcomes worse. Diagnosing these cancers early is challenging because current tests are invasive, expensive, and sometimes unreliable. Our research explores the utility of molecule called microRNA-375 in diagnosing lung NENs through a minimally invasive blood test. We collected blood samples from 135 patients with suspected lung cancer and found that patients with lung NENs had significantly elevated concentrations of microRNA-375 relative to patients with non-NEN lung cancer or a non-cancer diagnosis. Tissue samples collected from the tumours of patients with lung NENs contained microRNA-375 whereas those with non-NEN diagnoses had no detectable microRNA-375, providing evidence that microRNA-375 is being produced in the lung NEN cells. We also found blood concentrations of microRNA-375 were significantly higher in patients with lung NENs whose cancer had spread to additional organs compared to those whose cancer was localized to their lungs. This suggests that microRNA-375 may play a role in how these cancers spread.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

N-myristoyltransferase Isoform-Specific Signatures: From Colorectal Screening to Breast Cancer Outcome Prediction - Poster #649

Presenter: Anuraag Shrivastav, University of Winnipeg/Paul Albrechtsen Research Institute, CancerCare Manitoba

ABSTRACT_ID#2790

ABSTRACT

N-myristoyltransferase (NMT) plays a critical role in immune function and tumor growth. In humans, NMT exists in two isoforms (NMT1 and NMT2). Our research has identified unique molecular signatures (UMS1-8) based on the differential localization and expression patterns of NMT1 and NMT2 in immune and tumor cells. This has led to the development of screening diagnostics for colorectal cancer (CRC) and predictive and prognostic diagnostics for breast cancer (BC). We have developed an immunohistochemical (IHC) blood test based on UMS1 for the detection of colorectal adenomatous polyps (AP) and cancer and validated its high sensitivity and specificity for AP (85% and 86%) and CRC (95.5% and 83.5%) in a prospective, blinded clinical study. In a retrospective study comprising 598 BC patients' primary tumor samples, we determined the differential expression and localization of NMT1 and NMT2 (UMS 2-8) by IHC analysis. We correlated these findings with clinical outcomes, as defined by recurrence-free survival (RFS) and overall survival (OS). UMS3 predicted a significant association with better clinical outcomes, as represented by RFS (HR = 0.70, P = 0.0304) and OS (HR = 0.71, P = 0.0306). Whereas UMS5 and UMS8 predicted worse treatment response for RFS (HR= 1.49, P = 0.0014 and HR = 1.56, P = 0.0027, respectively) and OS (HR = 1.54, P = 0.0055 and HR = 1.74, P = 0.0006, respectively). Our findings suggest that UMS2-8 are potential markers for predicting the likelihood of recurrence of BC and could become part of routine first-line prognostic and predictive tests.

LAY SUMMARY

N-myristoyltransferase (NMT) isoforms are novel cancer markers: We have identified two key proteins, NMT1 and NMT2, which exhibit distinct patterns of expression in blood cells and cancer cells. These "unique molecular signatures" (UMS) serve as distinct fingerprints that indicate the presence of cancer and its characteristics. Simple Blood Test for Colon Cancer: One of these signatures (UMS1) led to a new, blood test for detecting colorectal polyps and cancer. This simple blood test, with higher accuracy than fecal-based tests, offers a promising method for the screening of colorectal cancer. The test is highly valuable as it detects precancerous lesions, which could be removed by a follow-up colonoscopy, thus preventing CRC. Predicting Breast Cancer Outcomes: Other NMT signatures (UMS2-8) can help predict the aggressiveness of breast cancer and how patients may respond to treatment. For example, some signatures indicate a better outcome (UMS3), while others suggest a higher risk of recurrence (UMS5, UMS8), potentially informing personalized treatment plans.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Preventing Cancer in the Gender Minority Population: Preliminary Results from a Knowledge Synthesis of Existing Practice - Poster #650

Presenter: Ace Chan, University of British Columbia

Other Authors: Gabriela Kuczynski, Marina Khonina, Hannah Kia, Travis Salway, Fabio Feldman, Trevor Dummer

ABSTRACT_ID#2988

ABSTRACT

Background: Gender minority people are a part of the 2SLGBTQ+ community and are uniquely affected by stigma and discrimination that targets their gender identity and expression. Cancer prevention strategies must be strengthened in communities that experience systemic discrimination and oppression, as these factors can increase cancer risk by contributing to delayed or foregone healthcare. This scoping review aims to synthesize primary and secondary cancer prevention recommendations found in the literature for GM people. Methods: A search protocol was developed and refined in collaboration with a Community Advisory Board (CAB) and a Public Health librarian. Five databases—Medline, Embase, CINAHL, PsycINFO, and LGBTQ+ Source—were searched on August 13, 2023, using terms related to gender identity, cancer, and either cancer screening or cancer-causing substance use. Search results were imported into Covidence, a systematic review software. Results: A total of 2,660 articles were retrieved using the search terms. The analysis is currently ongoing, and preliminary findings from the title and abstract and full-text screening will be presented. Emerging themes so far include recommendations on GM-specific cancer screening intervals, GM-specific interventions targeting cancer-causing substance use, and the barriers and facilitators influencing GM cancer screening. Barriers to cancer screening include prior negative experiences with healthcare providers and a lack of provider knowledge about gender minority populations and their specific screening needs. Conclusion: GM people need consolidated primary and secondary cancer prevention strategies that are culturally tailored to them, and recommendations across prevention policies, programs, and/or activities need to be consistent.

LAY SUMMARY

We need to make cancer prevention efforts stronger in communities, such as gender minority people, that face discrimination and unfair treatment, because these experiences can lead people to delay or avoid getting the healthcare they need—putting them at higher risk for cancer. This scoping review aims to consolidate information about primary and secondary cancer prevention recommendations for gender minority people as preliminary results demonstrate that prior negative experiences with healthcare providers and a lack of provider knowledge about gender minority populations affect their screening uptake.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Machine learning algorithms and Web-Based Prognostic Tool for Ewing Sarcoma: A retrospective cohort - Poster #653

Presenter: Abdullah M Alrashidi, Royal College of Surgeons in Ireland

Other Authors: Abdullah M Alrashidi, Abdullah M Alharran, Dylan Wei Yang Stanton

ABSTRACT_ID#3119

ABSTRACT

Background and Aim: Ewing Sarcoma (ES) is an aggressive tumor with high rates of local recurrence and metastasis, which are often resistant to treatment. This study aimed to assess patient characteristics and prognostic factors using machine learning algorithms. **Methods:** We analyzed data from 726 ES patients. Descriptive and survival analyses were conducted in SPSS. Regression models (Linear Regression, LightGBM, XGBoost, Random Forest) predicted survival as a continuous outcome and were evaluated using mean absolute error (MAE), mean squared error (MSE), and R-squared (R^2). Classification models (Logistic Regression, Decision Tree, Support Vector Machine, Random Forest) predicted 1-, 3-, and 5-year survival status, evaluated by accuracy, precision, recall, and F1 score. SHAP values interpreted feature importance. Models were trained on 80% of the dataset and tested on 20%. **Results:** The Logistic Regression model achieved the highest accuracy for 1-year survival (0.80), followed by 3-year (0.62) and 5-year (0.70). LightGBM was the best regression model (MAE 2.87, MSE 13.45, R^2 0.40). SHAP analysis identified age as the most influential prognostic factor; race and marital status contributed minimally. **Conclusion:** This study demonstrates that age, tumor size, and AJCC M are key prognostic factors for ES survival, regardless of treatment received. Prospective, large-sample studies are needed to further validate these findings. **Keywords:** Ewing Sarcoma; survival prediction; machine learning; prognostic factors; SHAP values.

LAY SUMMARY

Key Insights from Our Research: We studied Ewing Sarcoma, a rare but aggressive bone cancer affecting mostly children and young adults, to understand what factors influence how long patients survive after diagnosis. Using advanced computer models called machine learning, we found that a patient's age, whether the cancer has spread (metastasis), and the size of the tumor are the most important predictors of survival. We created a simple online tool that doctors can use to estimate a patient's individual risk and better guide treatment decisions. This tool could help patients and families get more personalized information about their outlook.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Cervical Cancer Screening Using Fluorescent Interference Contrast Devices with Multiplex Capability for DNA and Protein - Poster #654

Presenter: Shuk On Annie Leung, McGill University Health Centre

Other Authors: Fatemeh Behrouz, Erica Mandato, Rus Trana, Ayyappasamy Sudalaiyadum Perumal, Dan Nicolau

ABSTRACT_ID#3286

ABSTRACT

Background: Cervical cancer is the fourth most common cancer in women globally, with 604,000 new cases and 342,000 deaths annually. As cytology (Pap test) transitions to HPV DNA testing, the latter improves sensitivity for detecting pre-cancerous lesions and enables self-sampling. However, its high false-positive rate leads to a 40% increase in colposcopy referrals, contributing to overtreatment and resource strain. Standard assays like ELISA lack the sensitivity and multiplexing capability needed to detect low-abundance biomarkers in cervical samples. Objective: To apply Fluorescence Interference Contrast (FLIC), a nanostructure-enhanced fluorescence platform that amplifies signal using silicon-on-metal substrates, improving signal-to-noise ratio by up to 4-fold, to detect protein (p16, Ki67) and HPV DNA markers (HPV-16 and HPV-18) associated with cervical dysplasia and cancer. Methods: We evaluated FLIC for detecting cervical cancer protein biomarkers (p16, Ki67) and HPV DNA markers (HPV-16 and HPV-18) using purified targets, spiked biofluids, and positive clinical samples. Results: FLIC enabled detection at concentrations of two orders of magnitude lower than ELISA for p16, demonstrating its suitability for low-biomarker settings. We also implemented automated image analysis, reducing quantification time to under 5 minutes when compared to hours of manual image analysis. DNA assay validation and protocol optimization are ongoing, with HPV 16 and 18 probes validated. Conclusions: With these four biomarkers, we aim to establish a multiplex screening panel platform. This sensitive screening approach has the potential to reduce excessive procedures and at the same time improve equity and access to screening through its potential to analyze self-collected samples.

LAY SUMMARY

- A novel nano-engineered platform using Fluorescence Interference Contrast (FLIC) technology, which amplifies signal is being used to detect protein (p16, Ki67) and HPV DNA markers (HPV-16 and HPV-18) associated with cervical dysplasia and cancer.
- We have demonstrated higher sensitivity for p16 detection compared to ELISA by 2-fold.
- This sensitive screening approach has the potential to reduce excessive procedures and at the same time improve equity and access to screening through its potential to analyze self-collected samples.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Visual Outcomes of Juxtapapillary & Juxtafoveal Biopsy in Uveal Melanoma - Poster #655

Presenter: Adam Wandzura, University of Alberta

Other Authors: Trafford Crump, Matthew Larocque, Kelsey Roelofs, Ezekiel Weis

ABSTRACT_ID#3347

ABSTRACT

Introduction Uveal melanoma is a rare tumour usually diagnosed without confirmatory biopsy; instead, clinical examination and imaging are used for diagnosis. A reason for this is the hesitancy of ocular oncologists to disturb the fovea or optic nerve, risking visual compromise. Minimal evidence supports this claim. The purpose of this study is to analyze visual outcomes of patients who underwent biopsy of uveal melanoma near the fovea or optic nerve. **Methods** A prospective consecutive case series of patients who underwent fine needle aspiration biopsy in Alberta, Canada, was conducted. Patients with tumours within 1.0mm from the fovea and optic nerve (juxtafoveal/juxtapapillary) were included. The primary outcome of interest was a clinically significant decrease in vision at 6 months, defined as a loss of 3 or more lines on the ETDRS visual acuity chart. **Results** Thirty patients were included in this study. Baseline visual acuities were a median 20/30+2 and decreased to a median 20/40+2 (range, 20/16 to 20/100) at 6 months. 6 patients (13.3%) experienced a clinically significant loss of 3 or more lines of vision at 6 months. **Conclusion** To our knowledge, this is the first Canadian study examining outcomes of juxtapapillary and juxtafoveal biopsy in uveal melanoma. Our results support the notion that biopsy near these critical structures can be performed without severe short term visual compromise. This encourages further prospective studies to ensure diagnostic and prognostic standards of care are met for this rare type of uveal melanoma.

LAY SUMMARY

1. Biopsy of all cancers is important for telling us the type of cancer, and how dangerous it is.
2. Uveal melanoma is a rare eye cancer which can be difficult to obtain a biopsy from inside the eye.
3. We show that biopsy of these cancer's can be done safely in the short term, even if located beside critical eye structures.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Inclusive cancer screening: Developing evidence-informed recommendations for breast, cervical and colorectal cancer in trans and gender diverse communities - Poster #656

Presenter: Ariana Del Bianco, Canadian Cancer Society

Other Authors: Elizabeth Holmes, Tracy Torchetti

ABSTRACT_ID#3179

ABSTRACT

Objective: There is a lot of evidence supporting the effectiveness of screening for breast, cervical and colorectal cancers at a population level. But trans and gender diverse (TGD) communities face significant barriers to care and are less likely to get screened than cisgender people. We aimed to create inclusive, evidence-informed screening recommendations for breast, cervical and colorectal cancer tailored to TGD communities to support informed decision-making and encourage early detection. **Methods:** A mixed methods approach was used. A literature review was done to gather evidence and best practices in cancer screening among TGD people. Results were synthesized to develop recommendations based on current evidence. An environmental scan was conducted to understand and apply inclusive language to the recommendations. Experts in cancer screening and care among TGD communities were consulted to review and validate the final recommendations. **Results:** We created recommendations and updated content for cancer.ca to empower TGD communities to understand their breast, cervical and colorectal cancer screening needs and take action. Up-to-date, inclusive and relevant information and guidance speaks to the realities of TGD people and promotes the importance of early detection. **Conclusion:** TGD people should get screened for cancer because the earlier cancer is found, the better the outcome and treatment options will be. CCS is continuously working to fill gaps in appropriate and tailored information for TGD people to make informed decisions about their health.

LAY SUMMARY

Research on cancer among trans and gender diverse (TGD) people is extremely limited, which makes it challenging to inform practice and address cancer disparities in these communities. TGD communities are underserved by the healthcare system and are less likely than cisgender people to be screened for breast, cervical and colorectal cancers. Cancer screening guidelines are often based on sex assigned at birth and use binary terminology that does not reflect the realities of TGD communities. Current screening guidelines for TGD populations are largely based on expert opinions. Healthcare providers and individuals should work together to determine the best screening approach for them, considering where they are in their transition, benefits and limitations, personal risk, values and preferences. Healthcare providers should be gender-affirming and try to perform the test in a way that reduces discomfort. TGD people should be screened for cancer appropriately and in a safe setting. Getting screened for cancer is an important part of regular medical care for everyone because the earlier cancer is found, the better the outcome and treatment options will be.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Colorectal Cancer Screening Uptake Among Chinese Immigrants in Canada: Exploring Associated Factors Using Structural Equation Modeling - Poster #657

Presenter: Theresa Vo, Faculty of Medicine/Memorial University of Newfoundland

Other Authors: Ying Cao, Eric Lu, Lixia Yang, Robin Urquhart, Yanqing Yi, Peizhong Peter Wang

ABSTRACT_ID#2857

ABSTRACT

Introduction Cancer screening uptake is influenced by multiple factors. We will evaluate factors impacting colorectal cancer (CRC) screening among Chinese immigrants in Canada. This study aims to (a) adapt and validate a European-developed health system literacy instrument, and (b) assess the direct, indirect, and total impact of factors, including socio-demographics, healthcare needs, and health literacy, on CRC screening uptake. Method: A cross-sectional survey was conducted from March 11th to July 19th, 2024, among Chinese immigrants aged 30 or older who has lived in Canada for at least 6 months. Of 797 respondents, 681 were eligible for analysis. For objective 1, 25 items were developed from adapting the HLS12-NAV instrument, a literature review and patient partner inputs. Content validity was assessed by 12 colleagues who are working in the healthcare system. Factor analysis and confirmatory factor analysis will be used to evaluate structural validity, along with reliability and construct validity (internal and external testing). For objective 2, we will assess health system literacy, e-health, and general health system literacy in participants aged 50 or older and use Andersen's Behavioral Model to categorize independent variables into three domains of the model, including predisposing, enabling, and need factors. A generalized structural equation modelling will be utilized to evaluate their direct and indirect impact on CRC screening uptake. Expected findings The study aims to validate an appropriate tool for measuring health system literacy and identify important pathways impacting CRC screening uptake among Chinese immigrants in Canada, which may inform potential interventions and policy changes.

LAY SUMMARY

Understanding health system literacy: Difficulties in navigating Canadian healthcare system can impact colorectal cancer (CRC) screening uptake among Chinese immigrants in Canada. Our health system literacy instrument can assist us to learn how well people can understand and use healthcare system, known as health system literacy Understanding other pieces of colorectal cancer screening: Along with understanding of the screening availability and how to get it, other factors, such as cancer behaviour, e-health literacy, health literacy, socio-demographics, having a family doctor, can interact and impact CRC screening uptake among Chinese immigrants in Canada. A comprehensive picture about associated factors on CRC screening: Using a method called structural equation modeling, we can provide strong evidence about multiple factors that directly and indirectly influence CRC screening uptake. This helps identify where future implementations or policies can fill gaps to improve CRC screening uptake among Chinese immigrants in Canada.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Nova Scotia Colon Cancer Prevention Program: Results of key screening program indicators from 2012-2023 - Poster #658

Presenter: Arlinda Ruco, St. Francis Xavier University

Other Authors: Devbani Raha, Eileen Kilfoil, Melissa Howse, Aisha Lofters, Jill Tinmouth, Michelle Murray, Robin Urquhart, Michael Stewart

ABSTRACT_ID#2464

ABSTRACT

INTRODUCTION: The Nova Scotia Colon Cancer Prevention Program (NSCCPP) was established in 2009 and became province-wide mid-2011. The purpose of this study was to report on key performance indicators of the NSCCPP since inception. **MATERIALS/METHODS:** The NSCCPP mails out approximately 185,000 fecal immunochemical test (FIT) screening kits per year to all Nova Scotians aged 50-74 years old after an even-year birthday via Canada Post. The program database along with linkage to the province's cancer registry was used to report on the following indicators: screening participation, test positivity rate, pre-colonoscopy assessment rate, diagnostic follow-up, and CRC detection rate. **RESULTS:** Screening participation has increased from 24% in 2012-2013 to 40% in 2022-2023. FIT positivity was higher among men (10%) than women (6%) in 2023. Pre-colonoscopy assessment of those requiring additional follow-up has been completed by 100% of program participants. 81% to 85% of program participants have completed a diagnostic colonoscopy through the program. The cancer detection rate over the ten-year period was 2%. **CONCLUSION:** These results provide an understanding of program performance over time and add to the literature on performance of organized colorectal cancer screening programs.

LAY SUMMARY

Colon cancer screening participation has increased over time since implementation of a provincial screening program in Nova Scotia. There remains a large proportion of individuals who are eligible for screening but do not participate.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Repeated Breast Cancer Screening Uptake Among Chinese Immigrants in Canada - Poster #659

Presenter: Theresa Vo, Faculty of Medicine/Memorial University of Newfoundland

Other Authors: Robin Urquhart, Yanqing Yi, Peizhong Peter Wang

ABSTRACT_ID#2858

ABSTRACT

Introduction Breast cancer (BC) screening enables early detection, improving treatment outcomes and survival rate. However, immigrant women have a lower screening uptake than non-immigrants. Multiple factors influence screening adherence. This study will assess repeated breast cancer screening uptake and factors impacting it. **Method** We will conduct a secondary data analysis using CanPath databases of five provinces: Alberta, Atlantic, British Columbia, Ontario, and Quebec. We include women aged 50-74 years at baseline, excluding those with self-reported breast cancer at baseline or follow-up. Screening adherence is defined as receiving a mammogram within the past 3 years. Using Yang and Hwang's framework, BC screening adherence is considered as our outcome. Independent variables regarding demographics, ethnic background, socio-economic status, lifestyle, personal medical history, family history with breast cancers will be categorized into four latent variables in the framework: 'macrostructural/contextual factor', 'predisposing factor', 'resources or enabling factors', and 'need for healthcare'. A descriptive analysis will summarize variables across five provinces. Direct standardization will compare breast cancer screening rate (with 95% confidence intervals) among ethnic women groups with different age intervals. We will choose non-immigrant group as a standard population. A mixed-effect logistic regression model will assess the influence of various factors overtime on the repeated breast cancer screening. **Expected findings** We anticipate identifying disparities in BC screening uptake among ethnic groups and determining immigrant-related factors influencing adherence to screening guidelines, offering evidence to inform equitable healthcare policy and practice. **Current status** Data access request has approved, and preliminary results will be presented at the conference.

LAY SUMMARY

Immigrant women are less likely to get regular Breast Cancer (BC) screening compared to non-immigrant women in Canada, which may delay early detection and treatment. Cultural background, income, language, healthcare access, health needs are crucial factors that impact whether women follow recommendations in BC screening guidelines. This study will help identify which ethnics group have not received BC screening as recommended and what factors are associated this decision, using CANPATH database from five provinces. Our findings can support policies and implementations to improve access and reduce disparities in BC screening among women from diverse ethnic backgrounds.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Endoscopic Angular-Diverse Optical Coherence Tomography for Early Cancer Detection - Poster #660

Presenter: Adrian Tanskanen, BC Cancer Research Institute

Other Authors: Jeanie Malone, Eric Brace, Pierre Lane, Calum MacAulay

ABSTRACT_ID#2757

ABSTRACT

Motivation: Endoscopic optical imaging enables high resolution ($\sim 30\ \mu\text{m}$) visualization at shallow depths ($\sim 1\text{-}2\ \text{mm}$), making it well suited to detect epithelial changes and stratification associated with dysplastic progression. Unfortunately, label-free optical imaging techniques suffer from low-contrast, obscuring subtle changes in tissue structures. Background: We demonstrate a novel optical imaging technology: angular-diverse optical coherence tomography (ADOCT). ADOCT generates triplicate co-registered volumetric images corresponding to different angles of tissue backscattering: brightfield, darkfield and high-angle darkfield. Dysplastic progression is characterized by changing size, shape and DNA content in epithelial nuclei, which may alter the tissues angular scattering. We hypothesize that combining the triplicate images enables access to angular backscattering as an intrinsic contrast agent. Objectives: We aim to develop and characterize the novel ADOCT system for a planned in vivo lung cancer imaging study. Three objectives are pursued towards this goal: tailor the design of ADOCT catheters for endobronchial imaging, characterize system resolution and contrast, and assess capabilities imaging ex vivo porcine lung. Methods: The catheters are fabricated to be narrow ($< 0.9\ \text{mm}$), long to reach distal airways ($\sim 2\ \text{m}$), and capable of generating up to 5 cm long volumetric images. The system's angular scattering response is characterized by imaging sub-resolution microspheres ($< 10\ \mu\text{m}$). Ex vivo porcine lung is imaged in a negative pressure chamber by inserting the catheters through the working channel of a bronchoscope to mimic clinical imaging. Impact: ADOCT provides volumetric images of whole airways angular backscattering, potentially localizing microscopic nodules, aiding earlier detection, and biopsy guidance.

LAY SUMMARY

We have developed a novel endoscopic optical imaging technology (angular-diverse optical coherence tomography) which can provide high-contrast imaging of whole airways in three-dimensions. Angular-diverse optical coherence tomography is label-free and non-ionizing, using changes in the angular backscattering distribution of light as an intrinsic contrast agent. Dysplastic progression is known to cause changes in the angular backscattering distribution of epithelial tissue. We hypothesize that this technique will be capable of detecting cancer earlier and more precisely than the current state-of-the-art. This work is preliminary, characterizing the technology and imaging lung tissue ex vivo.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Pre-treatment biopsychosocial predictors of chemotherapy-induced peripheral neuropathy trajectories in individuals with breast cancer - Poster #661

Presenter: Charles-Antoine Auger, CHU de Québec – Université Laval Research Center

Other Authors: Antoine Frasier, Maud Bouffard, Frédérique Therrien, Sarah Béland, Anne Dionne, Robert H. Dworkin, Lucia Gagliese, Jennifer S. Gewandter, Philip L. Jackson, Sophie Lauzier, Julie Lemieux, Josée Savard, Lynn R. Gauthier

ABSTRACT_ID#3111

ABSTRACT

Background: Chemotherapy-induced peripheral neuropathy (CIPN) affects 43%–80% of people undergoing taxane-based treatment for breast cancer. While some recover, others experience persistent symptoms (≥ 3 months) or delayed onset (“coasting”). Prevalence and risk factors of these trajectories remain unclear. Aims: 1) Determine CIPN persistence, recovery, and coasting prevalence in the first 3 months after taxane-based treatment; 2) Identify pre-treatment biopsychosocial predictors of these trajectories. Methods: People receiving taxane-based treatment for breast cancer (stage I–III) completed the FACT/COG-NTX-4, assessing hand/foot numbness and tingling, at baseline (T0), post-treatment (T1), and three months later (T2). A Minimally Important Difference (MID) of ≥ 3.39 from T0 defined 4 trajectories: Persistence (MID-CIPN at T1 and T2; [G1]), recovery (MID-CIPN at T1 only; [G2]), coasting (MID-CIPN at T2 only; [G3]), and no MID-CIPN at either time [G4]. At T0, participants completed self-report measures of pain, physical and psychological well-being and underwent tests of thermal, touch, and vibration sensation/pain threshold, balance, and lower limb weakness. Sociodemographic, cancer, and treatment data were collected. Results: 102 participants (aged 51.57 ± 11.24 years; 66.7% paclitaxel, 33.3% docetaxel) were included. 34.3% were G1, 25.5% G2, 6.9% G3, and 33.3% G4. Using multinomial logistic regression, compared to G4, significant predictors for G1 included age (OR=1.107; 95%CI: 1.021–1.200) and expected pain after chemotherapy (OR=1.709; 95%CI: 1.146–2.580). For G2, predictors included expected pain (OR=1.500; 95%CI: 1.007–2.234) and physical well-being (FACT-PWB; OR=0.820; 95%CI: 0.728–0.924). No predictors were identified for G3. Conclusion: CIPN trajectories are heterogeneous. Older age and greater pre-treatment expected pain may increase persistent CIPN risk.

LAY SUMMARY

Not everyone experiences neuropathy the same way. After taxane-based chemotherapy, people followed different neuropathic symptom trajectories. Some had persistent symptoms, some recovered, some developed symptoms only after treatment ended (“coasting”), and others had no neuropathy at all. Pre-treatment factors may help explain these differences. Individuals who were older or anticipated experiencing more pain before starting chemotherapy were more likely to develop persistent neuropathy symptoms, whereas those who reported feeling in better physical health before treatment had greater chances of symptom resolution within three months. These findings could help improve patient care. Identifying those at higher risk before starting chemotherapy may allow for more personalized support and follow-up, leading to earlier interventions and possibly fewer long-term side effects.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Implementing cancer patient pathways in Nova Scotia: an evidence-based and inclusive approach - Poster #663

Presenter: Caroline Hovey, Dalhousie University

Other Authors: Sarah Scruton, Cynthia Kendell, Robin Urquhart

ABSTRACT_ID#2697

ABSTRACT

Background: Recent health authority reviews on cancer-related referrals and diagnoses in Nova Scotia have resulted in concerns about current diagnostic processes and practices. Nova Scotia Health's Cancer Care Program (CCP) has embarked on efforts to improve diagnoses through the creation of standardized diagnostic pathways. In 2024, the CCP approved four diagnostic pathways (pancreas, sarcoma, colorectal, testes), with another four expected to be approved (lung, head and neck, melanoma/skin, breast) this year. However, context-specific implementation supports and strategies have not yet been delineated in the context of these pathways. Methods: Using a descriptive qualitative design, three focus groups will be held with cancer patients/families and eight with healthcare providers, for a total of 66-88 participants. Focus groups will discuss participants' diagnostic experiences and how they believe standardized diagnostic pathways and rapid-access programs can be successfully implemented. Transcripts will be thematically analyzed using qualitative descriptive methods. The resulting findings, alongside use of the Expert Recommendations for Implementing Change (ERIC), will inform the selection of appropriate implementation strategies. Results: Data collection is ongoing. We anticipate identifying factors that shape the current diagnostic process and to gain insight on how these factors will influence standardized diagnostic pathways. These data will allow us to refine pathway design and develop tailored implementation strategies. Conclusions/policy implications: This research will investigate the experiences of key stakeholders to inform decision-makers on how to optimize cancer referral and diagnosis in Nova Scotia. The implementation of standardized diagnostic pathways will result in earlier diagnoses for cancer patients, leading to improved outcomes.

LAY SUMMARY

- We are currently consulting key stakeholders, such as cancer patients, family members, and healthcare providers, to identify local factors that influence the current cancer diagnostic process in Nova Scotia. To improve our understanding of patient experience, patients/families will describe how they navigated their diagnosis and what barriers they faced in the current healthcare system. Healthcare providers will share their knowledge of the current processes and highlight barriers to timely diagnosis and referrals.
- Focus group participants will provide informed opinions on the standardized diagnostic pathways and propose how they believe the programs should be implemented. These results will guide refinement of pathway design and predict barriers of implementing standardized diagnostic pathways and rapid-access programs.
- In addition, these findings will inform development of implementation supports and strategies to assist the integration of standardized diagnostic pathways. Successful implementation of standardized diagnostic pathways, supported by this research, will improve cancer related outcomes in Nova Scotia.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Understanding barriers to mammography: insights from Arabic-speaking refugee women in New Brunswick - Poster #665

Presenter: David Busolo, University of New Brunswick

Other Authors: Tracey Rickards, Sara Edge, Robin Urquhart, Cathy Holtman, Cecilia Moreira, Mariia Zaiachuk, Khawla Aldandan

ABSTRACT_ID#3208

ABSTRACT

Background: Arabic-speaking refugee women (ASRW) experience significant inequities in mammography yet we have limited understanding of their experiences. As breast cancer remains a leading cause of cancer-related mortality among women, early detection through screening is crucial. Therefore, understanding ASRW's experiences with mammography in New Brunswick (NB) is critical for developing effective, culturally safe programs. **Methodology:** We used a qualitative research study approach to explore ASRW's experiences of mammography in NB. Forty participants took part in semi-structured, in-depth interviews in English and Arabic. Interviews were digitally recorded, transcribed, and analyzed thematically using an iterative coding process. **Findings:** Four themes emerged as barriers to mammography use. On the theme of Getting there is half the battle, women faced long wait times, limited transportation, and lack of navigators, in their quest to access screening. On the theme of Not for someone like me, culture as it relates to modesty, and preference for female providers played a central role in hindering mammography participation. On the theme of Lost in the language, ASRW faced language barriers and a lack of adequate needed information for screening. On the theme of Emotions over everything, women were hesitant to participate in mammograms because of fear of cancer, pain, and mental strain. Despite the barriers, ASRW suggested ways to improve awareness, education and access to mammography. **Discussion:** This study highlights barriers to mammography in NB and the urgent need for equity-focused, culturally safe programs. Strategies including the use of bilingual navigators, provision of gender-and culturally safe care are desired.

LAY SUMMARY

ASRW face multifaceted barriers to mammography access including structural, cultural, linguistic, and emotional barriers which limit screening participation. Participants recommended increasing access to mammography through awareness, using bilingual navigators, gender- and culturally safe care. The interventions could involve using multi-stakeholder approaches in co-developing interventions with ASRW.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Staging investigations in patients with early-stage breast cancer planned for neoadjuvant therapy: yield and predictors of metastasis - Poster #666

Presenter: Camelia Ursu, University of Calgary

Other Authors: Kadhim Taqi, Susan Isherwood, Rabia Raheel, Julia Downey, Jeffrey Q. Cao, Sasha Lupichuk, Nancy Nixon, May Lynn Quan, Alison Laws

ABSTRACT_ID#3084

ABSTRACT

Introduction: Guidelines advise against staging investigations in early breast cancer; however, it remains controversial when neoadjuvant chemotherapy (NAC) is planned. Staging prior to NAC is commonly performed regardless of stage allowing for evaluation of diagnostic yield in an unselected cohort. Methods: We used the Alberta Cancer Registry to retrospectively identify all patients who presented with AJCC 7th edition clinical stage I-II breast cancer from 2020-2022 and were referred for NAC. Primary outcome was rate of de novo metastatic (M1) disease identified on staging investigations. Logistic regression was used to evaluate factors associated with M1 disease. Results: Of 529 patients planned for NAC, 515 (97.4%) underwent staging and were included for analysis. Most (91.1%) received guideline-concordant staging (CT chest/abdomen+/-pelvis and bone scan). The overall rate of M1 disease was 5.4% (n=28, 95%CI 3.8-7.7%), and significantly differed by AJCC TN stage (p=0.04): T1N0 0%, T2N0 1.4%, T1N1 9.0%, T2N1 7.6%, T3N0 5.4%. HR+/HER2- subtype was also associated with higher rates of M1 disease, even after adjustment for AJCC stage (adjusted p=0.02). Among N0 cases, no subtype had an M1 rate >5.3%. Among N1 cases, M1 rates were: HR+/HER2- 12.6%, HR-/HER2- 5.9%, HER2+ 5.3%. Additional investigations occurred in 21.8% and were associated with a 5 day longer median time to treatment (43 vs. 38 days, p=0.02). Conclusion: Staging is not indicated for cT1-3N0 breast cancer planned for NAC due to low yield. In node-positive cases, an 8–9% metastasis rate may justify staging particularly for HR+/HER2- subtype.

LAY SUMMARY

Staging scans have little value in early breast cancer without lymph node involvement. In patients with stage T1–T3, node-negative disease, staging rarely detected cancer that had already spread, suggesting these scans can often be avoided. Lymph node-positive disease, especially HR+/HER2- subtype, carries a higher risk of undetected metastasis. Among node-positive patients, particularly those with hormone receptor-positive, HER2-negative tumors, up to 13% had metastatic disease detected—supporting the use of staging in this group.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Bridging gaps in breast cancer screening: A qualitative study on healthcare providers' perspectives on Arabic-speaking refugee women's access - Poster #667

Presenter: David Busolo, University of New Brunswick

Other Authors: Cecilia Moreira, Mariia Zaiachuk, Khawla Aldandan, Tracey Rickards, Cathy Holtmann, Robin Urquhart, Sara Edge

ABSTRACT_ID#3330

ABSTRACT

Background: Healthcare providers (HCPs) play a critical role in facilitating access to mammography for Arabic-speaking refugee women (ASRW). Despite their frontline work, limited research has examined HCPs' perspectives on mammography access for ASRW in New Brunswick (NB). Understanding these perspectives is critical for developing effective and culturally appropriate screening programs. The purpose of this study was to explore HCPs' views on mammography access for ASRW and identify opportunities for improvement. Methodology: Eight (8) HCPs from healthcare facilities across NB participated in a qualitative study using individual semi-structured interviews. The interviews were digitally recorded, transcribed, and thematically analyzed using Dedoose. Measures were taken to ensure rigour. Findings: Three themes emerged: 'It's hard to get there and get in,' 'Culture shapes care,' and 'Lost in translation and knowledge gaps.' On the theme of 'It's hard to get there and get in,' HCPs described structural and systemic barriers that limit ASRW's access to mammography. On the theme of 'Culture shapes care,' participants described cultural differences and how they influence screening behaviour and patient-provider engagement. On the theme of 'Lost in translation and knowledge gaps,' participants described how language barriers, low health literacy, and limited awareness hinder screening uptake. Discussion: The study highlights the multi-faceted challenges ASRW experience in accessing mammography services as perceived by HCPs. The findings could be foundational for developing equity-focused, culturally sensitive, and trauma-informed community interventions that address structural and cultural barriers. The collaborative work involving ASRW and HCPs could help nurture trust, eliminate disparities, and improve screening outcomes.

LAY SUMMARY

There is a gap in research examining healthcare providers' role in facilitating access to mammography for Arabic-speaking refugee women (ASRW). Healthcare providers shared logistical, cultural, language and literacy barriers to screening uptake by ASRW. There is a need for screening programs integrate cultural safety, trauma informed approaches that foster collaborative, community-based solutions.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Implementation of an integrated cervical self-screening program in rural Uganda - Poster #668

Presenter: Gina Ogilvie, BC Women's Health Research Institute

Other Authors: Candice Ruck, Priscilla Naguti, Nadia Mithani, Amy Booth, Maryam Abomoslim, Quan Hong, Hallie Dau, Joel Singer, Laurie Smith, Carol Nakisige

ABSTRACT_ID#2745

ABSTRACT

Background Despite the WHO's cervical cancer elimination target of 70% of women screened twice by age 45, <20% of women in Sub-Saharan Africa have ever been screened, thus urgent, innovative strategies are needed. Unlike cytology, HPV samples can be self-collected vaginally, improving cervix screening accessibility. This study evaluates an integrated self-screening (SS) program within community-based, primary health care in rural Uganda. Methods Women aged 30-49 across 11 villages in Malongo sub-county, with no history of screening or cervical cancer, were eligible. Village Health Teams (VHTs) conducted door-to-door recruitment. Participants completed a survey assessing demographics, health history, and past health system interactions. After instruction, participants self-collected vaginal samples, which were sent to the health centre lab, then received HPV results and treatment with Visual Inspection with Acetic Acid (VIA) and thermocoagulation at Community Health Days. Results Of 1496 women invited, 1492 (99.7%) participated in SS (median age: 36 [IQR 31-42]), of which 27.7% tested HPV-positive (n=413); 76.9% (n=318) completed VIA, of which 91.5% (n=291) received thermocoagulation. 1414 participants (94.8%) completed the survey. Most were in a relationship (n=1217, 86%) and had ≤primary education (n=1254, 88.7%). Median pregnancies were 7/woman (IQR, 5-9). A logistic mixed effects model showed that HIV-positivity at baseline was associated with a significantly increased likelihood of HPV positivity (OR: 1.98, 95%CI: 1.35-2.91). Conclusion Offering SS through VHT-led community visits was successful, demonstrated by high attendance and follow-up treatment. Findings support the need for improved access to community-integrated screening services for cervical cancer in rural Uganda to reach WHO screening target.

LAY SUMMARY

This integrated cervix screening program in Malongo sub-county, Uganda, offering HPV-based self-collection through VHTs has been highly successful and represents a viable method of advancing the WHO's screening target of 70% of women screened with a high-performance test by age 35 and again by 45 years of age. An extremely underscreened population has received screening, with readily accessible treatment when necessary. Moreover, HIV positive women, who are more likely to test positive for HPV, have received screening and treatment. Collaboration with existing community health services and community health teams is integral to feasible, accessible, and acceptable cervical screening in this very rural location.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Biocompatible Silicon Quantum Dots as Next-Generation Markers for Cancer Detection and Imaging - Poster #669

Presenter: Dorra Gargouri, Applied Quantum Materials

Other Authors: Artjima Ounkaewa, David Antoniuk, Jonathan Veinot, David Scott, Xuyang Liu

ABSTRACT_ID#2370

ABSTRACT

Silicon quantum dots (SiQDs) are nanomaterials that provide a versatile platform for enhancing cancer detection and precision diagnostics. Compared to traditional organic dyes, SiQDs offer high quantum yield, resistant to photobleaching, and tunable photoluminescence (visible to near-infrared wavelengths). While the optical properties of conventional quantum dots are also tunable and can be prepared using scalable methods, these materials comprise toxic metals which limit their use in biomedical applications. SiQDs offer tailorable surface chemistry and narrow size distributions that make them ideal for biological applications because they are heavy-metal-free. At Applied Quantum Materials Inc. (AQM), we have developed a methodology for producing high-purity SiQDs bearing customized surface chemistry, including conjugation to biomolecules and DNA aptamers (8 to 150 kDa). This presentation highlights AQM's ongoing biocompatibility studies of SiQDs that show better than 90% cell viability, even at concentrations up to 500 g/mL after 24 hours of incubation. Our studies included administration of various doses of SiQDs to determine the acute in vivo toxicity and biodistribution of functionalized SiQDs in mice. We also examined the SiQD biodistribution 24 hours and 3 days post-injection and found no residual fluorescence, suggesting effective clearance. Combined, these results highlight the low toxicity and favorable clearance profile of SiQDs, supporting their potential for biomedical applications.

LAY SUMMARY

We developed luminescent silicon quantum dots (SiQDs) with tailored surface chemistries that allow them to attach to a range of biomolecules, including DNA aptamers and proteins up to 150 kDa in size. To ensure they are safe for biomedical use, we performed thorough biocompatibility testing in both cell cultures (in vitro) and animal models (in vivo). In cell studies using HeLa, MRC-5, and HepG2 cells, the SiQDs showed low toxicity—even at high concentrations—with cell viability remaining above 90%. The SiQDs were easily taken up by cells without causing harm. Animal studies also showed no signs of organ damage or abnormalities, supporting their safety. These results suggest that acid-functionalized SiQDs are biocompatible and suitable for medical applications, though further studies are ongoing to assess long-term safety and how the SiQDs spread in the body. Together with our partners, we are using these SiQDs to develop targeted tools for detecting breast cancer cells and a liquid biopsy platform that can identify circulating tumor cells (CTCs) in blood samples. We have also created SiQDs linked to DNA aptamers that target glioblastoma, with the goal of aiding in fluorescence-guided brain tumor surgery.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Modelling MCED as a Primary Screening Option in Canada: A Simulation Study - Poster #671

Presenter: John M. Hutchinson, University of Calgary

Other Authors: Robert J Hilsden, Dylan E O'Sullivan, Yibing Ruan, Darren R Brenner

ABSTRACT_ID#2813

ABSTRACT

Introduction Improvements to cancer screening could lead to earlier detection of cancer and improved outcomes. Multi-cancer early detection testing (MCED) can detect several types of cancer through a liquid biopsy. By improving screening accessibility and participation, MCED could be an effective way to improve cancer screening outcomes in Canada. However, the potential effectiveness of population-level implementation of MCED is unknown. Microsimulation is a method to estimate the impact of MCED on cancer outcomes and system impacts. **Objectives** We aim to estimate the impact of introducing MCED as a complement to existing screening programs for breast, colorectal, lung and cervical in Canada. The primary outcomes are the number of additional cancers detected, stage at diagnosis, the reduction in mortality and the impacts on total system costs. **Methods** We will use the OncoSim microsimulation platform to estimate the effect of implementing MCED in screening colorectal, cervical, breast and lung cancers. For each of the four cancer-type specific models, we will use MCED sensitivity and specificity as input parameters based on early data from published trials and observational studies. Factors such as screening initiation age, percent of participating population and screening interval will vary across scenarios to simulate a range of possibilities. Each scenario will be compared to a “Base Case”, which represents the current state of screening in Canada. **Conclusion** The results of this study could inform the potential feasibility of MCED use in Canada. This study may provide policy insights for future evaluations of new multi-cancer screening modalities.

LAY SUMMARY

Multi-cancer early detection testing (MCED) can detect cancer in many different organ systems using a single blood test and has the potential to improve the performance of cancer screening programs. To evaluate the potential impact of MCED in Canada, we are completing a microsimulation study using OncoSim. We will model the effect of this new screening technology within existing breast, colorectal, cervical and lung cancer screening pathways. Using these simulation models, we will estimate the number of additional cancers detected, the reduction in mortality and the impacts on system costs associated with MCED.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Area-based sociodemographic disparities and cancer screening and stage at diagnosis in Ontario - Poster #672

Presenter: Jennifer Brooks, University of Toronto

Other Authors: Ashley Mah, Feifan Xiang, Maryam Darvishian, Philip Awadalla, Aisha Lofters, Victoria Kirsh, Trevor Dummer

ABSTRACT_ID#3149

ABSTRACT

Introduction The Ontario Breast Screening Program (OBSP) provides screening mammograms every two years for women aged 40 years and older. Our objective was to understand how screening adherence and cancer characteristics differed by area-based sociodemographic indicators. **Methods** All women diagnosed with breast cancer (2001-2023) aged 51-74 years were identified in the Ontario Cancer Registry. Quintiles of the 'racialized and newcomer' domain of the Ontario Marginalization index were used to characterize neighborhood-level proportions of recent immigrants and visible minority groups. Cancers were categorized as screen-detected, interval, or not screen detected and by stage at diagnosis. Standardized differences (SD) and chi-squared tests were used to compare proportions as appropriate. **Results** The sample included 120,988 screen-eligible individuals (mean age at diagnosis 62 years). A slightly higher proportion of individuals in the lowest (4.7%) versus the highest (4.2%) quintile of the Race and Newcomer Index were diagnosed with stage IV disease ($p=0.03$). These differences were most pronounced among individuals in the lowest neighbourhood income quintile (4.5% stage IV in the lowest versus 5.8% the highest) ($p=0.02$). These same individuals also had a lower proportion of screen-detected cancers. (SD=21.7%). **Conclusions** These findings highlight significant disparities in cancer screening and stage at diagnosis based on geographic socioeconomic indicators. Individuals living in areas of lower-income with a high proportion of racialized individuals exhibited lower rates of screen-detected cancers and higher rates of late-stage disease. More needs to be done to reduce these inequities in access to screening.

LAY SUMMARY

Breast cancer screening helps to identify cancer early when it is easier to treat. People living in lower income neighbourhoods with a high proportion of racialized individuals are less likely to have a breast cancer detected through screening and therefore are more likely to be diagnosed with a later stage cancer. More needs to be done to get these individuals screened to reduce disparities.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Germline-mediated immunoediting in cancer - Poster #673

Presenter: Kathleen Houlahan, McMaster University

Other Authors: Erika Apel, Alia Chaudhary, Christina Curtis

ABSTRACT_ID#3167

ABSTRACT

Cancer represents a wide spectrum of molecularly and morphologically diverse diseases. Individuals with the same histopathological classification can have tumours with drastically different molecular profiles and clinical responses to treatment. It remains unclear as to when during the disease course these differences arise and why some tumours are addicted to one oncogenic pathway over another. Somatic genomic aberrations occur within the context of an individual's germline genome, which can vary across millions of polymorphic sites. It is unknown the extent to which germline differences influence the somatic evolution of a tumour. We previously discovered that germline variants in highly expressed and amplified genes influence somatic evolution by modulating immunoediting at early stages of breast tumour development. Specifically, we demonstrated that the burden of germline-derived epitopes in recurrently amplified genes, such as ERBB2 (encoding HER2), selects against somatic gene amplification in pre-invasive and invasive breast cancer. Leveraging ~30,000 primary tumours with whole genome or exome sequencing, we extend our findings beyond breast cancer, demonstrating germline-mediated immunoediting generalizes across cancer types. A subset of tumours can overcome this immune-mediated negative selection resulting in tumours that are more aggressive, and lymphocyte depleted. Notably, signals of immunoediting are stronger in females than males, underscoring sex bias in anti-tumour immune responses. Taken together, these data indicate the germline genome plays a role in dictating somatic evolution in cancer. Exploiting germline-mediated immunoediting may inform the development of biomarkers that refine risk stratification.

LAY SUMMARY

1. A person's inherited DNA can affect which mutations in a tumour remain, by influencing how strongly the immune system attacks the tumour as it grows.
2. This phenomenon, termed germline-mediated immunoediting, is stronger in females than males.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Risk factors for developing subsequent primary colorectal cancers among adult cancer survivors: implications for targeted prevention and screening - Poster #674

Presenter: Dylan E. O'Sullivan, Cancer Care Alberta, Alberta Health Services

Other Authors: Matthew Warkentin, Robert Hilsden, Darren Brenner, Yibing Ruan

ABSTRACT_ID#3033

ABSTRACT

Background: Cancer survivors may carry elevated risks of developing subsequent primary cancers at different sites, including colorectal cancer (CRC), and may benefit from targeted screening approaches. The objective of this study was to characterize the risk and identify risk factors for developing subsequent primary CRC among survivors of different-site first primary cancers (FPC). Methods: We included all adults diagnosed with a first cancer between 2000 and 2021 recorded in the Alberta Cancer Registry and who survived at least six months. We estimated incidence rate differences and standardized incidence ratios (SIR) for CRC development compared to those without a history of cancer. For common FPC sites we conducted multivariable Fine-Gray subdistribution hazard models to identify risk factors for subsequent CRC. Results: Among 172,928 non-CRC cancer survivors, 1812 developed subsequent CRC. Compared to those without a history of cancer, cancer survivors had an elevated risk of developing CRC (SIR = 1.27, 95% CI: 1.21-1.33) with a wide variation in risk across FPC sites. Importantly, cancer survivors were also at an elevated risk of developing late stage (III/IV) CRC and the risk remained consistent during different time periods after FPC. The majority of subsequent CRC occurred among those aged 50+. Depending on the FPC site, risk factors for subsequent CRC included older age at diagnosis, male sex, overweight/obese body mass index, radiation, and chemotherapy. Conclusions: Adherence to general population CRC screening guidelines are important for cancer survivors, and targeted approaches for specific high-risk survivorship groups could be warranted

LAY SUMMARY

In this study, cancer survivors were found to be at elevated risk of developing colorectal cancer (both overall and a late stages) compared to those without a history of cancer. Risk factors for subsequent colorectal cancer included older age, male sex, overweight/obese body mass index, radiation, and chemotherapy. Adherence to general population colorectal screening guidelines are important for cancer survivors, and enhanced screening among specific high-risk survivorship groups could be warranted.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Projected cancer stage distribution and incidence in Canada - Poster #675

Presenter: Ioana Nicolau, University of Calgary

Other Authors: Kirk Graff, Yibing Ruan, Rochelle Garner, John Than, Claude Nadeau, Mary Upshall, Darren Brenner

ABSTRACT_ID#2421

ABSTRACT

Background: Cancer is a leading cause of morbidity and mortality in Canada. While cancer stage is strongly associated with survival, information on stage distribution in Canada is limited. Projecting cancer incidence by stage is critical for cancer control planning. **Objectives:** To project cancer incidence in Canada from 2025 to 2050 and project stage distribution. **Methods:** We used the OncoSim-All Cancers Model (v 3.6.5.7), a microsimulation tool supported by the Canadian Partnership Against Cancer with model development by Statistics Canada, to project health and economic outcomes for 32 cancer sites. In this study, we incorporated stage-specific projections using updated population growth trends. **Results:** In 2025, we project that the most common stage at diagnosis for the four most common cancers will be stage IV for lung cancer (51% of cases), stage I for breast cancer (45%), stage II for prostate cancer (53%), and stage III for colorectal cancer (30%). From 2025 to 2050, cancer incidence is projected to increase by 23% to 62% across cancer sites, primarily due to population growth and demographic changes. The incidence of cancer in individuals over 80 is expected to more than double during the study period, from 44,700 to 106,700 cases. Lung cancer, the most common cancer, is projected to increase from 30,200 to 43,600 yearly cases (44% increase). **Conclusions:** Here we provide the first comprehensive estimates of cancer stage in Canada across cancer types. These findings highlight the need for enhanced screening programs, resource allocation, and cost-effective treatment strategies to mitigate the projected impact.

LAY SUMMARY

Cancer is expected to rise considerably in Canada over the next 25 years, especially among older adults. By 2050, the number of new cancer cases in people aged 80 and older is projected to more than double. The stage at which cancer is diagnosed varies by cancer type and affects treatment and survival. For example, most lung cancers are diagnosed at an advanced stage (stage IV), while breast cancer is more often caught early (stage I). This study provides the first national estimates of cancer cases by stage in Canada, helping guide better health system planning. These projections can help decision-makers improve early detection, prepare for future demand on cancer care services, and ensure resources are used effectively.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Turning the Tide: Reducing Barriers to Increase Cervical Cancer Screening in Canada - Poster #676

Presenter: Lananh Nguyen, University of Toronto

Other Authors: Phillipe Descampes, Mairead O'Connor, Ody Neisingh

ABSTRACT_ID#2618

ABSTRACT

Context: Cervical cancer is a largely preventable with vaccination and regular screening. However, participation in screening programs remains suboptimal across Canada. Each province and territory have their own unique program structure, and several still only offer opportunistic screening. Most alarming is cervical cancer incidence is now increasing 3.7% per year since 2015 in Canada.¹ In fact, cervical cancer is now the fastest increasing cancer in Canadian females.¹ **Methods:** The ACCESS Consensus Group (ACCESS) is an international group of leading clinicians, researchers, patient advocates and women's representatives from Canada, Europe and the US working to accelerate cervical cancer screening participation, share best practices and ensure policy reflects the latest scientific and programmatic evidence. Their first initiative was to develop a White Paper containing recommendations to increase cervical cancer screening participation, especially among under-screened women. **Results:** ACCESS reached consensus on six recommendations: 1. Develop cervical cancer national elimination plans; 2. Implement targeted and culturally-relevant education, information and awareness-raising initiatives, with emphasis on under-screened women; 3. Improve the accessibility of cervical cancer screening; 4. Support healthcare providers (HCPs) to increase participation in cervical cancer screening; 5. Support the creation of national cervical cancer patient advocacy groups and coalitions; 6. Ensure health insurance appropriately covers screening in all high-income countries.² **Conclusions:** By implementing these recommendations, decision makers in Canada can turn the tide and reverse the increasing rate of cervical cancer in Canada. **References:** 1.Canadian Cancer Society. 2.ACCESS Consensus Group.

LAY SUMMARY

Participation in screening and vaccination programs remains suboptimal across much of Canada. Screening rates also are worse in underserved and indigenous populations who have unique challenges in accessing screening. Urgent action is needed to address low and declining participation rates and stop women dying from cervical cancer, which is now the fastest increasing cancer in Canadian females. By strengthening organized screening programs, implementing targeted and culturally-relevant education, information and awareness-raising initiatives, removing various barriers to access cervical cancer screening and advocating for increasing cervical cancer screening being offered by HCPs, we can address the low and declining screening participation rates in Canada. Although promising, ACCESS cautions against the broad use of self-sampling and raises concerns about test performance in real-world settings and implementation challenges, including potential loss to follow up after a positive self-test result. Self-sampling should be reserved as an option for under-screened women at this time and clinician-sampling should remain the preferred option for women who regularly attend screening.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Trends in the incidence of human papillomavirus-related cancers in Canada from 1992 to 2022 - Poster #677

Presenter: Ioana Nicolau, University of Calgary

Other Authors: Kirk Graff, Corinne Doll, Kristin Black, Inge MCM de Kok, Darren Brenner, Heather Bryant

ABSTRACT_ID#2928

ABSTRACT

Background: Human papillomavirus (HPV) is a well-established cause of cervical cancer and is increasingly recognized as an important risk factor for non-cervical cancers, including oropharyngeal, anal, vulvar, vaginal, and penile cancers. While cervical cancer incidence has previously declined due to screening programs, trends in non-cervical HPV-related cancers in Canada are less understood. **Objectives:** To assess temporal trends in the incidence of HPV-related cancers in Canada by sex and cancer type. **Methods:** We analyzed data from Statistics Canada for HPV-related cancers diagnosed between 1992 and 2022 in Canada (excluding Quebec and Nova Scotia). Age-standardized incidence rates were calculated, and annual percentage changes (APC) with corresponding 95% confidence intervals (95% CI) were calculated using Joinpoint regression. **Results:** Incidence rates of HPV-related cancers increased in males and females. Cervical cancer incidence increased slightly since 2013 (0.9%, 0.2- 2.9). Anal cancer incidence increased in males and females (2.6%, 0.2–10.4 since 2013; 1.9%, 1.6-2.3 since 1992, respectively). Oropharyngeal cancer incidence in males since 2012 (6.6%, 3.6-21.2) and since 1992 for females (1.6%, 0.1-3.6). Penile and female genital cancer also showed similar increases. **Conclusion:** These findings reinforce the need for HPV vaccination programs and enhance public health initiatives for the prevention and early detection of HPV-related cancers, particularly for HPV-related non-cervical cancers. It will be important to further examine the proportion of these cancers directly attributable to HPV versus other causes, to better understand the potential impact of current and future interventions.

LAY SUMMARY

Anal and oropharyngeal cancer incidence rates are increasing in Canada among both males and females. Males had higher annual percent increase in anal and oropharyngeal cancer incidence rate than females. These rising trends highlight the importance of reinforcing the importance of HPV vaccination programs in both males and females and raising awareness about the risks of HPV-related cancers.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Risk Assessment for Interval Lung Cancer Using AI and Low-Dose CT Imaging - Poster #678

Presenter: Liton Devnath, BCCRC

Other Authors: Ian Janzen, Stephen Lam, Ren Yuan, Calum MacAulay

ABSTRACT_ID#2947

ABSTRACT

Interval lung cancers (ILCs) are those that arise after a negative low-dose CT (LDCT) lung screening scan but before the next scheduled screening, typically within 1–2 years. Compared to baseline-detected cancers, ILCs are often more aggressive and diagnosed at a later, less treatable stage. We developed an AI-based tool to forecast future lung cancer risk using features extracted from annotated lesions of confirmed ILC cases and their earlier LDCT scans. The deep learning was trained using sequential follow-up scans from the PanCan study (n=26) and tested on the International Lung Screening Trial (ILST: n=10). Our aim is to calculate a patient-level risk score by examining fixed ROIs (lesion and contralateral non-lesion from opposite lung). We validated the model on follow-up scans where the ILC had yet to be detected to determine if the model was able to identify the presence of predictive alterations within area that eventually developed the lesion. For the test cases (ILST) at the individual section level the model achieved an accuracy of 0.82 and at the patient level a true positive rate of 100% for identifying ILCs in test patients at both the ILC lesion time of detection (CT0) as well as 1–2 years prior to ILC detection and 57.14% for more than 2 years prior to ILC detection. We plan to expand the non-lesion dataset areas in the lung with lesion and contralateral lung. These results demonstrate that our approach can detect sub-visual changes in the lung that predict future cancer risk.

LAY SUMMARY

AI-based tool developed to forecast future lung cancer risk using features from confirmed interval lung cancer cases and earlier LDCT scans. Deep learning trained using sequential follow-up scans from the PanCan study and tested on the International Lung Screening Trial. Model achieved an accuracy of 0.82 at the individual section level and a true positive rate of 100% at the patient level.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Colonoscopy completion after a positive stool-based colorectal cancer screening test: factors and recommendations from a global systematic scoping review of the literature - Poster #679

Presenter: Jawad Chishtie, Alberta Health Services

Other Authors: Sharon Mah, Megan Campbell, Reilly Parkinson, Muhammad Mughal, Amanda Cisar, Marcus Vaska, Gary Teare, Kamala Adhikari Dahal

ABSTRACT_ID#2798

ABSTRACT

Introduction Colorectal cancer (CRC) is globally the third most diagnosed and second leading cause of cancer death. Screening involves a stool-based tests (SBT) such as fecal immunochemical test (FIT) followed by diagnostic colonoscopy. Colonoscopy completion in Alberta following a positive FIT (FIT+) varies from 62-74% within 6 to 12 months. This scoping review synthesizes evidence on global factors and interventions, influencing colonoscopy completion after an abnormal SBT/FIT. Methods All peer-reviewed and grey literature in English till December 2023, examining patient, provider and system related factors to colonoscopy completion, from 4 major databases, 2 grey literature sources and citation chaining were included. A joint display of quantitative, qualitative and mixed methods findings was developed, employing the CFIR 2.0 Framework. Results Reported patient barriers from 146 articles and reports included: health literacy, fear, income, immigrant status, and perceived low risk; facilitators included: prior screening, higher perceived risk, and trust. Provider barriers involved: lack of knowledge, time constraints, and not trusting the SBT results; facilitators included patient counseling, referral and follow-up. Health system factors included: wait times, tracking and access for remote populations. Multi-level interventions were most effective in improving colonoscopy rates. These included patient navigation, SMS/phone reminders, provider alerts and increased colonoscopy capacity. Conclusions Improving colonoscopy completion requires multi-level, equity-focused interventions addressing patient, provider, and system-level barriers. Future research should prioritize effectiveness and qualitative studies on provider interventions and related factors.

LAY SUMMARY

Timely colonoscopy after a positive stool-based test is essential for early detection and treatment of CRC, and reducing deaths. 2. Multi-level interventions are most helpful in improving colonoscopy completion after a positive FIT. These include: Dedicated providers to help patients with or without a family physician to navigate the screening pathway Engaging primary care providers to counsel patients on the importance of preventive screening, and particularly completing colonoscopy in case of a positive FIT, reviewing results with patients and caregivers, reducing apprehensions and offering supports for completing colonoscopy Improving processes at colonoscopy clinics for reducing fear and apprehensions that patients might have Developing patient reminder systems Developing provider alerts in Electronic Health Record systems. 3. Tailored messaging and adaptable approaches for diverse populations (indigenous, immigrants, non-English speakers) are necessary for achieving health equity in CRC screening.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Concordance Between Self-Reported and Administrative Data on Cervical Cancer Screening in Alberta's Tomorrow Project - Poster #681

Presenter: Jian-Yi (Jason) Xu, Alberta Health Services

Other Authors: Sara Nejatinamini, Paige Zubrigg, Corin Kuang, Grace Shen-Tu, Jennifer Vena

ABSTRACT_ID#3038

ABSTRACT

Cervical cancer (CC) screening is essential for cancer prevention and early detection. While administrative data provide reliable records, they often lack individual-level details needed to understand screening predictors, gaps that self-reported surveys can help address. This study evaluated the level of agreement between these data sources using data from Alberta's Tomorrow Project (ATP), a prospective cohort launched in 2000 to investigate cancer-related risk factors. In 2017, ATP participants completed health and lifestyle surveys, including questions about timing of the most recent Pap test. Eligible women (age 37-70; n=17,974) were linked to records from the Alberta Cervical Cancer Screening Program (2012-2017). Agreement was evaluated using weighted Cohen's kappa and sensitivity/specificity metrics. Almost 1 in 5 participants reported screening in surveys but were not found in administrative records. The agreement was substantial for binary outcomes (screened/not screened; Gwet's AC1 = 0.762), with high sensitivity (99.9%) for detecting screened individuals. The agreement declined when categorizing by time (<6, 6-12, 12-24, 24-36, or >36 months) since last screening (kappa = 0.330). Older age was significantly associated with reporting discrepancies (p = 0.011). Data was merged by prioritizing administrative records. Screening status (up-to-date vs. not) was categorized on merged data based on provincial guidelines and predictors of screening behaviors was explored with generalized estimating equations. Rural residence (OR = 1.179) is emerging as a key predictor for not up-to-date status. Integrating administrative data with survey data can improve data accuracy, while retaining the ability to explore predictors and identify targets for CC screening promotion.

LAY SUMMARY

The Importance of Data Integration: Linking self-reported survey data with administrative health records enhances the accuracy of cervical cancer screening assessments by reducing recall bias and adjusting for confounding variables. Researchers should be cautious when relying solely on survey data or administrative data, particularly for time-sensitive measures or among older people where misreporting may be more common. **Screening Gaps and Associated Risk Factors:** A considerable proportion of eligible women were 'not up-to-date' with cervical cancer screening, highlighting a critical gap in preventive care. This group represents an important target for interventions to increase screening uptake. Lower screening rates were linked to rural residence and other factors.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Magnetic Particle Imaging Lymphography: a novel technique for lymph node mapping - Poster #682

Presenter: Nitara Fernando, Western University

Other Authors: Olivia Sehl, Benjamin Fellows, Chinmoy Saayujya, Paula Foster

ABSTRACT_ID#2736

ABSTRACT

During metastasis, tumour cells drain to nearby lymph nodes, the first of which are named the sentinel lymph node(s) (SLN). SLN biopsy (SLNB) determines if metastasis has occurred. Traditionally, SLNB involves injecting a Technetium-labeled tracer peritumourally, pre-operative imaging with SPECT to locate the SLN, and surgery guided by a gamma probe to remove them. Limitations include short tracer half-life which can make scheduling the SLNB difficult, and radiation dose to patients and healthcare workers. Alternatively, magnetic localization, with superparamagnetic iron oxide nanoparticles (SPIONs) as the tracer and a magnetometer to detect SPIONs in the SLN during surgery can be used, however, this lacks pre-operative imaging. Magnetic Particle Imaging (MPI) is a new imaging modality that directly detects SPIONs, holding potential for pre-operative imaging in magnetic SLNB. Canada's only MPI system is installed at Robarts Research Institute at Western University. To develop MPI lymphography, it is essential to determine the optimal SPIONs for SLN mapping. Tracer characteristics (size, shape, coating, sensitivity, resolution) play a role in SLN detection and are important for implementation into a clinical workflow. This study assesses the in vivo pharmacokinetics for SLN mapping with MPI in a murine model, using five commercially available SPIONs of varying iron core sizes and surface coatings. We show that Synomag-D tracers provide higher MPI signal at the SLN and show the potential to detect higher echelon nodes (HENs). Mannose and Polyethylene glycol coated tracers exhibit increased clearance from the injection site compared to plain dextran coated SPIONs.

LAY SUMMARY

A sentinel lymph node biopsy (SLNB) is a surgical procedure done to determine if cancer has spread to nearby lymph nodes, called sentinel lymph nodes (SLN). This procedure can be done by administering iron particles around the tumour, which will travel to SLNs. One limitation is that there is no pre-operative imaging done to visualize and guide the SLNB. In this study, we show that Magnetic Particle Imaging (MPI) can be used for pre-operative lymph node imaging to support and guide SLNB. MPI is a new imaging technology that detects only the iron particles, without interference from surrounding tissue. This results in clear "hot-spot" images where the particles are located. The signal strength reflects how much iron is present, making MPI quantitative. Clinical MPI is on the horizon with multiple human-scale MPI scanners under development. The very first clinical application of MPI under development is called MPI lymphography (MPIL). We tested different particles for MPIL. Particle surface coatings affect lymphatic drainage. PEG and Mannose coatings increased clearance from the injection site. With MPI, iron in nodes can be detected up to 8 days after injection, allowing for flexible surgery timing and the potential for delayed SLNB procedures.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

The Fecal Immunochemical Test for Post-Polypectomy Surveillance to Reduce Unnecessary Endoscopy (FIT2RUN) Study: Preliminary Results - Poster #683

Presenter: Reynaldo Nambayan, University of Calgary

Other Authors: Robert Hilsden, Steven Heitman, Courtney Maxwell, Andrew Schell, Jill Tinmouth, Catherine Dube, Darren Brenner

ABSTRACT_ID#2412

ABSTRACT

Introduction Surveillance colonoscopies for colorectal cancer (CRC) generate considerable demand on colonoscopy resources. Data are limited on the test performance of fecal immunochemical tests (FIT) for advanced colorectal neoplasia (ACN) detection in individuals with well-characterized index pathology and/or family history of CRC/polyps. ACN is defined as CRC or advanced adenoma/serrated polyp. This observational study aims to estimate ACN prevalence at colonoscopy among individuals undergoing post-polypectomy surveillance for personal/family history of colorectal neoplasia. Collected data will estimate test performance characteristics of the FIT as a surveillance tool in these populations. Methods Patients with a personal/family history of low/high-risk polyps and/or CRC/polyps referred for colonoscopy at the Colon Cancer Screening Centre (CCSC) in Calgary, Alberta, Canada are enrolled to the study. Participants complete a Health & Lifestyle Questionnaire (HLQ) and a FIT (15 µg Hb/g of stool positivity threshold). Data collected include demographics, FIT results, index colonoscopy findings and CRC risk factors. Results 511 participants with an average age of 61 years (SD=8.9) were enrolled. 482 (94%) of the participants had a negative FIT result, with an ACN detected in 46 (9%). Among the 29 (5.7%) participants with a positive FIT result, ACN was detected in 6 (1.2%). Preliminary FIT sensitivity and specificity are 0.12 and 0.95, respectively. Conclusion Participant recruitment will continue to facilitate powered estimates of the FIT as a post-polypectomy surveillance tool. Additional exploratory analyses will develop prediction models to guide FIT or colonoscopy use for post-polypectomy surveillance. Future study results will inform screening guidelines as colonoscopy demand increases.

LAY SUMMARY

Increased demand for colonoscopies for screening and post-polypectomy surveillance purposes signifies the importance of finding alternative methods for colonoscopy to ease clinical burden. Prediction models generated from the fecal immunochemical test (FIT) performance characteristics in detecting advanced colorectal neoplasia (ACN) among average-risk individuals can help guide the use of FIT or colonoscopy for post-polypectomy surveillance.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Understanding the Health Beliefs of Average Risk, 50–74-Year-Old Individuals Associated with Using a Fecal Immunochemical Test (FIT) for CRC Screening in Canada - Poster #684

Presenter: Patil Mkysartinian, Colorectal Cancer Canada

Other Authors: Joanne Crawford, Barry Stein

ABSTRACT_ID#3037

ABSTRACT

Early detection of colorectal cancer (CRC) is significant for better survival outcomes. Currently CRC screening rates across Canada fall below the national target of 60%, with no province or territory meeting this benchmark. This study aims to examine the current perceptions (health beliefs) related to using the Fecal Immunochemical Test (FIT) for CRC screening in Canada. This study aims to examine the current perceptions (health beliefs) related to using the Fecal Immunochemical Test (FIT) for CRC screening among average risk Canadian adults aged 50 to 74 years. A mixed method study, comprising of an online survey and one-on-one interviews, was developed to explore sociobehavioural factors (e.g., perceived benefits, perceived barriers, and cues to action) towards using a FIT for CRC screening. The online survey, available in both English and French, was launched on Colorectal Cancer Canada's social media platforms, patient partners, and community partners in April 2025. The survey is still ongoing and has collected 107 responses to date, with a goal of reaching 385 participants by September 2025. Semi-structured interviews will begin in August 2025, with plans to conduct between 9 to 17 interviews. Quantitative data will be analyzed using descriptive and inferential statistics, while qualitative data will undergo thematic analysis. The findings will help inform targeted interventions and public awareness campaigns led by CCC, and guide policymakers in developing strategies to increase CRC screening uptake, particularly among underserved populations. Ultimately, this research aims to support earlier detection and improve CRC outcomes across Canada.

LAY SUMMARY

Early detection for colorectal cancer (CRC) is important for saving lives. The five-year survival rate for CRC is 90% when found early but drops to 17% when found at later stages. Despite the availability of screening programs, many average-risk Canadians aged 50 to 74 years are not getting screened for CRC, especially equity-deserving populations such as immigrants and low-income households. Understanding health beliefs is key to increasing CRC screening participants. The study aims to identify and explore health beliefs and attitudes guided by the Health Belief Model (HBM) constructs—such as perceived susceptibility, perceived severity, perceived benefits, perceived barriers, cues to action, and self-efficacy. This research has the potential to make a significant impact on CRC participation rates in Canada by addressing key sociobehavioural factors that influence screening participation. The research findings will help create more effective CRC screening programs and public campaigns. The findings can help policymakers and screening program leaders develop targeted strategies to boost CRC screening among underserved groups and improve early detection. Colorectal Cancer Canada will also use the insights to create campaigns and resources that raise awareness and support increased screening across Canada.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Increasing team-based primary care capacity for lung cancer screening - Poster #685

Presenter: Madison Fullerton, Praxus Health

Other Authors: Kara Patterson, Chelsea D'Silva

ABSTRACT_ID#2631

ABSTRACT

Lung cancer remains the leading cause of cancer-related deaths in Canada, but early detection can significantly improve treatment outcomes and survival rates. Many jurisdictions across Canada are implementing lung cancer screening to identify lung cancer at earlier stages. Criteria for lung cancer screening varies between provinces, but all provinces require a risk assessment based on tobacco smoking history. This creates an extra demand on already burdened primary care providers and teams. Quality improvement practice facilitation builds capacity for prevention practices, including lung cancer screening, when opportunities arise in a patient interaction. Team-based practice facilitation empowers team members to champion quality improvement practices in their primary care setting. As part of this national initiative, fifteen practice facilitators from across Canada are completing specialized training focused on lung cancer screening and quality improvement methods. These facilitators are working within their primary care settings to integrate lung cancer screening processes and enhance the ability of care teams to initiate risk assessment conversations during patient encounters. Evidence-based tools are being co-developed in alignment with provincial screening program guidelines, to support primary care teams to increase their capacity to have informed conversations and conduct risk assessments for lung cancer screening. This initiative will share both quantitative and qualitative insights from the training and implementation of the practice facilitation model to explore feedback on the training, impact on lung cancer screening, and perceived impact on workflows. The refined resources will be shared with primary care teams to support the scale and spread of this initiative.

LAY SUMMARY

Early detection of lung cancer can improve treatment outcomes and survival rates. Lung cancer screening programs are available or launching across Canada. Eligibility for lung cancer screening varies among provinces, but all require a risk assessment, based on age and commercial tobacco smoking history. The risk assessment creates an extra burden on primary care teams in addition to identifying eligible patients and having informed decision making conversations. Team-based practice facilitation builds capacity for integrating lung cancer screening processes with non-physician team members. This initiative has trained 15 practice facilitators across Canada where lung cancer screening has been implemented. Evidence-based resources to identify eligible patients, conduct risk assessments and support informed conversations for lung cancer screening are being co-developed and evaluated.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Evaluating the Readability, Understandability, Actionability and Health Content of Web-Based Patient Education Materials on Cervical Cancer Screening Through an Equity Lens - Poster #686

Presenter: Petra Famiyeh, Women's College Hospital

Other Authors: Mohaddesa Khawari, Shari Dworkin, Aisha Lofters, Gina Ogilvie, Arlinda Ruco, Laurie Smith, Mandana Vahabi, Kimberly Devotta, Melanie Jeffrey, Nadia Mithani, Tara Jeji, Vijayshree Prakash, Carlee Ranger, Sundas Shamshad, Bee Lee Soh, Nila Devi, Harjeet Kau

ABSTRACT_ID#2802

ABSTRACT

Background. Cervical cancer is preventable, yet inequities in screening access and uptake persist. Public education materials are central to promoting participation but often overlook the informational and cultural needs of diverse populations. To address this gap, we are co-evaluating web-based patient education materials (PEMs) on cervical cancer screening using a health equity lens in partnership with diverse Women and Individuals with a Cervix (WIC) Methods. This patient-partnered, interdisciplinary, content analysis examines English-language PEMs focused on cervical cancer screening, HPV self-sampling, and Pap testing sourced from health authority websites across Canada. A coding framework and data extraction domains were co-developed and refined with patient partners. The framework includes three core components: readability (Flesch-Kincaid), understandability and actionability (Patient Education Materials Assessment Tool – PEMAT), and equity-oriented content aligned with Levesque et al.'s five dimensions of access. The coding schema will be systematically applied across all materials. Thematic analysis will inform recommendations for improving the clarity, inclusivity, and accessibility of education materials. **Results.** A total of 53 materials were identified from 11 provincial and territorial websites; none from Yukon or Nunavut. Most materials had a grade 7 reading level or above with only one meeting the optimal grade 6 level. PEMAT and accessibility scoring are underway. Findings will be analyzed with patient partners to co-develop evidence-informed recommendations for improving public health messaging on cervical cancer screening. **Conclusion.** Outputs of this study include the co-development of a guideline document for health agencies seeking to create public-facing resources that are inclusive and accessible.

LAY SUMMARY

Public outreach and awareness materials play a key role in cervical cancer screening participation. Reviewing the materials used to promote cervical cancer screening will help us identify if they are easy to read, understand and act on by the public. Our study findings will inform the co-creation of a public outreach messaging guideline to support public health agencies improve accessibility of their screening programs.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Development of a blood-based colorectal cancer screening test using targeted metabolomics - Poster #687

Presenter: Liam Fitzgerald, University of Calgary

Other Authors: Dennis Orton, Karen Kopciuk, Hans Vogel, Robert Hilsden, Oliver Bathe

ABSTRACT_ID#2482

ABSTRACT

Introduction. Colorectal cancer (CRC) screening reduces both disease incidence and mortality. Unfortunately, screening compliance remains low, partly due to the limitations of currently available screening modalities. A potential solution to this problem is a screening blood test based on metabolomics. **Methods.** The Biocrates MxP® Quant 500 targeted metabolomics kit was used to quantify 628 metabolites in sera from a discovery cohort of patients with CRC (n=111), adenomatous polyps (AP; n=63), serrated polyps (SP; n=62), and disease-free controls (DFC; n=154). Multivariate statistical analysis was completed to derive meta-biomarkers capable of predicting the presence and absence of CRC, AP, or SP. A large external screening set (n=838) was used to validate our signatures. **Results.** Discovery meta-biomarkers were derived for CRC, AP, and SP comprised of 31, 32, and 62 metabolites, respectively. Accuracies were >84% and receiver operating characteristic analysis demonstrated areas under the curve (AUROC) >0.85 for all three conditions based on 7-fold internal cross-validation. Performance was preserved on external validation, with AUROCs of 0.96, 0.73, and 0.72 for CRC, AP, and SP, respectively. **Conclusion.** A metabolomics-based blood test designed to screen for CRC and premalignant polyps is promising. Future studies will be directed to further refinement of the biomarkers for AP and SP to enhance specificity and positive predictive value.

LAY SUMMARY

Our metabolomics-based blood biomarkers are uniquely designed to detect premalignant polyps as well as cancer. The signature has significant clinical potential, although further studies are needed to optimize the biomarkers for premalignant polyps.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Diagnostic Accuracy of the Progesterone Challenge Test to Guide Prevention in Endometrial Cancer: A Systematic Review and Meta-Analysis - Poster #690

Presenter: Aline Talhouk, University of British Columbia

Other Authors: Rachel Woima, Derek Chiu, Elise Abi Khalil, Sabine Halabi, Andrea Neilson, Laurence Bernard, Jessica McAlpine

ABSTRACT_ID#3157

ABSTRACT

Introduction: Most endometrial cancers (EC) arise from excess unopposed estrogen. The Progesterone Challenge Test (PCT) is a short, low-dose progesterone regimen used to determine if estrogens are present in sufficient quantity to trigger endometrial proliferation, which could lead to hyperplasia and cancer. Monitoring withdrawal bleeding after the discontinued progestogen defines a positive test (+PCT), which can identify populations at high risk for EC. We systematically evaluated the diagnostic accuracy of the PCT and its utility as a non-invasive test to direct screening and risk-reducing interventions. **Methods:** We searched multiple databases for studies published before January 20, 2025, that investigated the results of the PCT compared to endometrial sampling in postmenopausal asymptomatic women. The studies were assessed for quality, and the proportion of +PCT, sensitivity, specificity, and predictive values were analyzed for two target conditions. **Results:** We identified 19 articles with 3902 participants who underwent PCT. The +PCT rate was 23% (95% CI, 13%-34%), which was not significantly different for different risk populations. The type of progestin used had a significant impact on the +PCT rate. The sensitivity and specificity were 96% (95% CI, 86%-100%) and 86% (95% CI, 74%-96%), respectively. PPV was 33% (95% CI, 16%-53%) and NPV 100% (95% CI, 100%-100%). **Conclusion/Implication:** The PCT is a sensitive and specific non-invasive test that can detect precancerous or cancerous endometrial pathology. Future work should assess this test's utility, acceptability, and cost-effectiveness on well-defined high-risk populations.

LAY SUMMARY

We systematically reviewed the literature on the Progesterone Challenge Test as a non-invasive test to direct screening and risk-reducing interventions for endometrial cancer. The Progesterone Challenge Test is both specific and sensitive in detecting endometrial pathology. The Progesterone Challenge Test could be used to help direct asymptomatic postmenopausal individuals, especially those at increased risk for endometrial cancer, to screening and risk-reducing interventions.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Examining Racial and Ethnic Disparities in Colorectal Cancer Screening Outcomes Within Nova Scotia - Poster #691

Presenter: Maria Albaba, St. Francis Xavier University

Other Authors: Melisa Howse, Devbani Raha, Eileen Kilfoil, Michael Stewart, Arlinda Ruco

ABSTRACT_ID#2836

ABSTRACT

Background: Early detection through organized screening programs like the Nova Scotia Colon Cancer Prevention Program (NSCCPP) is key to reducing colorectal cancer (CRC) incidence and mortality. However, race/ethnicity data from programmatic screening programs in Canada remain limited. **Methods:** We conducted a retrospective cohort study to examine if racial and ethnic disparities in fecal immunochemical test (FIT) positivity rates exist among individuals screened through the NSCCPP between 2021 and 2023 who self-reported their race/ethnicity. Descriptive statistics, chi-square analyses, and multivariable logistic regression were used to examine associations between FIT outcomes and demographic variables. **Results:** A total of 162,199 individuals returned a FIT kit during the study period. Of these, 70,453 were removed due to missing race/ethnicity data, and 844 were removed whose FIT result could not be processed. Among the final sample of 90,902 participants, the overall FIT positivity rate was 9.2%. Males exhibited higher positivity rates than females (11.1% vs. 7.7%). The positivity rate was 9.3% among Caucasians and 8.8% among Racialized groups, with no statistically significant difference ($p=0.08$). Logistic regression identified age and sex as significant predictors of FIT positivity, but not race/ethnicity. **Conclusion:** These findings highlight the need for further research to explore disparities by race/ethnicity and improved data collection regarding race and ethnicity.

LAY SUMMARY

Older age and being male were linked to a higher chance of a positive FIT screening result, which aligns with previous research. We did not find a significant association between race/ethnicity and FIT screening results when comparing Caucasians to all other race/ethnicities. Many people did not report their race or ethnicity, which limits our understanding of possible differences and highlights the need for better data collection.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Towards in vivo optical fallopian imaging for tubo-ovarian cancer detection - Poster #693

Presenter: Jeanie Malone, BC Cancer Research Institute

Other Authors: Adrian Tanskanen, Lien Hoang, Jessica McAlpine, Pierre Lane, Calum MacAulay, Andrea Neilson

ABSTRACT_ID#3194

ABSTRACT

MOTIVATION: Tubo-ovarian cancers are the most lethal gynecologic cancer; there are currently no effective screening measures. The most common and aggressive tubo-ovarian cancer subtype, high grade serous carcinoma (HGSC) originates in the fallopian tubes before seeding to the ovaries and peritoneal cavity. This work explores the feasibility of endoscopic fallopian imaging for the early-stage detection of tubo-ovarian cancers. **BACKGROUND:** We have previously demonstrated localization of HGSC in resected fallopian tubes with optical imaging catheters. These catheters use optical coherence tomography (OCT) to capture high-resolution (~30µm) three-dimensional imaging of the fallopian lumen. **METHODS:** A pilot study (n=40) is underway to image fallopian tubes in advanced stage tubo-ovarian cancer patients undergoing salpingo-oophorectomy. Under general anaesthesia, the imaging catheter is inserted into each fallopian tube through hysteroscopic guidance and OCT is collected. Feasibility is assessed through cannulation time/success and image quality. Diagnostic potential is assessed through comparison of imaging features with histologic findings mapped along the length of the fallopian tube. **RESULTS:** Preliminary work has demonstrated successful ex vivo cannulation of the fallopian tubes through the uterus. The mucosa is well visualized after a saline flush which separates overlapping plicae. Circumferential stratifications suggest visualization of the myosalpinx. From preclinical studies, we expect HGSC to appear more homogenous and less attenuating compared to surrounding tissue. Recruitment is anticipated to begin summer 2025. **IMPACT:** This study is the first step towards translation of an optical imaging device for the detection of tubo-ovarian cancers, which may ultimately support early detection or new care pathways for patients.

LAY SUMMARY

Ovarian cancer is one of the most lethal gynecological conditions in Canada, owing to a lack of early-stage detection methods. The most common ovarian cancers begin in the fallopian tubes, so we aim to develop tools to examine the fallopian tubes for early signs of cancer. We are conducting a trial imaging the fallopian tubes in tubo-ovarian cancer patients with a small imaging catheter immediately before surgical removal of their fallopian tubes as part of their normal treatment. From looking at resected tissue, we have demonstrated that tubo-ovarian cancers can be seen with this device but need to assess whether this can be achieved in patients to see if this approach could be used to detect early cancers.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Colorectal and breast cancer screening adherence among cancer survivors: A systematic review and meta-analysis - Poster #694

Presenter: Sanjeev Bista, University of Calgary

Other Authors: Hannah Harsanyi, Momtafin Khan, Rey Nambayan, Marcus Vaska, Yibing Ruan, Robert J. Hilsden, Darren R. Brenner, Dylan E. O'Sullivan

ABSTRACT_ID#2948

ABSTRACT

Background: Cancer survivors have been shown to be at an elevated risk of developing subsequent colorectal (CRC) and breast cancers (BC) compared to the cancer-free general population. Adherence to CRC and BC screening guidelines among cancer survivors is important to reduce the morbidity and mortality associated with subsequent cancers. **Methods:** We conducted a systematic review of the prevalence and predictors of adherence to CRC and BC screening among non-CRC and non-BC survivors. Meta-analyses were conducted for predictors that were reported in at least 3 studies. Random effects models were used to pool estimates. **Results:** A total of 62 studies were included in this review. The pooled prevalences of CRC and BC screening were 0.52 (95% CI: 0.45, 0.58) and 0.66 (95% CI: 0.61, 0.70), and the prevalence varied considerably by the type of first primary cancer. Significant predictors of adherence to CRC screening included having a health care provider, being insured, older age, and higher income and education, while significant predictors of adherence to BC screening included being instructed to participate in screening, a higher number of visits to healthcare facilities, having a cancer specialist as a care provider, having an earlier staged cancer, the presence of no comorbidities, white race, and younger age. **Conclusion:** Among cancer survivors, the overall rates of CRC and BC screening are lower than the target level for population-based screening, particularly for CRC. Factors associated with non-adherence can be used to develop programs and interventions to increase uptake and adherence to screening among cancer survivors.

LAY SUMMARY

Cancer survivors have been shown to be at an elevated risk of developing subsequent colorectal (CRC) and breast cancers (BC) compared to the cancer-free general population. Despite this elevated risk, rates of adherence to CRC and BC screening among cancer survivors is well below the target for population-based screening. Factors associated with non-adherence identified in this study can be used to develop programs and interventions to increase uptake and adherence to screening among cancer survivors.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Immune Regulatory Gene Expression Profiling of Bronchoalveolar Lavage Specimens for Distinction of Lung Adenocarcinoma from Benign Lesions - Poster #695

Presenter: Emilia Lim, The University of British Columbia

Other Authors: Mark Trinder, Peiyao Wang, Will Lockwood, Julia R Naso

ABSTRACT_ID#3079

ABSTRACT

BACKGROUND: Bronchoalveolar lavage (BAL) is a minimally invasive procedure that is commonly used to obtain a cytologic diagnosis of malignancy for patients found to have an abnormal lung nodule by medical imaging. However, these specimens often sample the peri-lesional inflammatory environment without capturing any malignant cells. We hypothesized that assessing the gene expression of this inflammatory environment could help discriminate samples as being malignant or benign and reduce the need for further invasive testing. **METHODS:** This retrospective study included BAL specimens reported as negative for definite malignant cells. After additional sampling or 2 years of imaging follow-up patients were classified as having adenocarcinoma (n=13) or a benign nodule (n=11). RNA was extracted from formalin-fixed paraffin-embedded BAL cell blocks and expression was quantified by nanoString using the nCounter® PanCancer Immune Profiling Panel (770 genes). **RESULTS:** Gene set enrichment analyses based on KEGG/GO terms found that pathways such as T-cell receptor signalling, PD-L1 expression/PD-1 checkpoint, and alpha-beta T cell activation were significantly down-regulated for adenocarcinoma cases relative to benign controls (q-values<0.05). A gene expression signature of ICAM2, C3, CARD9, TGFB2, PNMA1, and IL22RA2 was able to discriminate adenocarcinoma cases from benign controls with 100% sensitivity and 100% specificity. This finding was reproduced with 100% sensitivity and 100% specificity using an external transcriptomic microarray dataset of BAL specimens (malignant n=9; benign n=6; Kuo et al. 2018). **CONCLUSION:** Gene expression profiling of BAL specimens shows promise for distinguishing malignant from benign pulmonary lesions in limited cytology specimens where no malignant cells are identified.

LAY SUMMARY

Patients that have lung lesions identified by medical imaging are often investigated for cancer by minimally invasive procedures that sample the lesion. However, microscopic examination of these samples often only identifies inflammatory cells rather than the malignant cells needed to make a diagnosis of lung cancer. Here, we show that the expression of genes related to the immune system can help identify which samples come from patients with lung cancer versus those that come from patients with a lung lesion un-related to cancer (even when no malignant cells are identified).

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Earlier Screening, Better Outcomes? Revisiting Breast Cancer Screening Guidelines for Women in Their 40s - Poster #697

Presenter: Nguyet Ngo, University of Toronto

Other Authors: Jennifer Brooks

ABSTRACT_ID#2307

ABSTRACT

While mammography screening improves early detection and reduces mortality for individuals aged 50-74, its extension to those aged 40-49 remains debated. In Canada, screening eligibility varies by province/territory, with most programs starting at age 50. Ontario recently lowered the screening eligibility of the organized screening program to age 40 in 2024, but at the time of this study, only those aged 50 and older were eligible. Using data from Ontario Health (Cancer Care Ontario), we compared the stage at diagnosis and 5-year survival rates among 18,639 women aged 40-51 years diagnosed with breast cancer (2009-2017). Individuals aged 50 and 51 were stratified into those who had a previous screening through the provincial screening program (i.e., screeners) and those who had not (i.e., non-screeners). Individuals aged 40-49 had comparable stage at diagnosis and 5-year survival rates to unscreened individuals aged 50-51. Meanwhile, screened individuals aged 50-51 demonstrated the earliest stage at diagnosis and the highest 5-year survival rate. This suggests extending screening to women aged 40-49 may yield comparable advantages of earlier detection and survival. Our analysis demonstrates the arbitrary nature of an age-based approach (i.e., starting at age 50) to determine screening eligibility. We demonstrated that outcomes for women aged 40-49 in Ontario resemble those of unscreened women aged 50-51, who were just above the eligibility cutoff. In contrast, screened women aged 50-51 had better outcomes. This suggests that women in their 40s could benefit from the earlier detection and treatment that comes with participation in an organized screening program.

LAY SUMMARY

Mammography screening helps detect breast cancer earlier and reduce mortality rate in people aged 50-74, but it is still debated whether screening should extend to individuals aged 40-49. Breast cancer screening guidelines vary across Canadian provinces/territories, with most programs' eligibility age starting at 50. Ontario recently lowered the screening eligibility of the organized screening program to age 40 in 2024, but at the time of this study, only those aged 50 and older were eligible. Using data from Ontario Health (Cancer Care Ontario), we compared breast cancer stage at diagnosis and 5-year survival rates of 18,639 women aged 40-51 diagnosed with breast cancer. Women aged 50 and 51 were split into those who had a screening (screeners) or those who had not (non-screeners). We found that women aged 40-49 had similar stage at diagnosis and 5-year survival rates as non-screeners aged 50-51. In contrast, screeners aged 50-51 demonstrated the earliest stage at diagnosis and highest 5-year survival. Our analysis showed that relying solely on age (i.e., age 50) to determine screening eligibility may be arbitrary. These findings suggest that women in their 40s could benefit from earlier detection and treatment through participation in an organized screening program.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Brain metastases from gastrointestinal malignancies: real-world analysis of clinical and pathological features - Poster #698

Presenter: Steven Cook, Princess Margaret Cancer Centre, University Health Network

Other Authors: Yvonne Bach, Sokaina Akhtar, Chloe Gui, Elena Elimova, Gelareh Zadeh, Kevin Wang, Eric Chen

ABSTRACT_ID#3225

ABSTRACT

Background: Brain metastasis (BM) from gastrointestinal cancers (GICs) is rare, has few treatment options, and is associated with poor prognosis. We investigated clinical characteristics, treatment outcomes, and survival patterns of patients with brain metastases from GICs. **Methods:** 93 patients with BM from GICs treated at the Princess Margaret Cancer Centre between November 1997 and August 2024 were included. Clinical and pathological features were obtained through a retrospective chart review. Descriptive statistics were performed, and overall survival (OS) was estimated using the Kaplan-Meier method. **Results:** The median age was 57.2 years (range: 27.6-88.3), 55.9% were male, and colorectal cancer (CRC) patients made up 64.1%. The median time interval from GICs diagnosis to brain metastasis diagnosis was 23.0 months (range: 0 -121.5). The median time interval for CRC patients to develop BM was 43.6 months (range: 0-105), longer than those from gastroesophageal (10.0 months, range: 0-122) and other GICs (12.3 months, range: 0-51.5) ($p=0.02$). Median OS after BM diagnosis was 7.9 months (95% CI: 6.0-10.6). The site of primary cancer was not significantly associated with OS following BM diagnosis ($p=0.21$). Combination therapy with surgical resection and radiation is associated with improved OS ($p=0.006$). **Conclusions:** BM from GICs is rare but carries a poor prognosis. CRC patients had the longest latency between primary diagnosis and development of BM. Surgical resection followed by radiation provides the best outcomes for these patients. Therapies aimed at controlling BM are urgently needed.

LAY SUMMARY

Brain metastases from gastrointestinal cancers are associated with poor outcomes. In our study of 93 patients, median survival after brain metastasis diagnosis was 7.9 months. Survival after brain metastasis diagnosis did not differ significantly between patients with different primary GI cancers. Patients who received surgery followed by radiation therapy had significantly improved survival outcomes compared to surgery or radiation alone. The time to brain metastasis development differed among primary GI cancer types. Colorectal cancer patients had the longest median interval (43.6 months), compared to gastroesophageal (10.0 months) and other GI cancers (12.3 months).

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Same Profile, Different Risk: Inconsistent Categorization Across Breast Cancer Risk Assessment Tools - Poster #699

Presenter: Nguyet Ngo, University of Toronto

Other Authors: Jennifer Brooks

ABSTRACT_ID#2474

ABSTRACT

Breast cancer risk assessment tools estimate an individual's risk of developing invasive breast cancer and guide decisions on genetic testing, screening, and prevention. While many tools are validated in population cohorts, their consistency at the individual risk level is unclear. We compared seven publicly available tools: MyCancerIQ, iPrevent, BCRAT (Gail model), IBIS/Tyrer-Cuzick (MagView and Ikonopedia), Breast Cancer Surveillance Consortium (BCSC), and Cedars-Sinai. We created 36 simulated profiles varying by age at risk estimation (e.g., 43, 54, 62), breast density (heterogeneously dense or unknown), and family history (e.g., number of first-degree relatives, age at diagnosis). Using these profiles, we assessed agreement across tools in assigning risk categories (average, higher than average, high). Substantial variability in risk categorization was observed, particularly at younger ages. Of the 12 different profiles created for individuals aged 43 years, 10 (83.3%) received all three risk categories, depending on the tool used. This inconsistency decreased with age, occurring in only 4 profiles (33.3%) at age 54 and none at age 62. These discrepancies appear to stem from how tools consider risk factors. For example, IBIS models classified individuals as higher risk based on family history alone, while MyCancerIQ, iPrevent, and BCSC required both family history and dense breasts. The Gail model and Cedars-Sinai appeared to rely more on age. Depending on the tool used, the same individual could receive different risk information, corresponding to different screening recommendations. This underscores the need for tool consensus and awareness of their inconsistencies among clinicians and individuals to inform screening decisions.

LAY SUMMARY

Breast cancer risk assessment tools estimate an individual's risk of developing breast cancer and guide screening and prevention decisions. We compared seven publicly available tools: MyCancerIQ, iPrevent, BCRAT (Gail model), IBIS/Tyrer-Cuzick (MagView and Ikonopedia), Breast Cancer Surveillance Consortium (BCSC), and Cedars-Sinai. We created 36 simulated profiles varying by age at risk estimation (e.g., 43, 54, 62), breast density (unknown or heterogeneously dense), and family history scenarios (e.g., number of affected first-degree relatives, age at diagnosis). With these profiles, we examined how consistently tools assigned risk categories (average, higher-than-average, high). Results showed substantial inconsistency, especially in younger age profiles. Most 43-year-old profiles received all three risk categories depending on the tool used. Consistency improved for older profiles (ages 54 and 62). Differences appeared to be due to how the tools weighed risk factors. IBIS classified individuals as higher risk based on family history alone, while MyCancerIQ, iPrevent, and BCSC required both family history and dense breasts. The Gail model and Cedars-Sinai appeared to rely more on age. These inconsistencies may lead to different and possibly conflicting screening recommendations for the same individual. Healthcare providers and individuals should be aware of these differences when interpreting risk results and making screening decisions.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Expansion of the First Biobank in Saskatchewan, Canada - Poster #700

Presenter: Sylvie Clairefond, University of Saskatchewan

Other Authors: Laura Hopkins, Mark Milne, Mary Kinloch

ABSTRACT_ID#2629

ABSTRACT

Background: Established in October 2021, the University of Saskatchewan (USask) Biobank is the first institutional biobank in Saskatchewan, Canada. **Methods:** The biobank initially focused on collecting gynecologic oncology samples. Collection began in Saskatoon, and after six months, the same samples started to be collected in Regina. In parallel, the biobank implemented standard operating procedures and acquired the Advanced Tissue Management Application software, and received certification from Canadian Tissue Repository Networks. In fall of 2022, we adapted our consent form for a global collection of oncology samples from all adult patients. The collection of breast, gastrointestinal and bone and soft tissue samples began. In collaboration with a pediatric team, the biobank also set up a consent process dedicated to minor patients for inclusion of minor patient. **Results:** Since its opening, the USask biobank has enrolled over 360 patients, storing tissue, blood and ascites fluid samples. The biobank holds the aliquots' associated data (diagnoses, treatment and follow-up). The biobank is involved in five projects led by the USask research teams and in two pan-Canadian projects of the Terry Fox Research Institute. The biobank has also been the focus of recruitment in the pediatric space as more clinician scientists look for access to biobanks. **Conclusions:** Expanding the biobank in Saskatchewan has shown to be a significant asset for medical and biomedical research. The USASK Biobank reduces costs and administrative burdens to the researcher. The access to a biobank provides rapid access to valuable resources, promoting innovation and collaboration within the Saskatchewan scientific community.

LAY SUMMARY

Expanding the biobank in Saskatchewan has shown to be a significant asset for medical and biomedical research. Access to a biobank allows rapid access to valuable data.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

Genomic Patterns and Clinical Implications in Gastroenteropancreatic Neuroendocrine Neoplasms - Poster #702

Presenter: Gordon Taylor Moffat, Queen's University

ABSTRACT_ID#2552

ABSTRACT

Background: Gastroenteropancreatic neuroendocrine neoplasms (GEP-NENs) are a heterogeneous group of cancers with increasing global incidence. Despite this, the role of tumor genomic profiling in clinical decision-making remains unclear due to limited studies, anatomical variability, and clinical heterogeneity. Given the challenges in therapeutic development for rare cancers, there is a pressing need to investigate how genomic insights can improve patient care and outcomes. **Methods:** We conducted a multi-institutional analysis of patients diagnosed with GEP-NENs between January 1, 2017, and March 31, 2024. Genomic and clinical data were obtained from Princess Margaret Cancer Centre, AACR Project GENIE, and cBioPortal for Cancer Genomics. Duplicates were excluded. Genomic alterations were summarized descriptively, and overall survival was assessed using Kaplan-Meier methodology. **Results:** A total of 2,381 GEP-NENs were sequenced, comprising 2,149 neuroendocrine tumours (NETs) and 232 neuroendocrine carcinomas (NECs). In GEP-NETs, the most frequently altered genes included MEN1 (26%), DAXX (14%), TP53 (12%), ATRX (12%), and TSC2 (11%), with pancreatic (58%) and small bowel (17%) primaries most common. GEP-NECs showed a distinct profile: TP53 (77%), APC (69%), KRAS (30%), RB1 (24%), and BRAF (18%), predominantly from colorectal origins (95%). Among GEP-NETs, median overall survival (mOS) ranged from 73 months (MEN1, ATRX) to 20 months (TP53) ($p < 0.01$). For GEP-NECs, mOS ranged from 13 months (BRAF) to 5 months (KRAS, RB1) ($p < 0.01$). Ongoing analyses include circulating tumor DNA (ctDNA), mismatch repair (MMR), and microsatellite instability (MSI) testing. **Conclusions:** These data highlight distinct mutation and survival patterns in GEP-NENs, supporting integration of genomic profiling into personalized clinical management.

LAY SUMMARY

1. Not all neuroendocrine tumors are the same. Our study looked at over 2,300 tumors from the stomach, pancreas, intestines, and other digestive organs. We found that well-behaved (NET) and aggressive (NEC) tumors have very different genetic makeups. This means they likely behave differently and may need different treatments.
2. Some gene changes are linked to how long people live. For example, patients whose tumors had changes in the MEN1 or ATRX genes lived much longer than those with changes in TP53. In aggressive tumors, changes in BRAF or TP53 were also linked to shorter survival. This information can help doctors estimate prognosis more accurately.
3. Genetic testing can help guide personalized care. By understanding which genes are changed in a person's tumor, we can better predict how their cancer will behave and what treatments might work. Our findings support the idea that more patients with these rare tumors should have access to genetic testing.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Radiomic Signatures of Microcalcifications Predict Upgrade Risk in ADH and Low-Grade DCIS - Poster #703

Presenter: Yu Xuan (Rita) Jin, University of British Columbia

Other Authors: Fumiya Inaba, Anthony Speranza, Sylvia Lam, Anita Carraro, Zhao Yang Chen, Peter Watson, Zuzana Kos, Martial Guillaud, Alan Nichol, Behnoush Mortazavi

ABSTRACT_ID#2797

ABSTRACT

Atypical ductal hyperplasia (ADH) and low-grade ductal carcinoma in situ (DCIS) are commonly excised due to a 20–30% risk of upgrade to invasive ductal carcinoma. However, most lesions do not upgrade, leading to unnecessary surgeries. Existing mammographic assessments lack precision in stratifying upgrade risk. This study applies radiomics to identify imaging biomarkers predictive of malignant upgrade, aiming to support risk-adapted management. 40 patients from BC Cancer (2015–2024) with biopsy-confirmed ADH or low-grade DCIS were retrospectively analyzed. Radiomic features were extracted from pre-biopsy diagnostic mammograms, focusing on segmented clusters of microcalcifications. Among the 17 upgraded cases, 88% were ADH and 12% low-grade DCIS. In the 23 non-upgraded cases, 91% were ADH and 9% low-grade DCIS. In both groups, the majority of patients had a BI-RADS score of 4. No significant correlation was found between diagnosis type (ADH vs. low-grade DCIS) and upgrade risk ($p = .3$) or BI-RADS score and upgrade risk ($p = .4$), highlighting the need for improved predictive tools. These are preliminary results of a larger endeavour to develop an AI model that correlates microcalcifications and mammographic metadata with objective risk estimates. Building from this dataset, a modified convolutional neural network (CNN) will be trained on 415 patients to classify upgrade status using a 70/15/15 training, validation, and testing split with performance assessed using AUC, sensitivity, specificity, and accuracy. Our research objective to correlate radiomic features with upgrade risk aligns with the goal of active surveillance trials (LORIS, LORD, COMET, LORETTA) to reduce unnecessary surgeries by 40%.

LAY SUMMARY

Atypical ductal hyperplasia (ADH) and low-grade ductal carcinoma in situ (DCIS) carry a 20–30% risk of becoming invasive cancer. However, the majority do not upgrade into invasive cancer, resulting in unnecessary surgical operations for these patients. Our research study uses imaging data and artificial intelligence (AI) to better predict which cases are risky for upgrade into invasive cancer. Specifically, our study applied radiomics (advanced image analysis) and a specialized deep learning model to identify early patterns in diagnostic mammograms that may signal future cancer progression, using data from 40 patients with biopsy-confirmed ADH or low-grade DCIS. Preliminary statistical analysis found no significant correlation between initial diagnosis or BI-RADS scores and whether a condition upgraded to invasive cancer. These results further support the need for an AI model that can analyze mammogram features such as microcalcifications and metadata along with clinical information to better predict upgrade risk. Our model aims to distinguish between low-risk patients who may safely avoid surgery and high-risk patients who need more aggressive treatment. This supports ongoing research efforts to move toward active surveillance in breast cancer care and could reduce unnecessary surgeries by 40%.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

Early Clinical Insights into a Novel Portable Breast Imaging Device - Poster #705

Presenter: Roger Tsang, University of Calgary

Other Authors: Jeremie Bourqui, Pedram Mojabi, Bobbie-Jo Docktor, Elise Fear

ABSTRACT_ID#3075

ABSTRACT

Introduction: Mammography is the gold standard for breast imaging and has proven utility in screening programs. However, limitations include radiation exposure, decreased sensitivity in dense tissue, patient discomfort, and access in remote areas. Ultrasound and MRI imaging represent useful adjuncts but also have associated challenges. A novel way to evaluate breast tissue, with a portable imaging device using microwave signals, can be of great utility. **Methods:** We highlight 3 studies evaluating the potential of microwave imaging: 1) a repeatability study which assessed multiple scans of healthy participants at different time points, 2) a tumour detection study which scanned biopsy-proven tumours in breast cancer patients, and 3) a treatment monitoring study which evaluated serial scans of breast cancer patients receiving neoadjuvant treatment to monitor treatment response. Ethics approval was obtained for all studies. **Results:** We demonstrated evidence of feasibility, safety, tolerability and practicality of the microwave imaging device across the 3 studies. Results indicated excellent reproducibility when scanning participants at 3 different time points (intra-class correlation coefficient greater than 0.9). Comparison of CC and MLO views of the cancerous and healthy breast tissue allowed for the detection of tumours and tracking of treatment-induced changes, particularly in patients with radiographically dense tissues. **Conclusion:** Our novel portable breast imaging device demonstrates promise in the interrogation of breast tissue across clinically relevant scenarios. Further investigative and validation studies with a newer generation device with improved resolution, portability, and automated image analysis are planned, supporting the goal of clinical adoption.

LAY SUMMARY

A new approach to breast imaging that utilizes low-power microwave signals has been developed by our team. The device is designed for safety (no heating occurs), as well as for patient comfort and portability. The signals used to create the images are sensitive to the water content in the tissues. This provides the ability to characterize the composition of the breast and identify changes when comparing between breasts and at different time points. Several studies have been performed with groups of healthy participants and breast cancer patients to evaluate potential clinical applications of the technology. We have examined the consistency of scans of the same participant collected at different time points. We have also explored tumour detection, as well as the feasibility of monitoring treatment response in patients receiving chemotherapy treatments prior to their breast cancer surgery. Our initial studies demonstrated promising results and show the potential of this portable breast imaging device across different clinical scenarios. This provides significant motivation for further improvements of the device technology, and we plan for more studies to test its usefulness in clinical settings.

Screening, Early Detection, Diagnosis and Prognosis

Sunday, November 2, 2025

The Quebec Cancer Research Network (RRCancer) accelerating oncology research in the province of Quebec

- Poster #707

Presenter: Véronique Ouellet, CRCHUM

Other Authors: Anne-Marie Mes-Masson, Sonia del Rincon, Quebec Cancer Research Network RRCancer, Jean-Yves Masson

ABSTRACT_ID#3155

ABSTRACT

The Quebec Cancer Research Network (RRCancer.ca), is a collaborative network, composed of >750 members with multidisciplinary expertise working toward improving the diagnosis, prognosis, as well as care and services for cancer patients across the province of Quebec. The initial vision is to create an infrastructure of high quality biobanking to catalyze translational research in oncology in Quebec. To date, more than 50 biobanks are affiliated with the network. To support this vision and align with international best practices, the RRCancer has been a founding member of the Canadian Tissue Repository Network that provides the research community with educational material, certification processes, policies, generic standard operation procedures (SOP), and a data management system (AtiM) which provides a comprehensive solution to track all biobanking activities. The RRCancer developed harmonized SOPs and informed consent forms (ICF) to favors cohesive interoperability across institutions and allows researchers to participate in larger initiatives. Finally, the network is composed of four different axes to realize its vision • BTD : Translational Research Axis and Bank for Tissue and Data in Solid Cancers • BCLQ : Quebec Leukemic Cell Bank and Hematological Cancer Research • TE : Experimental therapy • SSAE: Learning and Equitable Health System The Quebec Cancer Research Network (RRCancer) strengthens translational cancer research in Quebec through a harmonized biobanking infrastructure, standardized tools, and four strategic axes. By fostering collaboration and aligning with international best practices, the network enhances research quality and supports improved cancer care across the province.

LAY SUMMARY

A United Front Against Cancer The Quebec Cancer Research Network brings together over 750 experts from different fields to improve cancer diagnosis, treatment, and care across Quebec. **Powerful Biobanking for Better Research** The network supports over 50 biobanks that collect and manage biological samples, helping researchers make faster and more accurate discoveries in cancer research. **Working Together for Better Results** By using shared tools, standards, and four specialized research areas, the network ensures that scientists across Quebec can collaborate effectively and contribute to global cancer research efforts.

Screening, Early Detection, Diagnosis and Prognosis

Monday, November 3, 2025

CALR overexpression is independently associated with worse outcomes in laryngeal squamous cell carcinoma - Poster #708

Presenter: Wei Shi, Princess Margaret Cancer Centre

Other Authors: Jacqueline Law, Zeynep Baskurt, Pierre-Antoine Bissey, Justin Williams, Jennifer Y.Y. Kwan, Kenneth Yip, Fei-Fei Liu

ABSTRACT_ID#3039

ABSTRACT

Introduction: Laryngeal squamous cell carcinoma (LSCC) is a common head and neck malignancy, with approximately 50% of patients relapsing within five years, largely due to chemoradiotherapy resistance. This highlights the need for reliable predictive biomarkers and effective radiosensitizers. Previous in vitro studies from our laboratory elucidated the role of calreticulin (CALR) overexpression in radioresistance. The present study aimed to investigate CALR expression in LSCC patient samples and explore its clinical significance. **Methods:** CALR expression was assessed by immunohistochemistry in 143 LSCC patients, who underwent radiotherapy. Correlations with clinicopathological parameters and patient outcomes were analyzed. **Results:** Based on median expression levels, CALR overexpression was observed in 79 of 143 (55%) LSCC samples. CALR expression did not show significant correlations with tumor subsite, stage, smoking, alcohol consumption, p16 status, or HPV infection. Kaplan-Meier survival analysis revealed that CALR overexpression was significantly associated with poorer overall survival (OS), disease-free survival (DFS) (log-rank test, $p < 0.001$), and reduced recurrence-free survival (RFS) (Gray's test, $p < 0.001$). Additionally, multivariate analysis confirmed that CALR expression served as an independent prognostic factor in LSCC, after adjusting for gender, age, tumor subsite, and stage. Finally, analyses of 475 HNSCC cases from The Cancer Genome Atlas (TCGA) database enabled stratification of patients into high or low-risk groups based on CALR expression, reinforcing its clinical relevance. **Conclusion:** Our findings show that elevated CALR expression is commonly observed in patients with LSCC, and its overexpression serves as an independent predictor of poorer clinical outcomes.

LAY SUMMARY

- **CALR Overexpression Is Common and Significant:** Over half of the laryngeal cancer patients studied showed high levels of a protein called calreticulin (CALR), which was elucidated as a radiosensitizer from our previous in vitro studies. This overexpression was associated with worse survival outcomes, including overall survival, disease-free survival, and recurrence-free survival.
- **CALR Predicts Poorer Outcomes Independently:** High CALR levels predicted worse patient outcomes regardless of other factors like age, tumor location, stage, or lifestyle habits, making it a strong, standalone biomarker.
- **Potential for Patient Risk Stratification:** Analysis of a larger cancer database confirmed that CALR can help group patients into high- or low-risk categories, supporting its use in personalizing treatment plans.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

Associations Between Symptom Complexity and Acute Care Utilization Among Advanced Cancer Patients in a Regional Cancer Centre - Poster #709

Presenter: Aynharan Sinnarajah, Lakeridge Health; Queen's University

Other Authors: Philip Pranajaya, Vince Ho, Mengzhu Jiang

ABSTRACT_ID#2395

ABSTRACT

Background: Among advanced cancer patients already accessing palliative care, high symptom scores might lead to increased acute care utilization and poor patient outcomes. This study examines how a recently developed symptom complexity algorithm, which uses the 9 Edmonton System Assessment System–Revised (ESAS-r) symptoms to assign "low", "medium" or "high" complexity, is associated with acute care utilizations. Patients and methods: This was a retrospective observational cohort study of advanced cancer patients accessing palliative care services at a Regional Cancer Centre, and had completed at least one ESAS-r report between January 1, 2022–December 31, 2023. Chi-squared, Kruskal-Wallis tests, and multivariable binary logistic regressions were used to evaluate factors associated with odds of acute care utilization within 1 and 2 weeks of the patient's first symptom report. Results: Of the 559 patients, 22.4% (n=125) exhibited low symptom complexity, 32.2% (n=180) of patients exhibited medium symptom complexity, and 45.4% (n=254) exhibited high symptom complexity. 10.9% (n=61) of patients accessed acute care within 1 week and 19.3% (n=108) of patients within 2 weeks of their ESAS-r report. Multivariable analysis showed that, compared to low-complexity patients, high-complexity patients had significantly higher odds of 1-week acute care utilization (OR=2.83, 95%CI=1.18–6.77), but not within 2 weeks (OR=1.78, 95%CI=0.97–3.28). Conclusions: Patients with higher symptom complexity were more likely to access acute care within 1 week of their first ESAS-r report. These patients may benefit from more intensive palliative care follow-up and could be triaged using this symptom complexity tool, to provide more timely care.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Rural Urban Divide in Access to Palliative Care Services among Cancer Patients in Alberta, Canada - Poster #710

Presenter: Aynharan Sinnarajah, Lakeridge Health; Queen's University

Other Authors: Omar Abdel-Rahman, Hossein Farahanchi, Beverly Berg, Michelle Peterson Fraser, Pin Cai, Sharon Watanabe, Cara Bablitz, Anna Santos Salas, Simon Colgan, Jessica Simon

ABSTRACT_ID#2396

ABSTRACT

Objective: To assess the presence and extent of disparities in accessing palliative care (PC) services among cancer patients in Alberta, with a specific focus on the divide between patients living in rural areas versus those living in urban areas. **Methods:** This was a retrospective cohort study (based on administrative databases) of cancer decedents in Alberta, who died between 1 January 2013 and 31 December 2021. The outcome of interest is receipt of any form of a specialist PC service prior to death. A frequency analysis compared baseline sociodemographic factors between patients who received PC services vs those who did not receive it. A multivariable logistic regression model was conducted to assess for factors associated with lower access to PC services. **Results:** A total of 59076 cancer patients were identified and included in the current analysis. Patients who did not receive PC were more likely older, men, with high material and social deprivation, living in rural areas, and were treated in a regional cancer centre (as compared to an academic centre) ($P < 0.001$ for all comparisons). In a multivariable logistic regression model, adjusted for relevant sociodemographic confounders, patients living in rural areas had lower access to specialist PC compared to those living in urban areas (OR: 0.31; 95% CI 0.29-0.33). **Conclusion:** There are several disparities in accessing specialist PC services for cancer patients in Alberta along the lines of sociodemographic and geographic factors. Rural cancer patients continue to struggle with lack of access to quality PC services compared to urban cancer patients.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Mixed-Methods Exploration of Stakeholder Perspectives Toward Psychedelic-Assisted Therapies for Cancer-Related Psychosocial Symptoms - Poster #712

Presenter: Haley Schuman, University of Calgary

Other Authors: Linda Carlson

ABSTRACT_ID#3262

ABSTRACT

Background: Psychedelic-assisted therapy (PAT) is a promising intervention for cancer-related psychosocial distress, but its integration into care remains limited by implementation and policy gaps. Methods: A national cross-sectional survey (N = 742) and two qualitative interview studies were conducted to examine stakeholder perspectives on PAT. Survey respondents included PLWC (n = 519), healthcare providers (HCPs; n = 187), and policymakers (PMs; n = 36). The survey assessed attitudes (APQ), perceived knowledge, safety and effectiveness beliefs, and interest in training/access. Between-group comparisons were conducted using non-parametric and regression-based analyses. Semi-structured interviews with PLWC and HCPs further explored implementation barriers, motivations, and ethical considerations, analyzed using reflexive thematic analysis grounded in the Theory of Planned Behaviour. Results: PLWC endorsed the most favorable attitudes toward PAT, significantly higher than HCPs and PMs ($p < .001$), despite reporting the lowest perceived knowledge. PMs expressed the greatest safety concerns and preferred restricting PAT to advanced-stage cases. Psilocybin was perceived as the most effective agent across groups. Key implementation barriers varied: PLWC prioritized financial and provider access, while HCPs and PMs emphasized training, standardization, and evidence. Regression analysis showed that stakeholder group and prior psychedelic experience were positively associated with favorable attitudes, whereas higher self-rated knowledge predicted lower attitude scores ($p < .001$). Interviews echoed these patterns and highlighted ethical tensions around access and regulation. Conclusion: Despite promising receptivity, substantial implementation barriers persist. Addressing safety, training, and policy concerns—while centering stakeholder-informed solutions—is essential for ethical and equitable PAT integration into oncology care.

LAY SUMMARY

1. PLWC are open to psychedelic-assisted therapy (PAT) as a way to address emotional, spiritual, and existential suffering, particularly after treatment or in advanced illness. In our national survey, PLWC showed the most positive attitudes toward PAT, despite reporting the lowest perceived knowledge. Interestingly, those who rated themselves as more knowledgeable were sometimes less supportive of PAT, highlighting the need for accurate, balanced education. Many PLWC (92.8%) supported regional access, viewing PAT as a meaningful tool for coping with fear, loss of meaning, and suffering that conventional care often overlooks.
2. HCPs and policymakers are more cautious, expressing concerns about safety, training, legal uncertainty, and institutional readiness. Providers highlighted the need for clearer guidelines and more robust clinical evidence, especially as patient interest increases. Attitudes toward PAT differed across groups—patients were the most supportive, while providers and decision-makers were more hesitant due to systemic and professional constraints.
3. Interviews revealed complex, substance-specific views. Ketamine was valued for its rapid effects but critiqued for commercialization and dependency risks. Psilocybin sparked polarized views—some saw it as promising for existential distress, while others worried about destabilization. MDMA was viewed as promising for trauma but hindered by concerns about serotonin depletion and regulatory hurdles.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

Towards a Scalable Health System Approach to Advance Serious Illness Communication Training and Implementation - Poster #713

Presenter: Justin Sanders, McGill University

Other Authors: Sonia Skamene, Harvey Chang, Charles-Olivier Basile, Kajamathy Subramaniam, Ting Du, Tibor Schuster, Erik Fromme

ABSTRACT_ID#3306

ABSTRACT

Background Serious illness conversations (SIC) promote prognostic awareness and align care with patients' goals and values, improving patients' well-being. The Serious Illness Conversation Guide (SICG) is a structured tool used to train clinicians via in-person training. Instructors' and trainees' time commitment and scheduling logistics pose training challenges. This pilot RCT assesses whether a digital training delivery using desktop Virtual Reality (VR) presents a viable alternative to deliver SICG training. **Objectives** This pilot RCT aims to assess the feasibility and preliminary effectiveness of VR training compared to in-person training, to guide planning of a future larger non-inferiority RCT and to identify factors that predict/inhibit uptake and documentation of SIC among cancer clinicians. **Approach** Quantitative and qualitative data are analyzed from the training of 48 oncology physicians through in-person or VR sessions, using a convergent mixed-methods approach. **Outcome measures** including study enrollment and completion rates, training adherence and acceptability rates and time required for data collection. **Other study outcomes** include pre-post reports of clinicians' skills and confidence, training satisfaction score, presence of documentation of SIC following the training, and quality of documentation. Finally, clinician will be interviewed to elicit their perspectives on the facilitators, barriers, and outcomes related to SIC. **Interpretive description** is used as a methodology to generate knowledge useful for practice. **Anticipated Findings** We hypothesize that VR training is feasible and accepted. This study may provide an unprecedented opportunity to upscale communication skills training using VR as an effective alternative, meeting the needs of busy clinicians in diverse practice settings.

LAY SUMMARY

1. Attendants will be able to evaluate the feasibility and effectiveness of Virtual Reality compared to in-person training for teaching the Serious Illness Conversation Guide (SICG) to oncology clinicians.
2. Attendants will be able to recognize clinicians' satisfaction and confidence following SICG training via Virtual Reality or in-person format.
3. Attendants will be able to describe factors that facilitate or inhibit the uptake and documentation of serious illness conversations among oncology clinicians.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

A hybrid effectiveness-implementation study of the Managing Cancer and Living Meaningfully intervention across established psychosocial oncology and palliative care programs in Alberta's Calgary and South Zone -

Poster #714

Presenter: Janet de Groot, Arthur JE Child Comprehensive Cancer Centre

Other Authors: Andrea Feldstain, Jessame Gamboa, Fay Strohschein

ABSTRACT_ID#3326

ABSTRACT

Background: The brief evidence-based Managing Cancer and Living Meaningfully (CALM) psychotherapy effectively addresses common psychosocial concerns among people with advanced non-curative cancers across cultures. Implementation strategies are required to speed uptake of psychotherapies, such as CALM, to address clinical needs. **Aims:** The study aimed to develop and compare implementation referral strategies for CALM, assess the feasibility of training clinicians for CALM competence and assess patient adherence and satisfaction with CALM. Patient-reported outcomes were included to confirm effectiveness. **Methods:** A pragmatic single-arm Hybrid Type 3 effectiveness-implementation study with concurrent, mixed-methods design was used to evaluate implementation outcomes of referral strategies, while collecting clinical effectiveness data for CALM. The Consolidated Framework for Implementation Research framework informed interviews to obtain clinician and community cancer care organization leaders' perspectives on barriers and facilitators to CALM referrals. **Results:** Clinicians resonated with the CALM model, conveyed receptivity to change for beneficial interventions and valued a CALM coordinator in facilitating referrals with clinician and patient information. Patients referred to CALM through the psychosocial oncology program, after distress screening, had higher attrition ($X^2 = 6.3$, $p = .01$) than patients directly referred by frontline clinicians. Training multidisciplinary clinicians to CALM competence was limited by attrition. **Conclusions:** CALM implementation was feasible with new referral routes, facilitated by a CALM Coordinator and information resources, across established PSO and palliative care programs. Training early career clinicians may support integration of CALM into clinical practice and support clinical needs of patients with advanced cancer.

LAY SUMMARY

Direct referrals by front-line oncology and palliative care clinicians of patients with advanced cancer to Managing Cancer and Living Meaningfully (CALM) therapy are associated with less attrition than referrals through psychosocial oncology, based on distress screening. Within three months of beginning CALM, patients with advanced cancer report reduced depression and enhanced quality of life. Training of multidisciplinary early career multidisciplinary clinicians in professions regulated to provide therapy may support integration of CALM into the care of patients with advanced cancer

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

The Nature-Connected Oncology Research Exploration (N-CORE) Program: Exploring Nature-based Interventions in People Living with and Beyond Cancer - Poster #715

Presenter: Jamie Petersson, University of Calgary

Other Authors: Julie Deleemans, Raylene Reimer, Linda Carlson

ABSTRACT_ID#2615

ABSTRACT

Background: Increasing survivorship has led to a rise in chronic post-treatment gastrointestinal (GI) and psychosocial symptoms. Investigating interventions to improve these cancer-related outcomes is critical, and nature-based interventions (NBIs) have shown potential. This remains unstudied in people living with cancer (PLWC). The Nature-Connected Oncology Research Exploration (N-CORE) program seeks to review NBI literature in oncology, assess stakeholder attitudes, and explore the relationship between nature connectedness (NC) and GI and psychosocial health in PLWC. Methods: N-CORE includes three studies: a systematic literature review, a survey, and a pilot study. The review will explore NBI literature in oncology using PRISMA methodology. An online survey will collect demographics, clinical and lifestyle information, NC, and GI and psychosocial outcomes from a target of 385 PLWC, 100 caregivers, 50 healthcare providers, and 20 policymakers. Finally, a multi-week pilot study informed by the review and survey will assess the feasibility of an NBI, measuring gut microbiota, NC, and symptom outcomes pre- and post-intervention. Results: The systematic review will critically appraise NBI effects on cancer outcomes and reveal gaps in the literature. The survey will unveil interest, attitudes, barriers, and associations between NC and health symptoms using descriptive and inferential statistics in key groups of stakeholders. The pilot study will evaluate feasibility using accrual, retention, and data completion rates and determine preliminary effect sizes on key outcomes. Conclusion: N-CORE will identify literature gaps, stakeholder interest, and the feasibility of NBIs for cancer care, advancing knowledge on the role of NC in improving cancer-related symptoms among PLWC.

LAY SUMMARY

1. Reveal the gaps in the literature on NBIs in oncology.
2. Determine stakeholder interest surrounding NBIs in cancer care.
3. Investigate the feasibility of an accessible, cost-effective nature program for addressing psychosocial symptoms and balancing the gut microbiota.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Multiphasic and Multimodal Exercise Prehabilitation in Allogeneic Hematopoietic Stem Cell Transplantation: Feasibility to Date - Poster #716

Presenter: Julia Daun, University of Calgary

Other Authors: Jocelyn Cannon, Heejae Lee, Jessica Danyluk, Tanya Williamson, David M. Langelier, George J. Francis, Chad W. Wagoner, Sarah Perry, S. Nicole Culos-Reed

ABSTRACT_ID#3078

ABSTRACT

Background: Exercise prehabilitation is an evidence-based intervention for individuals living with and beyond cancer, yet limited research has examined it in those with acute leukemia likely to undergo allogeneic hematopoietic stem cell transplantation (allo-HSCT). This study evaluates the feasibility of delivering a tailored, multiphasic, multimodal exercise prehabilitation intervention in this underserved and medically complex population – the Alberta Cancer Exercise-Stem (ACE-Stem) study. **Methods:** ACE-Stem is a single-arm feasibility study in Calgary, Canada. Adults >18 years, diagnosed with acute leukemia, and with a high probability of proceeding to allo-HSCT are referred by their clinical team. Participants receive one-on-one exercise and health coaching sessions across pre-transplant (4-12 weeks), transplant (4-6 weeks), and post-transplant (12 weeks) phases. Sessions are tailored and delivered weekly by Clinical Exercise Physiologists in-person or remotely. Feasibility measures include tracking number of referrals received, enrollment and intervention completion proportions, adverse events, and participant satisfaction (via survey). **Results:** Recruitment, delivery, and data collection are ongoing. Since October 2022, n=44 patients have been referred to ACE-Stem and n=29 have enrolled (66% enrollment). Of those enrolled, n=9 have completed the intervention and n=11 have dropped out, resulting in 45% completion to date. N=9 are currently participating. No adverse events have occurred. Participant satisfaction has been high (89%). **Conclusion:** While the intervention has demonstrated good enrollment, safety, and been highly rated by participants, completion has posed a challenge. This preliminary feasibility work will inform a future fully powered trial to refine the delivery of tailored exercise prehabilitation for individuals with acute leukemia undergoing allo-HSCT.

LAY SUMMARY

- **IDENTIFYING A GAP IN CARE:** stem cell transplants are physically and emotionally demanding for individuals with acute leukemia. Exercise before treatment may help but has not been well-studied in this patient group.
- **OUR APPROACH TO CLOSE THE GAP:** we are providing a personalized exercise and health coaching program tailored around to the transplant phases. We track enrollment, intervention completion, safety, and participant satisfaction.
- **EARLY RESULTS:** since October 2022, 44 people were referred and 29 have enrolled (66%). No safety issues have occurred, and participants report high satisfaction, but keeping people in the program has been a challenge. These insights guide program improvements and future research.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

The Teachable Moment: Barriers to physical activity discussion in clinical care with non-curative cancer patient - Poster #717

Presenter: Jodi Langley, Dalhousie University

Other Authors: Grace Warner, Christine Cassidy, Robin Urquhart, Melanie Keats

ABSTRACT_ID#2408

ABSTRACT

Non-curative cancer patients warrant specific attention due to the nature and progression of disease. Evidence suggests that physical activity (PA) provides therapeutic benefits beyond general health. However, Oncology Care Providers (OCPs) could better use teachable moments during clinical visits to enhance patient receptivity to PA. Objective: This study aimed to explore the perspectives of patients, family/friend caregivers, OCPs, and health decision-makers on PA, and examine barriers to PA-related conversations in clinical settings. Methods: We conducted 24 semi-structured interviews guided by the Capability, Opportunity, Motivation-Behaviour (COM-B) model. Data were analyzed using directed content analysis, with a codebook informed by the COM-B framework. Results: Participants reported shared barriers in psychological capability, physical opportunity, and reflective motivation. A key barrier was limited knowledge about PA among both providers and patients, highlighting the need for targeted education to support informed decision-making and behavior change. While there was a clear desire for more PA information, participants also recognized system-level constraints that limit access to such knowledge and support. Conclusion: This study provides insight into how PA discussions can be better integrated into care for individuals living with non-curative cancer. Findings lay the groundwork for fostering more supportive and timely conversations about PA in oncology care. Clinical Implications: Clinicians should be equipped with the knowledge, resources, and confidence to initiate PA conversations throughout the cancer care continuum. Strengthening system-level supports and referral pathways will further enable the integration of PA into standard oncology care.

LAY SUMMARY

1. People with non-curative cancer want to be more active, but face barriers. Patients, family/ friend caregivers, oncology care providers and health decision makers all saw the value of physical activity, but many did not have enough information or support to make it part of clinical care discussion.
2. Oncology care providers often miss opportunities to talk about physical activity and many providers didn't feel confident starting conversations about physical activity. This, in addition to the lack of training around the knowledge missing made it harder to discuss physical activity during appointments.
3. Better education and system support are needed to help support oncology care providers and patients. To make physical activity a regular part of cancer care, oncology care providers need more training and clear pathways to refer patients to appropriate programs or resources.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

The VISTA Program: A Telepsychiatry Innovation Advancing Equitable Psychiatric Care for Cancer Patients - Poster #718

Presenter: Meaghan Wright, Ontario Health

Other Authors: Christian Schulz-Quach, Apurva Shirodkar, Catherine Chan, Ying Jiang, Laura Pazzano, Colleen Fox

ABSTRACT_ID#2869

ABSTRACT

Equitable access to medical psychiatry in cancer care remains a health systems challenge in Canada. The Virtual Psychiatry Assessment (VISTA) Program is a novel telepsychiatry initiative developed to address these persistent disparities for oncology patients and their care partners in rural and remote Ontario. Piloted from December 2024 to March 2025, the program integrated virtual psychiatric consultations into existing psychosocial models through a partnership between Ontario Health, Carepath (Bayshore HealthCare Ltd.), the Canadian Partnership Against Cancer (CPAC), and the University Health Network. The interim evaluation employed a mixed-methods approach, including quantitative tracking of service delivery metrics and qualitative data from stakeholder interviews and survey feedback. Of 17 patients enrolled as of March 2025, 14 completed the care pathway and received comprehensive psychiatric management plans, with median turnaround times of 22 days from referral to plan delivery. Patients reported high satisfaction with clinician engagement, clarity of next steps, and technological accessibility. Key enablers included team-based care models, stepped-care approach, pre-assessment efficiency, and centralized administrative support. Identified barriers included provider hesitancy to assume responsibility for psychiatric post-consultation management, referral restrictions tied to Ontario Health Insurance Plan (OHIP) billing codes, and communication gaps across care teams. The VISTA model demonstrates feasibility and early efficacy in improving access in psychosocial oncology, with implications for broader telepsychiatry implementation. These preliminary findings suggest the importance of aligning clinical, administrative, and technological components to enable scalability. Final evaluation results, including linked administrative health data and expanded patient/provider feedback, will be available in Fall 2025.

LAY SUMMARY

- Virtual psychiatry fills a critical care gap: Cancer patients and their caregivers in rural and remote areas often lack access to mental health support. The VISTA Program showed that virtual psychiatric care can provide timely, effective help—often within just two weeks—improving patients’ well-being during treatment.
- Team-based virtual care works well: The program’s success relied on close collaboration between psychiatrists, mental health clinicians, and administrative staff. Clear communication, structured team meetings, and shared responsibilities helped deliver care efficiently and support patients through a complex phase of their health journey.
- Barriers need to be addressed for long-term success: Some healthcare providers were hesitant to refer patients due to concerns about workload and billing requirements. To expand this program, it's important to make referrals easier, raise awareness among oncology teams, and support coordinated care between cancer specialists and family doctors.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

Transdiagnostic Interventions for Mental and Physical Health Symptoms in Oncology Populations Across the Lifespan: A Scoping Review - Poster #719

Presenter: Jorja Spratt, University of Calgary

Other Authors: Marin Hoh, Caitlin McClurg, Nicole Alberts, Joanna Fardell, Gregory Guilcher, Lindsay Jibb, Melanie Noel, Quynh Pham, Vinesha Ramasamy, Kathleen Reynolds, Fiona Schulte, Maya Stern, Jennifer Stinson, Perri Tutelman

ABSTRACT_ID#3052

ABSTRACT

Background: Individuals diagnosed with cancer commonly experience various physical (e.g., pain, fatigue, insomnia) and mental health (e.g., anxiety, depression) symptoms across the illness trajectory. Transdiagnostic interventions - treatments that target comorbid symptoms - may offer a more effective and efficient approach to address symptoms. However, little is known about their application in oncology. The aim of this scoping review is to map the extent and nature of empirical research on transdiagnostic interventions in oncology populations. **Methods:** This study follows Arksey & O'Malley's methodological framework for scoping reviews. Comprehensive searches were conducted in 5 scientific databases including: Medline, Embase, PsycINFO, Scopus and CINAHL. Articles eligible for inclusion were empirical studies published in English in peer-reviewed journals that examined interventions addressing two or more mental and/or physical symptoms in oncology patients or survivors of any age. Excluded articles were systematic or scoping reviews, case reports, commentaries, dissertations, conference abstracts, books, and protocols that do not include a psychology-based intervention. Full results will be presented. **Results:** The search yielded 10,620 studies. After the removal of duplicates, 6,970 unique studies were retained for screening. Following title and abstract and full text screening, these studies will be summarized descriptively, focusing on each study: study population, intervention used, symptoms targeted, and outcomes reported. **Discussion:** There is growing interest in transdiagnostic interventions for oncology populations. This review describes the current state of the field and will inform directions for future research, including the development of novel transdiagnostic interventions for individuals living with and beyond cancer.

LAY SUMMARY

Many people diagnosed with cancer experience mental (i.e., anxiety and depression) and physical health symptoms (i.e., troubles with sleep, pain, and fatigue) that often occur together and can make each other worse. Transdiagnostic interventions (interventions that address more than one symptom at a time) can help people manage symptoms in a way that is easier and more effective, but little is known about their use for people with cancer. The goal of this study is to summarize the current research on transdiagnostic interventions for people diagnosed with cancer. A search of previous studies in scientific databases was conducted using specific criteria. A total of 6,970 articles were found and will be screened. The title, description, and full text will be reviewed, and specific outcomes from each study will be noted. Treatments that target multiple symptoms for people with cancer is growing in interest. This study will summarize out the current knowledge of these treatments and will help guide future research to develop new transdiagnostic treatments for people living with and beyond cancer.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

The prevalence and natural progression of upper limb morbidity in breast cancer: three-year findings from the AMBER cohort study - Poster #720

Presenter: Margaret L. McNeely, Faculty of Rehabilitation Medicine, University of Alberta

Other Authors: Kerry Courneya, Shirin Shallwani, Leanne Dickau, Qinggang Wang, Jeffrey Vallance, S. Nicole Culos-Reed, Charles Matthews, Lin Yang, Christine Friedenreich

ABSTRACT_ID#3232

ABSTRACT

Introduction: Breast cancer treatments frequently result in upper limb morbidity, with effects reported to last more than 10 years. Impairments may include arm/shoulder pain, lymphedema, axillary web syndrome, limited shoulder mobility, and upper limb weakness, with symptoms associated with poorer upper limb function and quality of life. Objective: To explore the prevalence and natural progression of upper limb morbidity in females diagnosed with breast cancer participating in the AMBER study in Alberta Canada. Methods: Participants were recruited between 2012 and 2019. Objective measures of health-related fitness, body composition, upper extremity range of motion, axillary web syndrome, and lymphedema were performed within three months of breast cancer surgery, and at one- and three-year post-diagnosis. Results: 1,528 women participated in AMBER. Participants were a mean of 55 years (SD=11), most were White (87%), diagnosed with stage II or III (55%) breast cancer, and had breast conserving surgery (59%), and a sentinel lymph node biopsy (71%). For the present analysis, 1492 (98%), 1243 (81%) and 999 (65%) had objective measures of upper limb function at baseline, one and three years respectively. Upper limb morbidity was identified in 54% of participants after surgery and was associated with poorer upper limb function and higher pain. At three years, 30% of participants presented with upper limb morbidity, with a lymphedema prevalence of 5% after sentinel lymph node biopsy and 14% after axillary lymph node dissection. Conclusions: The AMBER data provide the opportunity to further explore medical, demographic, lifestyle, and clinical predictors of long-term upper limb morbidity.

LAY SUMMARY

- Women with breast cancer are at increased risk of developing upper limb morbidity such as shoulder pain and stiffness, and lymphedema after undergoing cancer treatments. The risk of upper limb morbidity is largely attributed to the type and extent of the breast cancer surgery (e.g., mastectomy and axillary lymph node dissection) and the use of radiation therapy. Of non-medical variables, age, lower levels of self-reported physical activity and higher body mass index (BMI) have been reported as risk factors for upper limb morbidity. Associations between objective measures of health-related fitness, including body composition, have yet to be fully explored.
- The AMBER cohort included a comprehensive collection of objective measures of upper limb range of motion, function and lymphedema as well as measures of health-related fitness, physical activity and body composition. Objective outcomes were assessed shortly after diagnosis, and at one- and three-year follow-up.
- At three-years, upper limb morbidity was identified in close to one-third of participants. Consistent with a high sentinel lymph node biopsy rate, however, the three-year prevalence of lymphedema was low. Understanding the relationship between potentially modifiable health-related fitness variables may serve to further guide intervention strategies to improve long-term upper limb morbidity outcomes.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Using a Serious Illness Conversation Guide to Improve the Quality of Life of Blood Cancer Patients - Poster #722

Presenter: Olivia Benguigui, Lady Davis Institute of Research

Other Authors: Dr. Sarit Assouline, Dr. Victoria Korsos, Dr. Chantal Cassis, Saima Ahmed

ABSTRACT_ID#3026

ABSTRACT

Background: Individuals with hematologic malignancies are more likely to die in acute care settings, including the ICU and under aggressive treatments, compared to those with solid tumours. This underscores the need for improved end-of-life (EOL) care in this population. The Serious Illness Conversation Guide (SICG) is a structured communication tool shown to reduce anxiety and depression in patients with advanced cancer^{1,2}. Objective: This ongoing study aims to evaluate whether implementing a serious illness conversation (SIC) with patients likely to die within 12 months from their hematologic malignancy reduces rates of death in acute care settings. Secondary objectives include assessing quality-of-life (QoL) outcomes and patients' perceived value of the SIC. Methods: Participants (n=34) are being recruited from the inpatient and outpatient hematology services at the Jewish General Hospital. Eligible patients are receiving active treatment and identified as at risk of dying within one year. Treating clinicians conduct an SIC with each participant. QoL is assessed before and after the SIC using the McGill Quality of Life Questionnaire³⁻⁵. Participants' charts are followed until death, and outcomes are compared to matched historical controls. Planned analyses include logistic regression for EOL outcomes (e.g., ICU death, aggressive care) and paired t-tests for pre-post QoL comparisons. Preliminary Results: Among the first 13 deaths, 30.7% occurred in acute care settings. Three of four acute care deaths occurred in the ICU under aggressive treatment; one occurred on a palliative ward. Conclusion: Preliminary findings suggest SIC may positively influence EOL care trajectories in hematologic malignancies.

LAY SUMMARY

Preliminary findings suggest that a substantial proportion of participants died in palliative settings and in alignment with their stated preferences after engaging in a timely Serious Illness Conversation (SIC) with their clinician. With full sample data and planned multivariable logistic regression analyses, we aim to demonstrate how SICs improve patient-centred care and end-of-life (EOL) outcomes in hematologic malignancies. Building on evidence from solid tumour populations, early feedback indicates that patients with hematologic malignancies and their families may also find the SIC intervention meaningful and emotionally supportive. As recruitment nears completion, we anticipate gaining further insight into the perceived benefits of SICs in this unique patient group. Beyond direct patient outcomes, this study may yield broader system-level benefits by identifying key facilitators and barriers to delivering high-quality EOL care in hematology. Findings will contribute to defining hematology-specific quality-of-life indicators and guiding improvements in how clinicians approach serious illness conversations, a topic with significant psychological and social implications for patients and their families.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

Steps toward improving the measurement of cancer pain management adequacy: a concept analysis of cancer pain overtreatment - Poster #723

Presenter: Lynn R. Gauthier, Laval University

Other Authors: Mathilde Bellavance, Gabrielle Deslauriers, Maud Bouffard, Maxime Bouchard, Sarah Béland, Lucia Gagliese

ABSTRACT_ID#3311

ABSTRACT

With increased cancer survival, prolonged pain-related opioid use may be rising, with unclear risks. While decades of pain management adequacy research have described undertreatment, overtreatment remains poorly defined and measured. This study aims to clarify the concept of cancer pain overtreatment to inform future measurement development. Using Joanna Briggs Institute scoping review methods, eligible articles defined elements of overtreatment, overprescribing, over-use, misuse or abuse in the context of adult cancer pain. MEDLINE, EMBASE, Cochrane Library, and CINAHL were searched from inception-August 2022. Two reviewers independently screened, reviewed, and categorized concepts as attributes, antecedents, or consequences using Walker and Avant's framework, as provider-, patient-, and system-oriented. 3469 titles/abstracts were screened. 599 full texts were assessed. 174 met inclusion criteria. 103 concepts were attributes (61%), antecedents (24%), or consequences (15%). Provider-oriented attributes (51%) included prescription misuse, high-dose prescribing, and prolonged opioid use. Patient+provider attributes (35%) included long-term or chronic use. Patient-oriented attributes (13%) included misuse, overuse, or abuse. One attribute was patient+society-oriented, reflecting sociopolitical context. No system-oriented attributes were identified. Patient-oriented antecedents (52%) included substance use history, psychological conditions, early trauma, risk of aberrant behaviours. Provider+system antecedents (36%) included guideline adherence and assessment tools. Most consequences were patient-oriented (80%), including persistent use, toxicity, dependency, tolerance, aberrant behaviours. A preliminary conceptual definition involving patient history and prescribing guidelines includes prolonged prescribing/use, misuse, and high opioid dose patient/provider-associated attributes. This study clarifies cancer pain overtreatment and reveals gaps, particularly the lack of system-level attributes and the need to examine sociopolitical antecedents and consequences.

LAY SUMMARY

As cancer treatments improve survival, there is an increased likelihood of prolonged opioid exposure to treat chronic pain. The risks of this remain unknown. Research focused on the adequacy of cancer pain management has exclusively considered undertreatment. Overtreatment has been underexplored, in part because validated measures do not exist. As a first step toward developing a measure, this study aimed to clarify the concept of cancer pain overtreatment. The study followed the Joanna Briggs Institute scoping review methods to identify articles that described elements of overtreatment, overprescribing, overuse, misuse, or abuse in the context of adult cancer pain management. 174 articles were included in the review from which 103 concepts of overtreatment were identified. Concepts were classified as attributes (definitions), antecedents (preceding factors), or consequences, that were provider-oriented, patient-oriented, or system-oriented. Most concepts were attributes that were provider-oriented or patient- and provider-oriented. Through attribute review, a preliminary conceptual definition of overtreatment was developed, considering patient history and prescribing guidelines, including prolonged prescribing/use, misuse, and high opioid doses. The review also revealed some important gaps, including system-oriented definitions and the need for more research into the sociopolitical risk factors and consequences of cancer pain overtreatment.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Toward Standardized Clinical Assessment of CIPN in Rehabilitation: Identifying Relevant Tools for Practice - Poster #724

Presenter: Antoine Frasier, CR-CHU de Quebec

Other Authors: Charles-Antoine Auger, Stephanie Bernard, Kadja Perreault, Maud Bouffard, Sarah Béland, Philippe Bérubé-Mercier, Frédérique Therrien, Philip L. Jackson, Julie Lemieux, Anne Dionne, Jennifer Gewandter, Lynn R. Gauthier

ABSTRACT_ID#3314

ABSTRACT

Background: Rehabilitation shows promise in managing chemotherapy-induced peripheral neuropathy's (CIPN) sensory and motor effects. Assessment tools' relative validity for identifying clinically relevant acute and chronic CIPN remains unclear. **Aim:** Identify sensory and functional patient-reported (PROs), clinician-reported (ClinROMs), and performance-based (PerfOMs) outcome measures most strongly associated with clinically relevant acute and chronic CIPN. **Method:** People with stage I-III breast cancer completed the FACT/GOG-NTX-4 at baseline (T0), post-taxane (T1), and three months later (T2). A clinical anchor-based Minimally Important Difference (MID) of ≥ 3.39 from T0 defined MID-CIPN at T1 and T2. PROMs (Short-Form McGill Pain Questionnaire-2 [SF-MPQ-2]; Neuropathic Pain Symptom Inventory; Brief Pain Inventory-Interference), ClinROMs (Total Neuropathy Scale-revised-Short-Form [TNSr-SF]; hand/foot-cold/hot pain threshold; cold/warm, vibration, mechanical detection threshold; Karnofsky Performance Status Scale), and PerfOMs (Short Physical Performance Battery [SPPB], Timed-Up-and-Go) were collected. Stepwise logistic regression identified T1/T2 sensory/functional measures associated with T1/T2-MID-CIPN presence. **Results:** 114 participants (52.30 ± 11.18 years old; 100% female; 62.3% paclitaxel, 36.8% docetaxel) were included. 64 (51%) and 49 (39%) had MID-CIPN at T1 and T2. T1-MID-CIPN was associated with T1-SF-MPQ2-Neuropathic subscale (OR=4.103; 95%CI: 1.780-9.453), T1-cold pain threshold-hand (CPT-H; OR=1.192; 95%CI: 1.025-1.386), T1-TNSr-SF (OR=1.335; 95%CI: 1.101-1.619). T2-MID-CIPN was associated with T2-TNSr-SF (OR=1.322; 95%CI: 1.084-1.611), T2-SPPB (OR=0.119; 95%CI: 0.015-0.949). **Conclusion:** Measures associated with acute CIPN were the SF-MPQ-2-Neuropathic PROM, and CPT-H (upper-extremity cold allodynia) and TNSr-SF (upper- and lower-extremity vibration sensation, reflexes, neuropathy/neuropathic pain) ClinROMs. Measures associated with chronic CIPN were the TNSr-SF and the SPPB (lower extremity functioning and balance) PerfOM. Future research should define clinically relevant thresholds and rehabilitation assessment guidelines.

LAY SUMMARY

Rehabilitation is a key component of supportive care to prevent and manage side effects of cancer treatments such as chemotherapy-induced peripheral neuropathy (CIPN). Many assessment tools are used in these care settings, with no consensus about which should be used. To measure clinically relevant acute CIPN, clinicians should consider using a combination of patient reported outcomes and clinician reported outcomes measuring neuropathic pain, cold pain sensitivity, and vibration sensitivity, reflexes, and neuropathy in the upper and lower extremities. To measure clinically relevant chronic CIPN, clinicians should consider using a combination of clinician reported outcomes and performance-based outcomes measuring vibration sensitivity, reflexes, and neuropathy in the upper and lower extremities, and lower-extremity balance and functioning. Future research is needed to understand clinically relevant thresholds for these tools and establish rehabilitation assessment guidelines.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

The Wish to Hasten Death and Death Anxiety in Medical Assistance in Dying: Overlapping But Distinct Constructs - Poster #725

Presenter: Madeline Li, Princess Margaret Cancer Centre

Other Authors: Stefan Aguiar, Eugenia Ubeira Amaral, Aliza Panjwani, Gilla Shapiro, Anne Rydall, Emma Hagopian, Robin Graham, Roberta Yael Klein, Anne Barbeau, Gary Rodin

ABSTRACT_ID#2852

ABSTRACT

Background. The desire for medical assistance in dying (MAiD) ranges from 2.5-10% in patients with advanced cancer. This study describes the relationship of distress variables to MAiD interest. **Methods.** Descriptive data are presented from a longitudinal study on the desire for MAiD in patients with advanced cancer. The sample comprised 330 patients at baseline and 44 that requested MAiD, of which 16 received MAiD. Pearson correlations were calculated between the Wish to Hasten Death (SAHD-A), Will to Live Scale (WTLS), Death and Dying Distress Scale (DADDS), Self Perceived Burden Scale (SPBS), Patient Health Questionnaire-9 (PHQ-9) depression scale, and Edmonton Symptom Assessment Scale-revised (ESAS-r) symptom burden. **Results.** Prevalence of high SAHD-A and DADDS: Across categories from no MAiD interest (2.4%, 33%) to MAiD request but died naturally (18%, 45%) and MAiD receipt (21%, 27%). Mean WTLS remained high across all groups. The DADDS was significantly positively correlated ($p \leq 0.01$) with SPBS ($r = 0.425$), PHQ-9 ($r = 0.527$), and ESAS-r ($r = 0.547$) in those with no MAiD interest. Among MAiD requestors, DADDS correlated with only SPBS ($r = 0.572$). **Conclusions.** The wish to hasten death remains low in most patients who request and complete MAiD, while the will to live remains high. Death anxiety is highest in patients who request, but do not receive MAiD. Death anxiety is associated with multiple types of distress among individuals not requesting MAiD, but only self-perceived burden among MAiD requester. There are many psychological states within MAiD interest potentially amenable to psychosocial interventions.

LAY SUMMARY

The wish to hasten death is low and the will to live remains high in most patients who request MAiD. Death anxiety is higher in patients who request MAiD, but lower among MAiD recipients. Findings may assist clinicians in recognizing psychological states in patients considering MAiD which are potentially amenable to psychosocial interventions.

Supportive, Palliative and End of Life Care

Sunday, November 2, 2025

Patterns of Aggressive End-of-Life Care in Advanced Cancer Patients - Poster #727

Presenter: Vikas Garg, Windsor Regional Hospital

Other Authors: Alejandra Ruiz Buenrostro, Katrina Heuniken, Rebecca Bagharol, Mohamed Yousef, Katrina Sajewicz, Suman Dhanju, Kirsten Wentlandt, John Kuruvilla, Stephanie Lheureux, Camilla Zimmermann, Breffni Hannon

ABSTRACT_ID#3277

ABSTRACT

Aggressive care (ED visits, hospital and ICU admissions, and systemic anticancer therapy [SACT] administration) at the end-of-life (EOL) is a recognized indicator of poor-quality cancer care. Our aim was to evaluate the prevalence of aggressive care during the last 15 and 30 days of life. Methods: Clinical and demographic data, as well as indicators of aggressive care in the last 15 and 30 days of life were retrospectively extracted from the electronic health records of patients with advanced cancer who died in 2019. Results: Out of 2,893 patients who died in 2019, a random sample of 639 (20%) was screened, and data from 443 eligible patients were analysed. Median age was 72 years (range: 30-102), 80% had solid tumours, while 20% had haematological malignancies. In the last 30 days of life, 176 pts (40%) had at least one ED visit, 231 (52%) had a hospital admission, 42 (10%) were admitted to the ICU, and 88 (20%) received SACT. In the last 15 days of life, 99 pts (23%) had at least one ED visit, 177 (42.5%) were admitted to the hospital, 42 (10%) were admitted to the ICU, and 37 (8%) received SACT. Patients with a shorter interval from diagnosis to death (<6 and <12 months) had higher odds of aggressive EOL care compared to patients diagnosed >6 and >12 months prior to death, respectively. Conclusion: A significant proportion of patients with advanced cancer received aggressive EOL care, underscoring the need for optimizing goals of care to enhance QOL.

LAY SUMMARY

Aggressive end-of-life care is common — 40% visited the emergency room and 20% received cancer treatment in the last month of life, often reducing quality of life. One in ten patients were admitted to the ICU in their final days, suggesting potential overuse of intensive treatments near death. Patients with shorter survival after diagnosis were more likely to receive intensive care, showing a need for earlier goals-of-care discussions.

Supportive, Palliative and End of Life Care

Monday, November 3, 2025

Survey of radiation therapists' current practices and perceptions of psychosocial and supportive care in Canada and Norway - Poster #728

Presenter: Sophie Bezanson, St. Francis Xavier University

Other Authors: Espen Aas, Kerry-Ann Smith, Lisa Di Prospero, Michael Velec, Arlinda Ruco, Safora Johansen

ABSTRACT_ID#2499

ABSTRACT

Purpose Many cancer patients undergoing radiation therapy report unmet psychosocial needs, which can negatively impact their treatment outcomes and quality of life. This study explored the current practices and perceptions of radiation therapists (RTs) practicing in Canada and Norway with respect to addressing the psychosocial and supportive care (PSSC) needs of their patients. **Methods** A cross-sectional study was conducted using an online evidence-informed survey of RTs in Canada and Norway that focused on: (1) demographics, (2) RTs' confidence level and perceptions related to PSSC, and (3) RTs' current practices related to PSSC. Descriptive statistics, Chi-square tests, and Mann-Whitney U tests were used to summarize the sample and compare differences between countries. **Results** A total of 210 RTs completed the survey (Canada: n=79; Norway: n=131). RTs in both countries identified PSSC as an important aspect of quality care. Canadian RTs expressed a greater desire to expand their scope of practice in PSSC ($p=0.001$). Norwegian RTs reported spending more time providing PSSC (Mean: 8.3h vs. 3.8h; $p<0.001$) and were satisfied with their current capacity. Barriers common to both countries included a lack of training and time constraints. Canadian RTs additionally reported limited institutional support. **Conclusions** Significant differences exist in the current practices and perceptions of RTs in Canada and Norway regarding PSSC delivery. However, Canadian and Norwegian RTs agree that engaging in PSSC ensures the best care for patients undergoing radiation therapy. With enhanced training, clear national guidelines, and institutional support, RTs can better address the PSSC needs of their patients.

LAY SUMMARY

Radiation therapists (RTs) in Canada and Norway perceived that providing psychosocial and supportive care (PSSC) is within their scope of practice and part of their daily treatment that ensures quality care for patients. Canadian RTs expressed a greater interest in expanding their knowledge and capacity to engage in PSSC compared to Norwegian RTs, which may be a result of differences in the organizational structure, workflow, and/or barriers between both countries. Canadian and Norwegian RTs demonstrated different approaches to addressing PSSC needs and faced unique barriers to providing such care, including time constraints, inadequate staffing, and limited institutional support. Targeted interventions, such as offering comprehensive training programs and promoting clear national guidelines, may better support RTs in delivering PSSC to patients undergoing radiation therapy.

Associations Between Early Breast Cancer Treatments and One-Year Changes in Health-Related Fitness: Results from the AMBER Cohort Study - Poster #729

Presenter: Fernanda Arthuso, University of Alberta

Other Authors: Ki-Yong An, Qinggang Wang, Renée Kokts-Porietis, Andria Morielli, Margaret McNeely, Jeff Vallance, S. Nicole Culos-Reed, Gordon Bell, Leanne Dickau, Myriam Filion, Stephanie Ntoukas, Jessica McNeil, Lin Yang, Charles Matthews, Christine Friedenreich, Kerr

ABSTRACT_ID#2589

ABSTRACT

Background: Early-stage breast cancer treatments adversely affect components of health-related fitness (HRF), an important component for treatment tolerability, recovery, and long-term health outcomes. Few studies have examined cancer treatment modality-specific effects on HRF. **Purpose:** To examine the associations of different breast cancer treatment modalities and combinations with changes in HRF from baseline to one year. **Methods:** Newly diagnosed early-stage breast cancer patients were recruited between 2012-2019 for the Alberta Moving Beyond Breast Cancer (AMBER) cohort study. HRF (i.e., cardiorespiratory fitness, muscle strength and endurance, and body composition) was assessed within 90 days of diagnosis and at one year. Treatment data were abstracted from medical charts. Analysis of covariance was used to test whether HRF changes differed between treatment modalities and combinations. **Results:** A total of 1350 participants (55.6 ± 10.7 years) were included. Women who received chemotherapy ($n=797$; 59%) experienced smaller increases in upper body strength (-1.7 kg, 95%CI: -3.0 to -0.5) and greater declines in lower body endurance (-118.0 kg, 95%CI: -216.6 to -19.3), total lean mass (-0.7 kg, 95%CI: -1.1 to -0.3), bone mineral density (-0.01 g/cm², 95%CI: -0.02 to 0.00), and bone mineral content (0.04 kg, 95%CI: -0.06 to -0.02) compared to the no-chemotherapy group. Other treatment modalities were modestly and inconsistently associated with HRF changes. Treatment combinations that included chemotherapy had the most negative impact on muscle endurance and body composition. **Conclusions:** Chemotherapy—either alone or in combination with other treatments—had the largest and broadest negative impact on HRF in early-stage breast cancer at one-year follow-up.

LAY SUMMARY

While treatments for small and localized breast cancer are often curative, they can have negative effects on physical fitness. This issue matters because greater physical fitness helps patients tolerate treatment better, recover more effectively, and maintain long-term health. Few studies have examined how specific types of cancer treatment affect physical fitness. In this study of 1,350 women with early stage and localized breast cancer, we explored the connections between different treatment, alone and in combination, with changes in physical fitness over one year. Women who received chemotherapy experienced greater reductions in lower body strength, muscle mass, and bone health compared to those who did not receive chemotherapy. Other types of treatment had smaller or less consistent effects on physical fitness. Treatment combinations that included chemotherapy had the most negative impact on muscle endurance and body composition. Chemotherapy, used alone or with other treatments, was associated with the greatest declines in physical fitness one year after treatment. Understanding how different breast cancer treatments relate to physical fitness changes can help guide the development of tailored exercise programs. These programs may help patients maintain or improve their fitness, manage treatment more effectively, improve recovery, and support long-term health.

Associations Between Health-Related Fitness, Physical Activity and Sedentary Behaviour with Chemotherapy Outcomes in the Alberta Moving Beyond Breast Cancer Cohort - Poster #731

Presenter: Andria R. Morielli, Alberta Health Services

Other Authors: Renée Kokts-Porietis, Lin Yang, Charles Matthews, Sasha Lupichuk, Gloria Urgoiti, Margaret McNeely, S. Nicole Culos-Reed, Jeff Valance, Leanne Dickau, Kerry Courneya, Christine Friedenreich

ABSTRACT_ID#3067

ABSTRACT

Background: Relative dose intensity (RDI) and pathologic complete response (pCR) are prognostic factors for breast cancer survival. Little is known about how nonpharmacological factors may be leveraged to improve these outcomes. We examined the associations between health-related fitness (HRF), physical activity (PA) and sedentary behaviour (SB) at diagnosis with RDI and pCR among newly diagnosed breast cancer patients receiving (neo)adjuvant chemotherapy in the Alberta Moving Beyond Breast Cancer Cohort. Methods: Between 2012 and 2019, 890 breast cancer patients receiving chemotherapy completed HRF (i.e., cardiorespiratory fitness, muscular strength and endurance and body composition) PA, and SB measures. Clinical data was abstracted from medical charts. Logistic regression was used to estimate multivariable-adjusted odds ratios (OR) and 95% confidence intervals (95%CI) for HRF, PA and SB exposures with RDI (<85%/≥85%) and pCR (no/yes). Results: 726 (81.6%) participants achieved ≥85% RDI. Positive dose-response relationships were observed between relative VO2peak (ORper 1 ml/kg/min=1.06, 95%CI=1.02-1.10), upper body strength (ORper 10 kg=1.26, 95%CI=1.02-1.56) and lower body strength (ORper 10 kg=1.08, 95%CI=1.01-1.14) with ≥85% RDI. Favorable body composition variables (e.g., lean body mass percentage) were positively associated with ≥85% RDI, whereas unfavorable body composition variables (e.g., waist circumference) were negatively associated with ≥85% RDI. For pCR, associations were observed for lean-to-fat mass ratio (ORper 0.1=1.15, CI=1.00-1.32), body mass index (ORper 1 kg/m²=0.89, CI=0.80-0.99) and total PA (ORper 10 MET-h/wk/yr=0.90, CI=0.83-0.97). Conclusion: HRF variables were associated with better treatment outcomes. Exploration of the feasibility and impact of implementing an exercise intervention among breast cancer patients awaiting chemotherapy is warranted.

LAY SUMMARY

Better physical fitness and body composition may improve a breast cancer patient's ability to complete chemotherapy; Better body composition may lead to greater tumor shrinkage in women receiving chemotherapy prior to surgery for breast cancer; There is a need to test the feasibility and effects of exercise on outcomes including physical fitness, body composition, in newly diagnosed breast cancer awaiting chemotherapy

From Patient Voice to Executive Authority: Transforming Cancer Research Leadership Through Lived Experience - Poster #732

Presenter: Carla Bossart-Pletzer, Northern Health Equity Consulting

ABSTRACT_ID#2517

ABSTRACT

Background: While patient engagement in cancer research has evolved significantly, most models confine cancer survivors to advisory roles rather than positions of executive authority. This perpetuates traditional power structures where patients share experiences but administrators set priorities and allocate resources. **Methods:** We analyzed existing models of patient leadership in cancer research organizations, examining governance structures, decision-making authority, and barriers to authentic patient leadership. We developed Northern Health Equity Consulting (NHEC) as a case study of patient-led organizational design, where cancer survivor lived experience serves as primary qualification for executive leadership. **Results:** Research revealed limited examples of patients in formal leadership positions within research organizations. The Canadian Cancer Trials Group provides the strongest model, with cancer survivors chairing governance committees with voting rights and protocol approval authority. Internationally, patient-founded organizations like the European Cancer Patient Coalition demonstrate executive leadership by cancer survivors, but traditional academic research institutions rarely elevate patients beyond advisory roles. **Discussion:** NHEC represents an innovative model where a cancer patient serves as Executive Director of a health equity consulting organization, maintaining autonomy while influencing system change. This approach challenges the traditional paradigm where patients are confined to "patient engagement" roles, instead demonstrating how lived experience can serve as qualification for executive leadership across organizational functions. **Conclusions:** The transition from patient advisor to executive leader requires intentional structural changes in how organizations value lived experience as professional qualification. Northern Ontario's context provides unique opportunities to pioneer patient-led approaches to cancer research leadership, moving beyond consultation toward authentic decision-making authority.

LAY SUMMARY

1. Most cancer patients are stuck giving advice, not making decisions. While hospitals and research organizations ask cancer survivors to share their experiences on advisory committees, they rarely give patients actual power to set priorities, allocate budgets, or lead programs.
2. The few patients who do hold leadership positions show impressive results. Organizations where cancer survivors chair committees with real decision-making authority have achieved measurable improvements in research relevance, patient recruitment, and care quality.
3. Moving from patient advisor to executive leader requires completely rethinking how we value lived experience. True patient leadership means treating cancer survivorship as a professional qualification, not just a personal story.

Driving innovation in ovarian cancer survivorship research through national patient and caregiver engagement: an Ovarian Cancer Canada and Cancer Research Society joint initiative - Poster #733

Presenter: Alicia Tone, Ovarian Cancer Canada

Other Authors: Jessica Lawson, Irina Navarrete, Patient Partners in Research (PPiR), Leslie Born, Dena Edwards, Dajan O'Donnell

ABSTRACT_ID#2795

ABSTRACT

Purpose To develop and implement a national ovarian cancer survivorship research competition driven by the greatest needs of patients and caregivers, while building a foundation for long-term impact on survivorship care and policy. **Methods** Twenty-six patients and caregivers from 9 provinces/territories worked with Ovarian Cancer Canada (OCC) and Cancer Research Society to co-develop a competition framework and 15-question bilingual, anonymous survey to define national survivorship priorities. Results were summarized by counts/percentages or thematic analysis of open-text responses and communicated to researchers as part of the RISE (Research Impact in Survivorship and Engagement) competition launch in April 2025. **Proposals** will be screened by academic experts (pass/fail), with patients and caregivers driving final rankings and funding recommendations. **Results** We received 134 survey responses (82% EN/18% FR), including from 118 (88%) people with ovarian cancer and 16 (12%) caregivers. 58% of participants were diagnosed between 2020-2024, 35% were diagnosed before age 50, 4% were First Nations, Métis or Inuk (Inuit), 31% had a household income less than \$75,000, 76% were married and 70% were from urban communities. The most disruptive challenges for patient respondents were related to physical (90%), emotional/mental (86%) and sexual (48%) health, navigating healthcare systems (49%) and changes in family dynamics (48%). In response to published competition guidelines based on our findings, we received 25 Expressions of Interest from 5 provinces; 19 (76%) applicants were new to OCC. **Conclusions** This unique competition driven by individuals directly impacted by ovarian cancer has garnered significant interest from patients, caregivers and multidisciplinary researchers.

LAY SUMMARY

People directly affected by ovarian cancer (as a patient or caregiver) are working with Ovarian Cancer Canada and Cancer Research Society to develop and implement a national funding competition on ovarian cancer survivorship, that is driven by the greatest needs of the community. A national survey of patients and caregivers revealed the most disruptive challenges faced following an ovarian cancer diagnosis, such as impacts on physical, mental/emotional and sexual health, changes in family dynamics and navigating the healthcare system. The open funding call resulting from this survey has attracted researchers from outside of Ovarian Cancer Canada's typical biomedical applicant pool, indicating wide interest in this topic and the value of a patient- and caregiver-driven, multidisciplinary approach.

"How AI Saved My Life at Kilometer 12": Artificial Intelligence as a Diagnostic Integration Tool for Complex Cancer Survivorship - Poster #734

Presenter: Carla Bossart-Pletzer, Northern Health Equity Consulting

ABSTRACT_ID#2518

ABSTRACT

Background: Cancer survivors with complex treatment histories often experience multi-system late effects that challenge traditional healthcare delivery models. Specialized care creates silos where symptoms are addressed separately without integration. **Case Presentation:** A 38-year-old survivor of stage 3 triple negative inflammatory breast cancer experienced exercise intolerance, autonomic dysfunction, and neurological symptoms two years post-treatment following maximum-dose chemotherapy, bilateral mastectomies, surgical menopause, and radiation. Traditional healthcare addressed symptoms separately. **Methods:** The patient engaged with AI technology over multiple sessions, providing detailed symptom history, treatment timeline, and physiological data. AI analysis identified connections between disparate symptoms, analyzed temporal relationships between stressors and symptom flares, and integrated current symptoms with documented chemotherapy late effects. **Results:** AI revealed that symptoms represented autonomic nervous system dysfunction secondary to chemotherapy-induced neuropathy, complicated by surgical menopause. AI identified specific triggers precipitating symptom cascades and explained exercise limitations. At kilometer 12 of a half-marathon, AI-informed understanding of warning signs prevented a potentially dangerous medical crisis. **Clinical Impact:** Following AI-assisted analysis, the patient avoided hospitalization, developed evidence-based activity modification strategies, improved healthcare provider communication, and reduced anxiety through symptom validation. **Implications:** AI demonstrated potential as a diagnostic integration tool for complex survivorship scenarios, providing cross-specialty knowledge synthesis, pattern recognition, and therapeutic validation unavailable through traditional care models. This approach could improve patient understanding, enhance care coordination, and reduce healthcare utilization. **Conclusions:** AI technology offers promising applications for complex cancer survivorship management, particularly for patients with multi-system late effects poorly served by current care models.

LAY SUMMARY

AI can connect the dots that doctors miss: Cancer survivors often have multiple health problems that seem unrelated, but doctors usually treat each symptom separately. AI was able to look at all the symptoms together and explain how they were connected - showing that heart problems, digestive issues, and exercise difficulties were all part of the same underlying damage from chemotherapy. Technology can prevent medical emergencies: By understanding how all these health problems worked together, AI helped identify warning signs that something dangerous was happening during a half-marathon. This prevented a potential hospitalization by recognizing the early signs of a medical crisis that might have been missed otherwise. Patients need tools to understand their own complex medical situations: Current healthcare is so specialized that no one person sees the whole picture of what cancer survivors are dealing with. AI provided a way to understand how cancer treatment affects the whole body over time, giving patients better language to communicate with their doctors and make smarter decisions about their health.

Assessing the therapeutic potential of group digital storytelling among women with premenopausal breast cancer: A pilot study - Poster #735

Presenter: Janet de Groot, Arthur JE Child Comprehensive Cancer Centre

Other Authors: Jessame Gamboa, Ana Herrera, Dana Male, Carly-Ann Haney, Laura Labelle, Chloe Hao, Kathleen Sitter

ABSTRACT_ID#3333

ABSTRACT

BACKGROUND: Post-treatment women with breast cancer wished to connect, share and process identity changes, self-care and managing expectations of returning to a 'normal life'. The use of a group framework for the digital storytelling (DS) creative process of making videos, increases connection and conversation among participants about shared experiences. Anecdotally individuals participating in DS describe the process as therapeutic. **AIM:** To evaluate the therapeutic efficacy of a novel short-term (6 session) DS group among women with pre-menopausal breast cancer. **METHOD:** Concurrent mixed methods study to support comparison of quantitative findings with qualitative data from individual participant interviews, to provide a nuanced perspective of the findings. **RESULTS:** Following DS group completion, women (n=7) with premenopausal breast cancer showed a mean reduction of PCL-5 scores of 9.7 (17.8), a clinically significant improvement with a moderate effect size (0.75 [-0.53, 2.03]). Most found developing a DS using photos, music and narrative about their cancer diagnosis and treatment overwhelming initially. Group support contribute to processing emotions and subsequently, participants described feeling calmer, more at peace and gaining a capacity to discuss their cancer experience more concisely. **CONCLUSION:** The pilot art-based therapy study conveys promising findings for the therapeutic potential of DS groups among women with premenopausal breast cancer. A larger scale (n> 100) multi-site study inclusive of diverse, young adult (18 to 39 years) women with premenopausal breast cancer to evaluate for statistically and clinically significant improvement across symptoms of depression, traumatic stress response and recurrence fears.

LAY SUMMARY

Among women with pre-menopausal breast cancer, a six-session digital storytelling group was associated with clinically significant reductions in trauma symptoms and gaining narrative competence in sharing the cancer experience. A multi-site larger scale study of group DS is required among the young adult population of women with pre-menopausal breast cancer to evaluate for clinically significant changes in psychological functioning.

Behaviour Change Interventions to Promote Healthy Lifestyles in Adolescent and Young Adult Childhood Cancer Survivors (AYA-CCS): A Scoping Review - Poster #736

Presenter: Charlotte G  linas-Gagn  , Universit   de Montr  al

Other Authors: Bilodeau Karine, Nikita Guarascio, Maxime Caru, Leandra Desjardins, S  bastien Perreault, Hallie Coltin, Caroline Laverdi  re, Serge Sultan

ABSTRACT_ID#2438

ABSTRACT

Context: Adolescent and young adult childhood cancer survivors (AYA-CCS) are at increased risk of chronic illnesses and long-term treatment-related effects. Adopting healthy behaviours can significantly improve their overall health and reduce both morbidity and mortality. This scoping review aims to map behaviour change interventions that promote healthy lifestyles among AYA-CCS. **Methodology:** A systematic search was conducted across multiple databases, following the Joanna Briggs Institute guidelines. Included studies involved interventions targeting health behaviours, specifically focusing on physical activity, nutrition, alcohol use, and tobacco consumption among childhood cancer survivors aged 15–39 at the time of the intervention. **Results:** A total of 3156 sources were identified, of which 39 met inclusion criteria. Interventions were categorized using Michie’s (2018) framework, which includes nine intervention functions and six sources of behaviour change. Our analysis indicates a strong focus on physical activity (n = 32) and a heavy reliance on education and training functions, as well as psychological capacity and reflective motivation. These findings contrast with existing literature that emphasizes the need for interventions targeting alcohol and tobacco use, key modifiable risk factors during this pivotal stage of development. **Discussion:** Our findings highlight a narrow focus in existing interventions, which tend to prioritize physical activity while underemphasizing critical behaviours such as nutrition, alcohol and tobacco use. This limited scope may reduce the overall effectiveness of current health promotion efforts. Furthermore, the dominance of educational strategies suggests a need to integrate more varied and evidence-based techniques, such as incentivization, environmental restructuring or modeling, to facilitate behaviour change.

LAY SUMMARY

- This review provides a foundation for advancing clinical practice and guiding the development of more comprehensive intervention programs. By identifying key intervention components and gaps, it supports efforts to improve the long-term health outcomes of AYA-CCS through targeted and multidimensional behaviour change strategies.
- Most health programs for adolescent and young adult survivors of childhood cancer (AYA-CCS) focus almost entirely on physical activity.
- Very few interventions address nutrition, alcohol and tobacco consumption, even though these are major risk factors for long-term health problems. · Interventions rely heavily on education and training, while more effective strategies, such as modelling, environmental restructuring, and enablement, are rarely used.
- Key takeaway: There’s a clear gap between what AYA-CCS need and what current programs offer. To truly support their long-term health, future interventions must be more balanced, comprehensive, and tailored to address a wider range of behaviours and barriers.

Causes of death among individuals diagnosed with head and neck cancer in Canada, 1992-2015: A population-based cohort study - Poster #738

Presenter: Claudie Laprise, Centre de recherche du CHUM

Other Authors: Theerthika Dillibabu, Sreenath Madathil, Belinda Nicolau, Karena Volesky-Avellaneda

ABSTRACT_ID#2490

ABSTRACT

Background: Individuals diagnosed with head and neck cancer (HNC) face elevated mortality risk due to the disease's aggressive nature, treatment complications, recurrence, and secondary malignancies. **Objectives:** To determine leading causes of death among Canadians diagnosed with HNC (1992-2015) and compare mortality risks to the general population. **Methods:** We identified Canadians diagnosed with HNC from the Canadian Cancer Registry, using the International Classification of Diseases for Oncology, 3rd Edition (C00-C14 and C30-C32.9). Cause-of-death data was obtained by linking records to the Canadian Vital Statistics Database for the period 1992–2015. To compare mortality risks of HNC patients to the Canadian general population, we calculated standardized mortality ratios (SMRs), standardized by sex and age. Using Poisson regression, we identified risk factors for mortality. **Results:** Among 97,655 individuals diagnosed with HNC between 1992-2015, most had oropharynx (22.1%), oral cavity (21.8%), or hypopharynx/nasopharynx/larynx cancers (37.6%). Of 57,895 deaths, leading causes were neoplasms (71.5%), heart diseases (8.2%), and respiratory diseases (2.8%). HNC patients had doubled mortality risk versus the general population. Additionally, HNC patients had elevated risk of death from cancer (SMR=5.45, 95%CI: 5.38-5.52), and suicide (SMR=1.44, 95%CI: 1.19-1.70). Among HNC patients, mortality was higher among males, those diagnosed at older ages, and during the first five years after diagnosis. There was a markedly reduced in mortality in recent diagnosis cohorts. **Conclusion:** HNC patients face elevated mortality risk from cancer and suicide compared to the general Canadian population. These findings demand intensive survivorship care integrating rigorous cancer surveillance, comorbidity management, and targeted mental health interventions.

LAY SUMMARY

Head and neck cancer (HNC) survivors face elevated cancer mortality, with a 5-fold higher risk of cancer death compared to the general population, with cancer accounting for over 70% of all deaths in this population. Suicide risk is higher among HNC survivors compared to the general population. Mortality patterns vary by patient characteristics: higher mortality occurs among males, older patients at diagnosis, and during the first five years after diagnosis, with recent diagnosis cohorts.

Factors associated with transition of cancer survivors to primary care post-treatment: unique insights from linked survey and administrative health data - Poster #739

Presenter: Cynthia Kendell, Dalhousie University

Other Authors: Lynn Lethbridge, Robin Urquhart

ABSTRACT_ID#2886

ABSTRACT

Background: Cancer survivors are increasingly discharged (“transitioned”) to primary care after treatment, however, practice is inconsistent within and across Canadian jurisdictions. Reasons for this are unclear, partly due to the limited availability of data on patient needs and other factors that may impact clinical decision-making regarding discharge. This study sought to address this gap via novel data linkage. Objectives: To examine the association between transition to primary care and (1) post-treatment patient needs (physical, emotional, practical), (2) receipt of services and supports, and (3) clinical and demographic factors. Methods: Data from the 2016 national Cancer Transition Survey for Nova Scotia (NS) respondents were linked to NS cancer registry and administrative health data. Respondents included individuals diagnosed with hematologic, breast, colorectal, melanoma, prostate, and AYA cancers (all cancer types) who were 1 to 3 years post-treatment. Transition to primary care was defined as having zero oncology visits during the follow-up timeframe (<2 visits for breast cancer). The factors associated with transition to primary care were identified using multivariate logistic regression. Results: Of the study cohort (n=1050), 23.7% transitioned to primary care post-treatment. When clinical and demographic factors were accounted for, only physical needs were associated with transition to primary care. Specifically, those who reported moderate to high levels of physical need were more likely to have been transitioned. Discussion: It is unclear whether individuals were transitioned because of their physical needs, or whether those who were transitioned had physical needs that were not met in primary care. Additional research is required.

LAY SUMMARY

Cancer survivors who were transitioned (i.e., discharged) to primary care after completed treatment reported higher levels of physical need compared to those who were not transitioned. It is unclear whether individuals with higher levels of physical need were more likely to be transitioned to primary care, or whether individuals who have been transitioned to primary care were more likely to have higher levels of unmet physical need. Additional research is required to better understand the impact of physical need on oncologists' decision-making regarding the transitioning of cancer survivors to primary care for follow-up.

The Theory of Planned Behavior's Prediction of Physical Activity in Newly Diagnosed Women with Breast Cancer - Poster #740

Presenter: S. Nicole Culos-Reed, University of Calgary

Other Authors: Chad Wagoner, Christine Friedenreich, Kerry Courneya, Qinggang Wang, Jeff Vallance, Charles Matthews, Lin Yang, Margaret McNeely, Leanne Dickau, S. Nicole Culos-Reed

ABSTRACT_ID#2706

ABSTRACT

Background. Most breast cancer survivors do not meet recommended physical activity (PA) levels. While the Theory of Planned Behavior (TPB) has been used to understand PA, few studies have explored its longitudinal prediction of PA. This analysis evaluated the TPB's prediction, measured at diagnosis, of PA levels at one-year post-breast cancer diagnosis. **Methods.** Women with Stage I-IIIc breast cancer participating in the Alberta Moving Beyond Breast Cancer (AMBER) study were included. TPB determinants (attitudes, subjective norms, perceived behavioral control, and intentions) toward recreational PA near diagnosis were assessed via questionnaire. ActiGraph GT3X+® devices measured light and moderate-vigorous PA (MVPA), and self-reported recreational PA was assessed with the Past Year Total Physical Activity Questionnaire one year after diagnosis. Structural equation models evaluated the TPB's prediction of PA. **Results.** 1,528 newly diagnosed women with breast cancer were enrolled (average 55.4 years of age). At diagnosis, positive attitudes ($b=0.51$) and greater perceived behavioral control ($b=0.16$) were associated with greater intentions for device-measured ($R^2=0.51$, $p<0.001$) and self-reported PA ($R^2=0.54$). Greater intentions for MVPA at diagnosis were associated with greater MVPA at one year ($b=0.13$; $p<0.01$), but intentions were not associated ($p>0.05$) with light-intensity PA ($b=0.07$) or self-reported recreational PA ($b=0.08$). **Conclusion.** The TPB variables were limited in their ability to predict PA one year after a breast cancer diagnosis. These findings highlight the need to explore additional factors that may impact long-term PA among women newly diagnosed with breast cancer, including how breast cancer treatment may modify PA intentions and behavior.

LAY SUMMARY

The TPB predicted future activity levels, but only for more intense exercise. Greater intentions through the TPB was associated with greater moderate-to-vigorous activity a year later; however, this was not the case for lighter physical activity or self-reported physical activity. Attitudes and perceived behavioral control were the most important factors in forming intentions. Women with more positive attitudes toward physical activity and who felt more in control of their ability to be active were more likely to intend to exercise. Intentions alone weren't enough. While intentions explained some differences in future activity, they didn't fully predict who stayed active, suggesting that other influences, like the impact of treatment, should be explored to better support long-term physical activity in breast cancer survivors.

The Cancer and Work Scale (CAWSE): A Validated Canadian Tool For Assessing Readiness, Success and Sustainability of Reintegration into the Workforce After Cancer - Poster #741

Presenter: Naomi Dolgoy, University of Alberta

Other Authors: Christine Maheu, Mina Singh, Wing-Lam Tock, Jennifer Robert, Andrea Vodermaier, Maureen Parkinson

ABSTRACT_ID#2659

ABSTRACT

Background: Workability following a cancer diagnosis and/or treatment has many known barriers to managing and sustaining employment. Returning to and sustaining employment after cancer presents significant challenges for individuals. In Canada, many approaches to rehabilitation, screening and assessment derive from international research and program development, requiring adaptations to use in a Canadian context. Prior to the validation of the Cancer and Work Scale (CAWSE), return to work (RTW) assessments used in cancer populations in Canada most often did not consider a Canadian content and/or lacked cancer-specific considerations, limiting clinical and occupational utility. **Purpose:** Our study developed and validated the CAWSE, a psychometrically robust Canadian-designed tool assessing RTW likelihood and employment sustainability in cancer populations, while also providing targeted intervention approaches. **Methods:** A two-phase cross-sectional study was conducted. Study I (n = 130) assessed content validity and construct development, leading to a refined 43-item CAWSE; study II (n = 216) utilized exploratory and confirmatory factor analyses in establishing structural validity, reliability, and responsiveness. Additional validation included correlations with cognitive difficulties, fatigue, anxiety and depression. **Results:** Factor analysis supported a seven-factor structure with 31 final items. Good internal consistency ($\alpha = 0.787$), construct validity, and moderate responsiveness (AUC = 0.659) were demonstrated. High sensitivity allowed for accurate identification of RTW difficulties, with an established cut-off score of 123.5 on the total CAWSE. **Implications:** The CAWSE fills a critical gap in cancer-specific vocational rehabilitation, offering Canadian and international healthcare providers a validated tool targeting interventions enhancing RTW outcomes and long-term employment sustainability.

LAY SUMMARY

There are significant barriers for people wanting to return to work and sustaining employment after a cancer diagnosis and/or treatment. Up until now, a major challenge in Canada was the lack of a Canadian and cancer-focused return to work assessment tool. This research will present the Cancer and Work Scale (CAWSE) which was tested and validated for use in assessing the likelihood of returning to work and the sustainability of employment in a cancer context. Our study used tested methods to develop an assessment that explores seven factors impacting work readiness and outcomes across 31 questions; the CAWSE fills a critical gap in cancer-specific work rehabilitation, offering Canadian and international healthcare providers a validated tool to use in assessing and intervention in cancer populations.

Identifying factors associated with fear of cancer recurrence - Poster #742

Presenter: Caroline Hovey, Dalhousie University

Other Authors: Robin Urquhart

ABSTRACT_ID#2695

ABSTRACT

Background: Fear of recurrence (FCR) is one of the most common unmet needs of cancer survivors and is increasing alongside the growing survivor population. Investigating factors that influence FCR is needed to identify who is most at risk and develop targeted strategies to support survivors. **Methods:** A secondary analysis of the Canadian Partnership Against Cancer's Experience of Cancer Patients in Transitions Study investigated three main objectives: (1) describe the prevalence of FCR in survivors by cancer type and survivor demographics; (2) investigate whether the provider of follow-up care is associated with the presence of FCR; and (3) investigate whether FCR is associated with access to specific resources. Associations between FCR and demographic, disease, and healthcare system characteristics were analyzed descriptively using quantitative statistical analyses (i.e., Chi squared tests). **Results:** Only 28% of the 13 319 survey participants reported FCR was not a concern. All but one demographic factor was found to have a statistically significant association with FCR; the largest differences were found in language, sex, age, and having children under eighteen. Those with no follow-up care provider reported the least FCR (29.0%), followed by family doctor (32.8%), cancer specialist (35.5%), and both (39.0%). Survivors who received support from additional providers and/or social media reported the most FCR. **Conclusion:** In addition to supporting previous research, these results identify survivors who are most at risk of experiencing FCR. Further, this investigation provides the basis for research into specific areas of follow-up care and strategies to support this unmet need.

LAY SUMMARY

- Most demographic factors produced a statistically significant association with fear of cancer recurrence (FCR); however, not all these associations may be large enough to produce a meaningful difference in a clinical setting. The largest associations were found for language, sex, age, dependent children, cancer type, metastatic disease, and treatment type. Some significant associations, such as rurality, level of education, and income, did not produce expected results.
- Respondents who received follow-up care from both a primary care provider and a cancer specialist showed the highest FCR. All resource-related factors were associated with FCR. However, those that produced the largest magnitude of difference were access to additional providers, difficulty accessing information, social media, and support groups. Although no causal relationships can be determined, the higher level of FCR in those seeing additional providers indicates a gap in the current system of follow-up in which survivors must search for support elsewhere.
- This investigation was able to provide insight into possible risk factors for experiencing FCR without having to perform costly data collection methods and impede on the lives of survivors. New investigations into factors associated with FCR should include thoughtful survey design including a validated measure of FCR.

Cancer Survivorship Models of Care: An Urgent Need for Better Solutions - Poster #743

Presenter: Doris Howell, Princess Margaret Cancer Research Institute

Other Authors: Wojtek Wiercioch, Homa Keshavarz, Usman Ali, Luis Enrique Colunga-Lozano, Amy Bustaman

ABSTRACT_ID#3244

ABSTRACT

Background: Post-treatment cancer survivorship follow-up (FU) models of care rely heavily on oncologist specialist-led care, which is unsustainable given the growing number of survivors. Shared care, nurse-led, primary-led, and patient led/initiated FU models of survivorship care could be appropriate, but effect comparisons are lacking. **Methods:** We conducted a systematic review and meta-analysis of RCTs comparing oncologist-specialist led-care (usual care) with these models of care on clinical outcomes (time to recurrence, detection of new cancers, mortality) and quality of life outcomes, pain, fatigue, emotional distress/depression and patient satisfaction. For continuous outcomes of interest, we used the change from baseline to immediate post-treatment and over time (mean change score, SD). DerSimonian and Laird random effects models with inverse variance methods were used to generate mean difference (MD) and standardized mean difference (SMD) for continuous outcomes, and risk ratio (RR) for dichotomous outcomes. **Findings:** 36 unique RCTs were included in meta-analysis. Breast cancer was most common (included in 18 studies, 50%), followed by colorectal and colon cancer (n=8, 22%), and prostate cancer (n=4, 11%). Shared care, primary care-led, nurse-led care were closely equivalent to oncologist specialist led-care with trivial to small effect differences for outcomes. Evidence for patient-led/patient-initiated models of care was less certain. **Conclusion:** As the population of cancer survivors continue to grow, there is a need to consider models of FU care that extend the role of nurses and primary care physicians to ensure equity of access to quality survivorship care that is personalized to risk and needs.

LAY SUMMARY

Dominant models of oncologist specialist led-care are not sustainable and personalized models of care that leverage alternate models of care such as shared-care, nurse-led and primary care-led may be relevant and appropriate for large number of adult post-treatment survivors. We conducted a systematic literature review to identify effectiveness of these alternate models of survivorship care (shared-care, nurse-led and primary care-led) on a range of clinical and quality of life outcomes with a goal to identify pan Canadian evidence-based recommendation on use of these alternate models for follow-up of adults post-cancer treatment. Small or trivial differences were shown for shared-care, nurse-led and primary care led models of adult post-treatment survivorship follow-up care in comparison to oncologist specialist-led care on clinical outcomes (time to detection of recurrence or new cancers, mortality), quality of life outcomes and patient satisfaction. Further research is needed to evaluate best practices for evaluating implementation and effectiveness of these alternate models of care and specifically patient-led/patient initiated models of care and to action improvements in self-management support as a core element. A national framework for implementation that enhances the role of primary care physicians, nurses, and survivors is needed and that ensures equitable access to survivorship care.

Physical Activity Interventions for Pain Management of Aromatase Inhibitor Induced Musculoskeletal Syndrome: A Systematic Review - Poster #744

Presenter: Jenna Sim, University of Calgary

Other Authors: Khloe Bedore, Elaine Gentleman, Imrose Bhullar, Hannah Cripps, Julia Daun, Jonathan Low, Arjun Ghuman, David Langelier, S. Nicole Culos-Reed, Lauren Capozzi

ABSTRACT_ID#3129

ABSTRACT

Introduction: Aromatase inhibitors (AIs) are a highly effective adjuvant therapy for hormone receptor positive breast cancer. A common side effect includes AI-induced musculoskeletal syndrome (AIMSS), which includes primarily myalgias and arthralgias. Many who experience AIMSS stop or pause treatment, increasing risk of recurrence. Physical activity (PA) can help manage many cancer-related side effects, however its effect on AIMSS is less known. Thus, this review aimed to synthesize the evidence on PA interventions for AIMSS, with a primary focus on AIMSS-related pain. Methods: MEDLINE, CHAHL, CENTRAL, and Embase were systematically searched for PA interventions for AIMSS. Titles, abstracts, and full-texts were screened in duplicate. Extraction was completed independently and captured intervention and participant characteristics, AIMSS symptoms, and risk of bias. Results were analyzed via narrative synthesis and harvest plots. Results: The search identified 3148 unique articles, from which 15 reported on PA interventions. Interventions included aerobic exercise (n=4), combined aerobic and resistance exercise (n=5), yoga (n=4), and Tai Chi (n=2). Clinically or statistically significant improvements in AIMMS-related pain were reported in 73% (n=11) interventions. Nine studies measured AIMMS-related pain directly (e.g., Brief Pain Inventory, visual analog scale), while two measured AIMMS-related pain indirectly via quality-of-life measures. Conclusions: Results of this review are promising; PA may reduce AIMSS-related pain. However, the extent of the literature is still relatively limited, thus it is difficult to draw strong conclusions. Future research should consider conducting high-quality intervention studies to determine the ideal components of PA interventions (e.g., frequency, intensity, time, type) to reduce AIMSS-related pain.

Incidence of subsequent primary cancers among survivors of hematopoietic cell transplantation identified using administrative data in Alberta, Canada - Poster #745

Presenter: Dylan E. O'Sullivan, Cancer Care Alberta, Alberta Health Services

Other Authors: Son Tran, Sanjeev Bista, Kareem Jamani

ABSTRACT_ID#3006

ABSTRACT

In Alberta, the use of allogeneic (allo) and autologous (auto) hematopoietic cell transplantation (HCT) in the treatment of high-risk hematologic and some solid malignancies began in the 1980s and has steadily increased. While the demand for HCT continues to rise in Alberta, and long-term survival is becoming increasingly more common among recipients, the risk of developing subsequent primary cancers (SPCs) after receiving HCT remains a significant contributor to morbidity, mortality and healthcare resource utilization among this unique survivorship population. Cancer screening guidelines exist for survivors of allo and auto-HCT; however, they are often based on data from non-HCT survivorship populations, often do not differentiate between auto and allo-HCT recommendations, and sometimes defer to general population screening guidelines. To fully understand the epidemiology of SPCs among allo-/auto-HCT survivors in Alberta, we used data from the Alberta Cancer Registry to identify all first and subsequent primary cancers for patients who had underwent HCT between 2001 and 2019 (using the DAD and NACRS administrative data sets) and survived for at least six months following their procedure. With follow-up until the end of 2023, 19.4% of HCT survivors developed an SPC (75% solid including non-melanoma skin cancer and 25% hematologic). HCT patients who developed an SPC had a significantly greater overall mortality rate compared to patients who did not develop an SPC. This work-in-progress aims to generate key evidence and fill-in data gaps to support future opportunities to enhance and direct appropriate screening/surveillance among a high-risk survivorship population.

LAY SUMMARY

While the demand for hematopoietic cell transplantation (HCT) continues to rise in Alberta, and long-term survival is becoming more common among recipients, the risk of developing another primary cancer within their lifetime after receiving HCT is a significant contributor to morbidity and mortality in this population. To understand how often this occurs for HCT survivors in Alberta, we retrospectively identified cancer cases using data from the Alberta Cancer Registry and searched administrative data sets from Alberta Health Services to capture whether and when the cancer cases underwent HCT, and if any of these cases occurred after the procedure. For patients in Alberta with an initial primary cancer diagnosis and had received an HCT, the overall risk of developing another primary cancer in their lifetime was 19.4% based on our preliminary findings. This work-in-progress aims to generate key evidence to support future opportunities to enhance and direct appropriate screening/surveillance among a high-risk survivorship population.

The Cancer Rehabilitation and Exercise – Advanced Cancer (CaRE-AC) Program: A Qualitative Exploration of Intervention Delivery Challenges, Successes, and Sustainability - Poster #746

Presenter: Kelcey Bland, University of British Columbia

Other Authors: Janna Griffioen, Grace Schouten, David Langelier, Helen McTaggart-Cowan, Sarah Neil-Sztramko, Kelly Mackenzie, Amrah Ahamed, Anthony Reiman, Jonathan Greenland, Jennifer Jones, Kristin Campbell

ABSTRACT_ID#3193

ABSTRACT

Introduction: Treatment advances have extended cancer survival, increasing the number of individuals living with advanced cancer. However, treatment can impair physical function and quality of life, making rehabilitation and exercise critical. The Cancer Rehabilitation and Exercise – Advanced Cancer (CaRE-AC) program offers exercise and education sessions tailored for advanced cancer. To foster ongoing success and scalability, barriers and facilitators to CaRE-AC delivery were explored. **Methods:** Semi-structured interviews were conducted with people with advanced cancer who participated in CaRE-AC (n=20) and healthcare providers involved in recruitment and intervention delivery (n=10). Transcripts were inductively coded using conventional content analysis and mapped onto the Consolidated Framework of Implementation Research domains (innovation, inner setting, outer setting, individuals, implementation process) to identify initial themes. **Results:** The innovation domain emphasized person-centered care through adaptability to patients' diverse needs. At the individual level, engagement was influenced by personal barriers (e.g., symptom burden), exercise self-efficacy, and positive provider experiences. The implementation process showed strengths in assessment delivery, structured education, and practitioner enthusiasm, with barriers including unclear recruitment, operational and communication logistics, and a lack of cohesion between education and exercise sessions. A supportive inner setting fostered holistic change, including trust in exercise abilities and validation of shared experiences. Outer setting barriers included limited resource awareness, technology access, and health events. Facilitators included sustained funding, long-term support, and continued connection post-intervention. **Conclusion:** Exploring patient and healthcare provider perspectives revealed factors influencing CaRE-AC implementation, from individual engagement barriers to system-level challenges. Insights generated may help refine and scale the CaRE-AC program.

LAY SUMMARY

1. Rehabilitation and exercise are important for individuals with advanced cancer: While treatment has extended cancer survival, it can also impact physical function and quality of life among people living with advanced cancer. The CaRE-AC program provides tailored exercise and education sessions to support patients in managing these challenges.
2. Understanding barriers and facilitators helps improve program delivery: Interviews with patients and healthcare providers revealed key factors influencing participation, including personal challenges like symptom burden, the importance of provider support, and logistical barriers, such as recruitment and session cohesion.
3. Insights from implementation research can enhance scalability: Identifying strengths and challenges in CaRE-AC delivery, such as sustained funding, trust-building, and continued patient engagement, may help refine the program and expand access to more individuals living with advanced cancer.

Predictors of Response to Nighttime Compression at Six-Month Follow-up in Individuals with Breast Cancer related Lymphedema - Poster #747

Presenter: Badr El-Bakkali El-Kasmi, University of Alberta

Other Authors: Mona Al Onazi, Naomi Dolgoy, Lori Radke, Margaret McNeely

ABSTRACT_ID#2763

ABSTRACT

Background: Breast cancer-related lymphedema is a common long-term consequence of treatment that diminishes quality of life. Observational evidence suggests that the application of night-time compression supports better long-term control of arm swelling. A parallel three-arm, multi-centre, fast-track randomized trial evaluated: (1) day-time compression garment alone (standard care), (2) day-time garment combined with night-time compression bandaging, and (3) day-time garment combined with a night-time compression system garment. Both night-time strategies produced significant short-term volume reductions. During follow-up (weeks 13–24), all participants used a night-time compression system garment. Objective: A secondary analysis was conducted to identify factors associated with a positive response to night-time compression at the 24-week follow-up of the randomized controlled trial. Methods: A positive response was defined as >10% reduction in lymphedema volume from baseline to week 24. Uni- and multivariable logistic regression examined associations between key medical, clinical, and lymphedema-specific variables and response to night-time compression. Results: Of 120 enrolled women, 118 (98%) completed the randomized trial and 114 (95%) completed the 24-week follow-up. Mean percent volume reduction from baseline to week 24 did not differ between groups (night-time compression system garment 16.5%; compression bandaging 13.4%; standard care 13.9%; $p = 0.85$). Sixty-two participants (54%) were classified as positive responders. Baseline mobility, lymphedema severity, and sleep quality were independently associated with treatment response. Conclusions: Women with mild lymphedema who had better sleep quality and mobility were significantly more likely to benefit from the addition of night-time compression during the maintenance phase of lymphedema treatment.

LAY SUMMARY

Nighttime compression can help reduce arm swelling in many women with breast cancer-related lymphedema when used along with standard daytime compression garments. Women with milder lymphedema, better sleep quality, and better mobility were more likely to experience meaningful improvements with nighttime compression. These findings can help personalize lymphedema care, allowing clinicians to better identify which patients may benefit most from adding nighttime compression during long-term treatment.

Advancing Exercise Recommendations for Bone Metastases: Evaluating the Uptake of Co-Designed Knowledge Mobilization Products - Poster #748

Presenter: Kelcey Bland, University of British Columbia

Other Authors: Sarah Neil-Sztramko, Michelle Nadler, Alana Chalmers, Rhoda Dinardo, Titilola Yakubu, David Langelier, Lauren Capozzi, Kirstin Lane, Shabbir Alibhai, Kelly Mackenzie, Karen McDonald, Jane Copp, Janet Papadacos, Margaret McNeely, Nicole Prestley, Sarah Weller, Leah K. Lambert, Kristin L. Campbell

ABSTRACT_ID#3166

ABSTRACT

Introduction: Exercise for individuals with bone metastases was once cautioned against due to risks (e.g., bone fractures), but evidence-based recommendations emphasize its benefits when tailored appropriately. Integrating published recommendations into clinical practice remains challenging. To bridge this gap, we developed knowledge products and dissemination strategies with patients, healthcare providers, and exercise professionals using experience-based co-design. **This study aimed to evaluate knowledge product uptake.** **Methods:** Twenty-nine knowledge users across Canada co-designed knowledge products (a patient handout, educational videos, health information form). Final products were graphic designed with the Canadian Cancer Society (CCS) and launched on the Bone Metastases and Exercise (BME) Hub (bit.ly/BMEhub) and CCS website (cancer.ca) in May 2024. A broad dissemination plan utilized partner organization outreach and social media, with a six-month evaluation via Google Analytics. **Results:** Dissemination involved 9 email blasts/newsletters from partner organizations, reaching approximately 7,731 recipients. Social media promotion, jointly tagged with CCS, generated 647 engagements, 34,104 impressions, a 1.9% engagement rate per impression, and 116 link clicks across Instagram, X, Facebook, and LinkedIn over two weeks. The BME Hub had 5,242 views and 2,413 active users in month one and averaged 294±74 active users and 700±147 page views per month thereafter. Resource engagement included 205 and 841 downloads of the health information form and patient handout, respectively. The patient education webinar accumulated 706 YouTube views. **Conclusions:** Knowledge product dissemination has enhanced accessibility and engagement with exercise recommendations for individuals with bone metastases. Continued dissemination and evaluation efforts aim to support sustained integration into clinical practice.

LAY SUMMARY

1. Exercise is beneficial for individuals with bone metastases: While there were past concerns about potential risks, such as bone fractures, research now shows that exercise can be safe, feasible, and beneficial for people living with bone metastases. The challenge is ensuring oncology healthcare providers, community-based exercise professionals, and patients can confidently apply these recommendations in practice.
2. Co-designed resources improve access to evidence-based exercise guidance: Patients, healthcare providers, and exercise professionals collaborated to create educational resources, such as patient handouts, videos, and health information forms, to increase the uptake and use of published exercise recommendations for people with bone metastases. These resources were made available through the Bone Metastases Exercise Hub (<https://www.cancerexercise.med.ubc.ca/bmehub/>) and the Canadian Cancer Society (<https://www.cancer.ca>) and promoted through other trusted oncology and exercise organizations to ensure wider accessibility.
3. Strong engagement suggests successful dissemination: Outreach efforts, including emails, social media campaigns, and website promotions, helped people find and use our co-designed resources. Data shows that thousands have engaged with the materials, and ongoing efforts aim to evaluate and integrate exercise recommendations more effectively into patient care.

The Alberta Moving Beyond Breast Cancer (AMBER) Cohort Study: Completion of Data Collection and Knowledge Translation Activities - Poster #750

Presenter: Leanne Dickau, Alberta Health Services

Other Authors: Christine M. Friedenreich, Lin Yang, Renée L. Kokts-Porietis, Qinggang Wang, Jeff K. Vallance, Margaret L. McNeely, S. Nicole Culos-Reed, Charles E. Matthews, Jessica McNeil, Andria R. Morielli, Kerry S. Courneya

ABSTRACT_ID#2912

ABSTRACT

Background: The AMBER study examines the role of physical activity (PA), sedentary behaviour (SB) and health-related fitness (HRF) in breast cancer survival. We describe the completion of our data collection and the completed and planned knowledge translation activities. **Methods:** We recruited 1,528 newly diagnosed stage I ($\geq T1c$) to IIIC breast cancer patients between July 2012 and July 2019. The following objective measurements were completed at baseline, 1- and 3-years post-diagnosis: blood draw, HRF measurements, lymphedema assessments and PA and SB using self-report and accelerometry. Questionnaires were also completed at these time points and at 5-years to collect patient characteristics, dietary intake, patient-reported outcomes, and barriers to PA. Medical chart abstractions are ongoing to capture treatment and outcomes after breast cancer for an additional 5-years. **Results:** Excellent retention was obtained with 1,353, 1,174 and 1069 participants completing years 1-, 3- and 5-assessments. The median follow-up to date is 9.3 years. To date, eight manuscripts have examined the baseline characteristics of the study population and changes from baseline to year 1. Longitudinal and survival analyses are planned to examine changes over time as well as the associations between PA, SB and HRF with survival. Retention in the cohort was enhanced by regular communication with participants through quarterly newsletters regarding study progress. **Conclusion:** This long-term cohort study that included direct measurements of PA, SB, and HRF, as well as comprehensive questionnaires, has achieved excellent retention and has already provided valuable insights on how PA, SB and HRF are impacted in breast cancer survivors.

LAY SUMMARY

Previous research, based on self-report measures, has suggested that physical activity can improve survival and patient-reported outcomes after breast cancer. The AMBER study was designed to incorporate objective measurements of physical activity, sedentary behaviour and health-related fitness to overcome some of the limitations of previous research. The AMBER study, with over 1500 newly diagnosed breast cancer patients, is one of the largest cohort studies with 7-day, device based physical activity measures in breast cancer patients. Participants were asked to complete comprehensive assessments of health-related fitness testing at baseline, 1-year and 3-year timepoints using gold-standard measurements for aerobic fitness, strength, and body composition. Despite the requirements to complete these objective measurements, in addition to providing blood samples and completing a set of questionnaires, excellent retention rates of 89% at year 1, 79% at year 3 and 74% at year 5 were achieved. Knowledge translation activities continue to keep participants informed on study progress, such as quarterly newsletters that include details of study publications and presentations. Each participant is followed for at least 10 years post-diagnosis with target completion by 2028/9 when the long-term impact of physical activity, sedentary behaviour and fitness on breast cancer outcomes and survival will be analyzed.

The role of fatty acid binding protein 4 and saturated fatty acids in cancer-related lymphedema progression - Poster #751

Presenter: Jacob Korodimas, University of Alberta

Other Authors: Karina Gomes, Xiaoyan Yang, Spencer Gibson

ABSTRACT_ID#2609

ABSTRACT

Cancer-related lymphedema (CRL) is a chronic, progressive condition characterized by lymphatic fluid accumulation and limb swelling, causing pain, fibrosis, adipose deposition, infections, and diminished quality of life—yet no curative treatment exists. Most lymphedema cases are secondary to cancer (most commonly breast cancer) or obesity. Fatty acid binding protein 4 (FABP4), an intracellular lipid chaperone, is elevated in obesity, breast cancer, and is associated with inflammation and metabolic dysregulation. Given links to obesity, late-stage fat accumulation in lymphedema, and FABP4's upregulation in related conditions, we investigated how saturated fatty acids (SFAs) contribute to lymphatic injury and whether FABP4 mediates these effects. We hypothesize that SFAs induce lipotoxicity in lymphatic endothelial cells by triggering endoplasmic reticulum (ER) stress, a process mediated by FABP4. Human dermal lymphatic (HDLECs), dermal microvascular (HDMECs), and umbilical vein endothelial cells (HUVECs) were treated with stearic and palmitic acid, then evaluated for cell death, oxidative stress, and ER stress using trypan blue staining, immunoblotting, and flow cytometry. SFA treatment caused greater cell death and oxidative stress in HDLECs compared with HUVECs and HDMECs. FABP4 expression was highest in HDLECs; knockdown or pharmacological inhibition decreased stearic acid-induced cell death, and apoptotic and ER stress protein expression. Additionally, we employed an endothelial-specific FABP4 knockout mouse model (C57BL/6-; Tek-Cre; hFABP4^{flox/flox}), surgically induced tail lymphedema, and monitored swelling over 28 days. Preliminary data show reduced tail swelling in knockout versus wild-type females. These findings suggest FABP4 mediates SFA-induced ER stress in lymphatic endothelia and modulating FABP4 activity may alter CRL progression.

LAY SUMMARY

Saturated fatty acids cause stress to the cells lining the human lymphatic system. Lymphatic cells treated with saturated fatty acids showed more stress and cell death compared to vascular cells. A protein called fatty acid binding protein 4 is implicated in lymphatic cell responses to saturated fatty acids. More of this protein was present in lymphatic cells compared to vascular cells, and when it was inhibited or knocked-down, saturated fatty acids caused less cell stress and cell death. Targeting fatty acid binding protein 4 may help reduce swelling in cancer-related lymphedema. From our preliminary studies, female mice lacking endothelial fatty acid binding protein 4 showed reduced swelling in a surgically-induced lymphedema model compared to controls.

Depressive symptoms and their associated factors among middle-aged and older adults in Canada ever diagnosed with cancer: Results from a population-based cross-sectional study - Poster #753

Presenter: Md Abdullah Al Jubayer Biswas, School of Public Health, University of Saskatchewan

Other Authors: Scott J. Adams, Prosanta Mondal, Lilian Thorpe, Punam Pahwa

ABSTRACT_ID#2602

ABSTRACT

Background: Depression significantly affects cancer patients' health, particularly middle-aged and older individuals who face the combined burden of cancer-related stress and the challenges of aging. While prior studies have explored depression in cancer patients, there is a lack of research on the prevalence of depressive symptoms in individuals aged 45 and older in Canada who have ever been diagnosed with cancer, as well as factors that increase the risk of these symptoms. Methods: We analyzed data from the 2015-2016 and 2017-2018 Canadian Community Health Surveys to assess depressive symptoms using the Patient Health Questionnaire-9. A minimal adjustment set of covariates was determined through directed acyclic graphs. We estimated prevalence ratios with Poisson regression accounting for (i) unequal probability of selection via weights provided by Statistics Canada, and (ii) design effects (stratification and clustering) via the Taylor linearization technique. Results: Nearly 60% of individuals 45 and older ever diagnosed with cancer reported depressive symptoms. Higher likelihood of experiencing depressive symptoms was associated with female gender, those with poor self-rated health, significant life stress, limited physical activity, a history of suicidal thoughts, a history of illicit drug use, and sleep difficulties. Additionally, using mental health services in the past 12 months was associated with a higher prevalence of depressive symptoms. Conclusion: Our study found a high prevalence of depressive symptoms among middle-aged and older adults ever been diagnosed with cancer. Our findings suggest that enhancing psychosocial screening within oncology care could help address unmet mental health needs in this population in Canada.

LAY SUMMARY

1. Depression affects nearly six in ten individuals aged 45 and older in Canada who have ever been diagnosed with cancer, making it a significant public health concern.
2. Certain groups are more at risk, including women, those with a female gender, those with poor self-rated health, those with significant life stress, those with limited physical activity, those with a history of suicidal thoughts, those with a history of illicit drug use, and those with sleep difficulties.
3. Many affected individuals already seek help, but existing services fall short. By improving mental health screening and expanding access to community-based support, especially for low-income and older adults, we can deliver more equitable and effective cancer care.

Investigating the Role of SFRP4 in Breast Cancer-Related Lymphedema - Poster #754

Presenter: Mandi Ma, University Health Network

Other Authors: Jeffrey To, Rada Tazhitdinova, Jennifer Kwan

ABSTRACT_ID#2537

ABSTRACT

Background: Breast cancer treatments, such as surgery or radiotherapy, can damage the lymphatic system, causing lymphedema in about 20% of breast cancer survivors. Risk factors for developing lymphedema include adiposity metrics such as elevated body mass index (BMI) and low-density (high adiposity) breast tissue. However, while these risk factors have been established in the literature, there is a poor understanding of the underlying biological mechanism of action. Previous single-cell RNA-sequencing of breast tissue revealed a significant elevation in gene expression of secreted frizzled-related protein 4 (SFRP4) in low-density breast tissue. Thus, this study aims to investigate the potential role of SFRP4 in contributing to an increased risk of lymphedema development. Methods: Human dermal lymphatic endothelial cells (hDLEC) were treated with increasing concentrations of SFRP4 protein. Subsequently, mRNA expression of genes related to lymphatic proliferation and vascular formation was evaluated using real-time qPCR. IncuCyte was used to measure cell proliferation. One-way and two-way ANOVAs with relevant post-hoc statistical testing were completed. A p-value of <0.05 was considered significant. Results: qPCR results revealed a dose-dependent increase in SPARC mRNA expression 48 hours after SFRP4 treatment ($n=3$, $p<0.05$). Preliminary data suggests a potential differential dose-response in proliferative ability 7 days after SFRP4 treatment ($n=3$, $p<0.05$). Conclusion: SFRP4 increases SPARC gene expression in hDLECs. hDLEC proliferation is also altered after treatment with SFRP4. Additional research will examine the relationship between SFRP4 and SPARC in lymphatic endothelial cell proliferation.

LAY SUMMARY

Secreted Frizzled-Related Protein 4 (SFRP4) may be associated with increased risk of breast cancer-related lymphedema. SFRP4 may contribute to changes in cell growth of human dermal lymphatic endothelial cells.

The KAPS-C Study: exploring Knowledge and Attitudes towards Probiotic Supplement use in people with Cancer – Preliminary Findings - Poster #755

Presenter: Julie Deleemans, University of Calgary

Other Authors: Muskaan Pamery, Linda Carlson

ABSTRACT_ID#2416

ABSTRACT

Background: People with cancer express growing interest in using probiotic supplements to manage symptoms. Yet research examining safety, effectiveness, and appropriate use within cancer care is scarce. This survey aimed to explore knowledge, attitudes, perceived benefits and risks toward probiotic use in cancer care. Methods: An anonymous, online, cross-sectional survey was administered to adults in Canada with cancer. Data on demographic and clinical outcomes, probiotic use, beliefs, knowledge, and patient reported outcomes for gastrointestinal (GI), anxiety and depression symptoms were collected. Descriptive and frequency analyses are reported. Results: Data collection is ongoing; 55 participants were included in this analysis. Elevated symptoms included constipation (M=75), belly pain (M=54), depression (M=54) and anxiety (M=55). 78% considered using probiotics; 53% reported recent use. Most reported low to moderate levels of confidence in probiotic knowledge (73%) and selection (58.2). Motivation for probiotic use included addressing GI symptoms (71%) and improving general health (73%). Half (49%) felt probiotics were useful at any time, with 58% endorsing safety post-treatment. Perceived benefits included improving GI health (73%), mental health (40%), digestion (60%), and immune function (38%). Perceived risks included drug interactions (30%) and side effects (24%). Preferred sources of information included oncologists (67%), nurses (72%), family doctor (71%), and dieticians (66%). Yet, only 56% felt they could speak to their doctor about probiotic use. Conclusion: People express interest and positive beliefs about potential benefits of probiotic use in cancer care. Enhancing patient and provider education is critical to support safe, evidence-informed probiotic use in oncology.

LAY SUMMARY

Key Insights:

1. Understanding attitudes and beliefs around probiotic supplement use in people with cancer - how do patients feel about probiotics and where do they get information from?
2. Perceived benefits and risks about probiotic use in cancer care - why are patients interested in using probiotics? What symptoms are they trying to address?
3. Sources of information on probiotics - where do patients get their information from? Where are the gaps in education for patients and health care providers?

Risk of developing a subsequent primary lung cancer among adult non-lung cancer survivors - Poster #756

Presenter: Matthew T. Warkentin, University of Calgary

Other Authors: Yibing Ruan, Darren Brenner, Alain Tremblay, Dylan O'Sullivan

ABSTRACT_ID#2995

ABSTRACT

Background: There are an increasing number of people living with and beyond a cancer diagnosis. Cancer survivors may carry elevated risks of developing subsequent primary cancers at different sites. We aim to characterize the risk of developing a subsequent primary lung cancer (SPLC) among survivors of first primary cancers (FPC). **Methods:** This study included all adults diagnosed with cancer in the Alberta Cancer Registry between 2000 and 2021 who survived at least six months. Survivors were followed from their FPC diagnosis until diagnosis of a SPLC, death, or administrative censoring. We estimated incidence rates and standardized incidence ratios (SIR) for SPLC development. SIR was the ratio of the observed to the expected number of SPLC. **Results:** There were 180,325 FPC survivors with 1.2 million person-years of follow-up who developed 2,865 SPLC. Cancer survivors had a higher risk of developing SPLC compared to those without a personal cancer history (SIR=1.37, 95% CI: 1.32-1.42). Thirteen out of twenty-one (62%) FPC sites had significant increases in SPLC risk and tobacco-associated FPC carried the highest risk of SPLC. When standardized to the age and sex distribution of a major lung cancer screening trial, five FPC had higher lung cancer incidence rates than the trial control arm. **Conclusion:** Cancer survivors carry a considerable risk of developing a SPLC after surviving an initial non-lung cancer, likely due to genetic susceptibility, shared exposures, and treatment-related sequelae. Survivors of some FPC have risks comparable to those eligible for lung cancer screening and may benefit from early detection initiatives.

LAY SUMMARY

There are a growing number of cancer survivors in Canada who may be at risk for developing another cancer at a different location. This study aimed to characterize the risk of developing lung cancer after surviving an initial non-lung cancer. People that have survived an initial non-lung cancer carry a risk of getting lung cancer that is considerably higher than people who have never had any cancer before. Thirteen out of the twenty-one cancer sites we investigated in this study carried an elevated risk of developing lung cancer. Some cancer survivors have a lung cancer risk comparable to people who were included in a major US lung cancer screening trial for high-risk individuals that were selected based on their age and heavy smoking history. This finding suggests that some cancer survivors may benefit from early lung cancer detection initiatives.

Prevalence of biopsychosocial symptoms among Head and Neck Cancer survivors: a systematic review and meta-analysis - Poster #757

Presenter: Lais Renata Cezario Santos, Faculty of Nursing, University of Calgary

Other Authors: Vagner Deuel Tavares, Colleen Cuthbert

ABSTRACT_ID#2411

ABSTRACT

Background: Although common, the prevalence and patterns of key biopsychosocial late effects in head and neck cancer (HNC) survivors are not well documented. **Objective:** This systematic review and meta-analysis aimed to identify the prevalence of biopsychosocial symptoms among HNC survivors. **Methods:** A systematic search and meta-analysis was conducted in Medline, Web of Science, Embase, PsycINFO, and CINAHL via OVID. Studies reporting prevalence rates of biopsychosocial symptoms after HNC treatment were included. Screening and risk of bias assessment were performed independently. Prevalence rates were pooled in a meta-analysis, and meta-regression was used to explore heterogeneity. **Results:** Of 5,993 studies, 56 met inclusion criteria and 43 were included in the meta-analysis. The overall prevalence of biopsychosocial symptoms was 28% (95% CI: 0.25–0.30), with high heterogeneity ($I^2 = 96\%$). Meta-regression identified sex as a significant covariate ($p = 0.00$). Among symptom types, physical symptoms were most prevalent (42%, 95% CI: 0.36–0.47), followed by social symptoms (26%, 95% CI: 0.19–0.32) and mental health symptoms (20%, 95% CI: 0.17–0.23). According to time since treatment, prevalence was 23% within 3 months (95% CI: 0.11–0.41), 24% between 3–12 months (95% CI: 0.16–0.34), and 20% after 12 months (95% CI: 0.17–0.23). **Conclusion:** Biopsychosocial symptoms affect over a quarter of HNC survivors (28%) and persist up to 1 year post treatment, with physical symptoms being most common highlighting the need for targeted supportive care in this population.

LAY SUMMARY

Biopsychosocial symptoms are common in HNC survivors: About 28% of people who survive head and neck cancer continue to experience physical, social, or mental health issues after treatment. These symptoms can last for up to a year or more. Physical symptoms are the most common: Among all types, physical problems (42%)—like pain, fatigue, or swallowing difficulties—are reported more often than social (26%) or emotional/mental health symptoms (20%). Sex matters: The results showed that gender influenced symptom levels, with women more likely to report higher symptom prevalence than men.

Risk of developing a primary lung cancer among women with a history of pre-menopausal or post-menopausal breast cancer - Poster #758

Presenter: Matthew T. Warkentin, University of Calgary

Other Authors: Riley Ngo, Yibing Ruan, Alain Tremblay, Darren Brenner, Dylan O'Sullivan

ABSTRACT_ID#2999

ABSTRACT

Background: In Canada, breast cancer is the most common cancer among women, and lung cancer is the leading cause of cancer death. Several studies have shown an increased risk of subsequent primary lung cancer (SPLC) among survivors of primary breast cancer (BC). However, less is known about the risk among survivors of early-onset (i.e., pre-menopausal) BC. We aim to characterize the risk of developing an SPLC among pre- and post-menopausal BC survivors in a Canadian context. **Methods:** We used data from the Alberta Cancer Registry to identify all adult (age ≥ 18) women with first primary BC diagnosed between 2000 and 2021 who survived at least 6 months. Survivors were followed from their BC diagnosis until SPLC diagnosis, death, or administrative censoring. We estimated incidence rates and standardized incidence ratios (SIR) for SPLC development. SIR was the ratio of observed to expected numbers of SPLC. **Results:** There were 9,330 and 27,173 pre-menopausal (age ≤ 50) and post-menopausal (age 50+) BC survivors with 79K and 196K person-years of follow-up who developed 54 and 494 SPLC, respectively. The incidence rate of SPLC for pre- and post-menopausal BC were 68.1 and 252.5 per 100,000, respectively. The relative risk of SPLC compared to the cancer-free population was 2.11 (95% CI: 1.58 to 2.71) and 1.37 (95% CI: 1.25 to 1.49) for pre- and post-menopausal BC survivors, respectively. **Conclusion:** Pre- and post-menopausal BC survivors carry a considerable risk of developing a SPLC. We have begun investigations to identify risk factors for SPLC development among younger BC survivors.

LAY SUMMARY

There is some evidence that women who have survived an initial breast cancer may carry an elevated risk for developing a subsequent primary lung cancer. However, most women in these previous studies were diagnosed with breast cancer after the age of 50 and much less is known about the risk profiles for those who have survived early-onset breast cancer (<50). Survivors of both pre-menopausal and post-menopausal breast cancer carry elevated risk for developing a subsequent primary lung cancer compared to the cancer free population. Future studies are needed to determine whether some breast cancer survivors could benefit from early lung cancer detection initiatives. Our team has begun investigations to identify the important risk factors, including treatment-related factors, that may contribute to the development of lung cancer among breast cancer survivors. These factors may help to predict which breast cancer survivors are at the highest risk of developing lung cancer.

Associations of sleep with quality of life among women with newly diagnosed breast cancer: baseline results from the AMBER cohort study - Poster #759

Presenter: Lin Yang, Alberta Health Services

Other Authors: Temoor Tayyab, Reneé Kokts-Porietis, Qinggang Wang, Jessica McNeil, Charles Matthews, Leanne Dickau, Jeff Vallance, Margaret McNeely, S. Nicole Culos-Reed, Karen Kopciuk, Kerry Courneya, Christine Friedenreich

ABSTRACT_ID#2660

ABSTRACT

Background Sleep problems are common among breast cancer survivors and are associated with poor quality of life. Yet, few studies have accounted for depression. This study examines whether depressive symptoms attenuate the association between sleep and quality of life in newly diagnosed breast cancer survivors. Methods Women with newly diagnosed early-stage breast cancer were recruited between 2012-2019 in Alberta, Canada, and completed the Pittsburgh Sleep Quality Index (PSQI) to assess global sleep quality. Quality of life was measured using the Short Form Survey (SF-36 version-2) to assess physical and mental well-being. Depressive symptoms were measured using Patient Health Questionnaire (PHQ-9). Multivariable linear regressions were used to estimate the association of sleep measures with physical and mental well-being. Depressive symptoms were evaluated as a potential confounder and effect modifier. Results Among 1454 breast cancer survivors with available data, 43% reported poor global sleep quality and 10.5% reported clinically meaningful depressive symptoms. Poor sleep quality was associated with lower physical ($\beta=-3.0$, 95%CI: -3.8 to -2.3) and mental wellbeing ($\beta=-5.7$, 95%CI: -6.7 to -4.7). These associations were attenuated to ($\beta=-2.3$, 95%CI: -3.1 to 1.5) and ($\beta=-1.2$, 95%CI: -2.1 to -0.3) after further adjusting for depression symptoms. Stratified analyses showed slightly stronger association in women with non-minimal symptoms, though this association was not clinically meaningful. Conclusion The association between sleep and quality of life was substantially attenuated after accounting for depressive symptoms. Longitudinal data are needed to clarify the temporal relationship and determine whether sleep can serve as an early indicator of emerging depression.

LAY SUMMARY

Sleep problems are common. In the Alberta Moving Beyond Breast Cancer (AMBER) study, a large scale longitudinal cohort of women with newly diagnosed early-stage breast cancer, we found that nearly half of women report poor sleep. These women with poor sleep tend to experience lower quality of life, physical well-being but particularly mental well-being, compared to those who sleep well. Although depression and sleep are closely linked, previous studies rarely accounted for depression when examining sleep and quality of life. In our study, once depressive symptoms were taken into account, the association between poor sleep and lower quality of life became much weaker. This suggest that part of the negative effect of poor sleep on quality of life may be due to underlying depression. Only about 10% of women in the study reported depressive symptoms at a level considered clinically meaningful. This raises the possibility that poor sleep could serve as an early warning sign of emerging depression. Ongoing research in the AMBER study will use long-term follow-up to better understand how sleep, depression, and quality of life evolve over time.

The Exercise for Cancer to Enhance Living Well (EXCEL) Study: Implementation Resources - Poster #760

Presenter: S. Nicole Culos-Reed, University of Calgary

Other Authors: Jonathan Low, Julianna Dreger, Margaret McNeely, Melanie Keats, Daniel Santa Mina, Linda Trinh, Kristin Campbell, Isabelle Doré, Colleen Cuthbert, Lauren Capozzi, Daniel Sibley, Thomas Christensen, Alexia Pichè, Kelly Mackenzie

ABSTRACT_ID#2970

ABSTRACT

Objectives/Purpose: Exercise for individuals living with and beyond cancer is critical for optimizing wellness and survival outcomes. The Exercise for Cancer to Enhance Living Well (EXCEL) trial has built implementation resources that support access for underserved rural and remote individuals living with and beyond cancer to safe and effective exercise programs. **Methodology:** EXCEL's hub and spoke framework includes (a) a training pathway, (b) referral networks, (c) screening, triage, and assessment resources, and (d) an 'exercise and educate' model (a 12-week program with two weekly classes, and integration of 5 behaviour change topics with resources). to deliver exercise programs online and in-person across Canada. **Results:** Since September 2020, EXCEL participants (n=1590) have enrolled from rural (74.5%) and urban (25.5%) locations. Average age of participants is 59, with 85% female and 51% breast cancer diagnoses. EXCEL includes 5 'hubs' across Canada that accept referrals and triage participants into classes from 'spoke' sites. To date, 125 QEPs have been trained (online modules, 5-hr training day, moderating and class delivery) and 15 are currently delivering EXCEL. Referrals are direct (18.7%) or indirect (15.7%) from healthcare provider networks or self-referral (65.6%), and participants are on (56.3%) or off-treatment (43.7%) at baseline. EXCEL's screening includes PARQ+ and cancer-specific questions, and triages participants to CEP-supervised (51.5%) or QEP-supervised (42.6%) classes. Post 12-week assessments include functional fitness measures (79.2% completion) and patient-reported outcomes (77.3% completion). **Conclusions and Clinical Implications:** EXCEL's comprehensive resources are driving positive exercise change, with ongoing supports in place to promote sustainable delivery.

LAY SUMMARY

1. Exercise is an evidence-based tool that enhance overall quality of life for individuals living with and beyond cancer. Moving this evidence to practice is essential to ensure wellness is supported within cancer survivorship.
2. Highlighting the resources developed for the EXCEL exercise trial provides information on the key processes and components that have been essential for program implementation and effectiveness.
3. Key EXCEL highlights include the training of qualified exercise professionals to deliver safe and effective programs; a hub and spoke model to connect resources in urban sites with participants across rural and remote locations; and developing a referral network from the healthcare providers to the exercise teams.

Methionine restriction as an adjunct dietary intervention to prevent chemobrain - Poster #761

Presenter: Michiru Hirasawa, Memorial University

Other Authors: Deogratias Riwa, Monawar Shahwan, Akshara Hubert, Anotida Charumbira

ABSTRACT_ID#2877

ABSTRACT

Chemotherapy-induced cognitive impairment (chemobrain) is experienced by up to 75% of patients with non-brain malignancies, which can persist for decades. As life expectancy prolongs for cancer survivors thanks to improved treatments, chronic side effects of cancer therapies must be addressed to preserve quality of life. Here, we used a chemobrain mouse model to test the efficacy of dietary methionine (Met) restriction, which is known to have antioxidative, anti-inflammatory and neuroprotective effects in neurodegenerative conditions and aging. Male C57BL/6 adult mice received 4-weekly i.p. injections of 5-fluorouracil (5FU, 60 mg/kg), a clinically relevant dose of a common chemotherapy drug, or saline (control) while consuming either normal- or low-Met diet. At 1-week post-injections, normal-Met+5FU mice displayed impaired spatial memory in the object location recognition test relative to normal-Met+saline controls. Ex-vivo electrophysiology on brain slices from these mice found 5FU-induced impairment in long-term potentiation (LTP) at hippocampal synapses, a form of synaptic plasticity associated with spatial memory. Furthermore, histological assessment found that 5FU decreased myelination of axons, while increasing astrocyte and pericyte reactivity in the hippocampus. Low-Met diet had no effect on its own in saline-treated mice, however, it prevented the adverse effects of 5FU on spatial memory, synaptic function, and cell morphology. We are currently assessing whether Met restriction reduces 5FU-induced oxidative stress. Overall, our results provide preclinical evidence supporting Met restriction as a dietary intervention to prevent cognitive side effects of chemotherapy. Importantly, dietary Met restriction is reported to have anti-cancer effects, which would be suitable as an adjunct cancer treatment.

LAY SUMMARY

Most cancer patients who receive chemotherapy develop cognitive impairment known as chemobrain. Common symptoms include problems in memory, attention, decision-making, and multi-tasking. Chemobrain impacts daily functioning and quality of life, which can last for decades. There are no effective treatments for chemobrain. Methionine, an essential amino acid found in protein-rich diets, can induce cognitive impairment when consumed in high quantities, while dietary methionine restriction is known to promote brain health. Thus, we tested the hypothesis that limiting methionine intake can prevent chemobrain. Our results indicate that dietary methionine restriction during chemotherapy prevents the adverse effects on the brain and memory function in mice. Thus, methionine restriction is a promising dietary intervention for preventing chemobrain.

A qualitative analysis on cancer survivors' experiences with distress and distress management after attending an interdisciplinary survivorship clinic - Poster #762

Presenter: Rachel Mintz, McMaster

Other Authors: Donny Li, Dr. Martin Chasen

ABSTRACT_ID#2901

ABSTRACT

Background: Cancer survivorship care necessitates a multidisciplinary, person-centred approach to support the physical, emotional, and social needs of individuals post-treatment. Recognizing distress as the "sixth vital sign" in oncology, survivorship services must prioritize both identification and mitigation of distress to empower patients and promote holistic recovery. The William Osler Health System (WOHS) established the first comprehensive Survivorship Clinic (SC) in the Greater Toronto Area, offering individualized care plans through interdisciplinary teams. Objectives: The primary objective was to explore patients' perceptions of distress and experiences with distress management at the WOHS SC. Secondary aims included identifying potential improvements to care delivery and evaluating the influence of socioeconomic factors on satisfaction and access to care. Methods: This prospective qualitative study included T1/T2 and N0/N1, ER-positive breast cancer survivors with at least one prior SC visit. Participants were recruited through physician referral and provided informed consent. Data was collected via semi-structured interviews and demographic questionnaires. Inductive thematic analysis was employed to analyze data. Results: Twelve female-identifying patients (median age 63) were interviewed. Participants were primarily South Asian (46.2%) and White North American (38.4%). Three key themes emerged: distress management, system navigation and administrative support, and barriers to accessing care. Structured follow-up and trusted providers were vital for emotional reassurance, though administrative inefficiencies contributed to stress. Socioeconomic disparities, especially transportation and language barriers, impacted care navigation and satisfaction. Conclusions: The interdisciplinary SC provided essential emotional and logistical support, though systemic and financial barriers remain. Findings inform targeted improvements in survivorship care across diverse populations.

LAY SUMMARY

Emotional reassurance through structured follow-up and trusted providers helped manage survivors' distress, though many still feared "falling through the cracks" post-treatment. Administrative gaps such as scheduling errors and unclear communication regarding available services disrupted care continuity and created frustration. Socioeconomic disparities, especially language and financial barriers, limited patients' access to care and influenced their sense of satisfaction and trust in the care they received.

Response to Thrombolysis in Acute Ischemic Stroke Patients with Cancer – A Post-hoc Analysis of the ACT Trial - Poster #763

Presenter: Ronda Lun, University of Calgary

Other Authors: Cody Doolan, Katrina Ignacio, Mjohammed Almekhlafi, Brian Buck, Luciana Catanese, Aleksander Tkach, Tolulope Sajobi, Richard Swartz, Bijoy Menon, Nishita Singh

ABSTRACT_ID#2782

ABSTRACT

Background: There is an increasing number of patients with cancer and acute ischemic stroke (AIS). We aim to compare outcomes in patients treated with thrombolysis for AIS with a history of cancer to those without. **Methods:** Data are from the phase 3, pragmatic ACT (Alteplase compared to Tenecteplase) trial in patients with AIS within 4.5h of onset. ICD-10 codes via administrative data linkage were used to identify cancer. Primary outcome was 90-day modified Rankin Scale [mRS] 0-2 indicating functional independence. Other outcomes included return to pre-stroke function, mortality, and bleeding. Analysis was done using logistic regression for binary outcomes adjusted for age, stroke severity, presence of cancer history, and onset-to-needle-time. Generalized linear regression model was used for numeric outcomes, with effect measures reported as adjusted risk ratios (aRR). **Results:** 37/1577 (2.35%) patients had a prior diagnosis of cancer. Cancer patients were older (median 76 years [IQR 71–88] vs 74 years [IQR 63–83], $p=0.024$) and had higher baseline NIHSS (median 13 [IQR 8.5–19] vs 9 [IQR 6–16]; $p=0.013$). At 90 days, cancer patients were less likely to achieve functional independence (aOR 0.33 [95%CI 0.15 – 0.75]), had higher mortality (aOR 3.75 (95%CI 1.76–7.75)], and longer lengths of stay (median 11.5 days [IQR 7 – 24.5]) vs 5 days [IQR 3 – 11] respectively), aRR 2.76 (95%CI 2.58 – 2.94) as compared to those without cancer. **Conclusion:** Patients with AIS and a cancer history had worse functional outcomes, prolonged length of stay, and higher rates of mortality as compared to those without cancer.

LAY SUMMARY

With the aging population and increasing number of cancer survivors, the number of cancer survivors with acute ischemic stroke (AIS) is increasing. The efficacy and safety of intravenous thrombolysis for acute ischemic stroke has never been formally studied in this population using the new thrombolytic agent tenecteplase. We present a post-hoc analysis of patients with a history of cancer vs those without a history of cancer from the tenecteplase vs alteplase randomized controlled trial (ACT). We found that AIS patients with a history of cancer had worse functional outcomes, prolonged length of stay, and higher rates of mortality as compared to those with no diagnosis of cancer, though they had similar rates of symptomatic intracranial hemorrhage. Future studies should evaluate if response to thrombolysis in cancer patients differ by clinical characteristics such as cancer site and stage. More work are needed in this area to improve outcomes for cancer survivors with stroke.

Conversations Towards Improving the Quality of Life for Individuals Living with Metastatic Breast Cancer in British Columbia - Poster #764

Presenter: Rachelle Payandeh, BC Cancer

Other Authors: Sarah Chae, Colleen Foran, Colleen Doyle, Helen McTaggart-Cowan

ABSTRACT_ID#2885

ABSTRACT

Background: Individuals with metastatic breast cancer (MBC) have a range of unmet needs when accessing quality care, which may considerably impact their quality of life (QOL). However, MBC remains an understudied research area. In this work, we aim to develop a collaborative research agenda to improve the QOL and care of individuals with MBC in British Columbia. Methods: We conducted a virtual World Café with individuals with MBC and clinicians in April 2025. Co-developed with patient research partners, the World Café consisted of: (1) information about current MBC research; (2) exploration of patient needs; and (3) discussion of barriers and facilitators to quality MBC care. Conversations from the World Café were analyzed to identify research priorities. The research priorities were then ranked by participants on an online survey. Results: Ten participants attended the World Café; of which, four were patients with MBC. Three barriers to quality care were underscored: 1) the difficulty with navigating the healthcare system; 2) the limited options for holistic care; and 3) underrepresentation of MBC patients in clinical research. Peer support was identified as an important facilitator to quality care. Suggestions for improved care included establishing peer or group support systems, and introducing a nurse navigator program to better coordinate care and fill gaps within the healthcare system. Conclusion: Individuals living with MBC face unique barriers and facilitators to quality care. The co-creation of a research agenda, which highlights the experiences of patients and clinicians, will ensure meaningful momentum to an understudied area of research.

LAY SUMMARY

Navigation of the healthcare system was identified as a barrier to quality care. Individuals with metastatic breast cancer desired a nurse or peer navigator to support their cancer care experiences. There are not enough treatment options that look at the body as a whole. Offering emotional or social support can improve care and quality of life for individuals with metastatic breast cancer.

A Pelvic Health eRehabilitation Program to address urogenital impairments after breast and gynecologic cancer: preliminary results of a proof-of-concept study - Poster #765

Presenter: Stephanie Bernard, Université Laval

Other Authors: Chloé Roy-Richard, Donna Pepin, Kadija Perreault, Lynn R. Gauthier, Mary-Ann Dalzell, Marie-Ève Lamontagne, Margaret McNeely

ABSTRACT_ID#3171

ABSTRACT

Introduction: Urogenital symptoms, including incontinence and vulvovaginal pain, are frequent among individuals treated for breast or gynecological cancer (BCG). Pelvic health physiotherapy can help alleviate symptoms in this population, but access to services is often limited. To address this issue, a 12-week online pelvic health rehabilitation program is being tested in a proof-of-concept study in Quebec and Alberta. Objective: To describe urogenital symptoms at baseline and at 6 weeks (halfway point) among the first 20 participants. Methodology: Adults aged ≥ 18 years, treated for BCG within the past 4 years and experiencing urogenital symptoms were eligible. The intervention included access to online resources, weekly virtual group sessions, and three virtual individual sessions with a pelvic health physiotherapist. Urinary symptoms (ICIQ-FLUTS), vulvar pain (VPAQ), and Perceived Self-Efficacy were assessed. Results: Among the 20 participants, 10 had breast cancer, 9 had gynecological cancer, and 1 had both. At baseline, 18 participants reported incontinence (ICIQ-FLUTS incontinence subscore: median 6 [range 0–11]); 14 reported vulvar pain (VPAQ pain severity: 2 [0–3]; interference with sexual function: 3.3 [0–4]). Perceived self-efficacy was 31 [13–50] on a 60-point scale. At six weeks, improvements were seen in the ICIQ-FLUTS incontinence subscore: 4 [0–9], VPAQ pain severity: 1 [0–3.3], interference with sexual function: 3 [0–4], and perceived self-efficacy: 41 [25–60]. Conclusion: Preliminary results reveal diverse urogenital symptoms among participants, underscoring the need for comprehensive, individualized care. This proof-of concept phase will inform the feasibility of the online delivery format.

LAY SUMMARY

People treated for breast or gynecological cancer often experience urogenital symptoms, such as urinary leakage and vulvar pain, which can affect daily life and sexual well-being. Our online rehabilitation program was developed to help manage these symptoms through pelvic health physiotherapy and improve access to care. Early results from the first 20 participants show a wide range of symptoms, with many reporting both incontinence and vulvar pain at the start of the program. These findings highlight the need for more support and personalized care that addresses the complexity and diversity of survivorship needs. Participants have completed the first 6 weeks of the 12-week program, which includes online resources and sessions with a pelvic health physiotherapist. We collected data on symptom severity and self-confidence in managing symptoms. While results are promising, we will continue monitoring progress to better understand the program's potential benefits.

Incidence and Mortality Impact of New-Onset Depression Among Patients with Colorectal Cancer: A Population-Based Cohort Study - Poster #767

Presenter: Vicki Cheng, University of British Columbia

Other Authors: Eric C. Sayre, Jonathan M. Loree, Sharlene Gill, Rachel A. Murphy, Mary A. De Vera

ABSTRACT_ID#2971

ABSTRACT

Background: The relationship between colorectal cancer (CRC) and depression remains poorly understood. We examined incidence and predictors of new-onset depression following CRC diagnosis and evaluated associations between post-CRC depression onset and all-cause and cancer-specific mortality. **Methods:** Using administrative health data from British Columbia, we conducted a population-based cohort study of individuals diagnosed with CRC from 2012–2018. Depression was identified using ICD-9-codes (296.2, 296.3, 296.5, 300.4, 309.x, 311). We calculated rates of new-onset depression post-CRC diagnosis and used Cox models to estimate hazard ratios (HR) and 95% confidence intervals for depression and mortality outcomes, in CRC patients without history of depression, and with+without history of depression (“full cohort”). **Results:** Among 13,986 CRC patients (44.8% female, mean age 66.0), depression incidence peaked in the first-year post-CRC diagnosis (3.54/100 PY without history of depression; 6.43/100 PY full cohort). In CRC patients without history of depression, predictors of new-onset depression included female sex (HR 1.40; 1.22–1.61) and stage IV (HR 1.77; 1.34–2.33). In the full cohort, predictors included female sex (HR 1.27; 1.14–1.42), stage IV (HR 1.56; 1.26–1.92), and history of depression (HR 8.06; 7.21–9.00). Among CRC patients without history of depression, new-onset depression after CRC diagnosis was associated with 97% higher all-cause (HR 1.97; 1.74–2.23) and 73% higher cancer-specific mortality (HR 1.73; 1.48–2.03), with similar associations observed in the full cohort (all-cause: HR 1.63; 1.46–1.81; cancer-specific: HR 1.51; 1.31–1.73). **Conclusion:** Our findings underscore the importance of identifying and managing depression in individuals with CRC throughout and beyond their care.

LAY SUMMARY

- The link between colorectal cancer (CRC) and depression is not yet well understood. We studied how often people develop depression after being diagnosed with CRC, what factors may increase their risk of depression, and how depression after a CRC diagnosis might affect risk of death.
- In this study of nearly 14,000 people in British Columbia diagnosed with CRC, new cases of depression were highest in the first year after CRC diagnosis—especially among those with a history of depression. We found that certain groups are more likely to develop depression after CRC, including females and individuals with stage IV CRC.
- Depression after CRC diagnosis was also linked to a higher risk of death. For example, in CRC individuals without a history of depression, those who developed depression after their CRC diagnosis had a 97% higher risk of dying from any cause and a 73% higher risk of dying from cancer specifically. These findings highlight the importance of recognizing and addressing depression in people with CRC.

Living with and beyond advanced cancer: Understanding patient needs and experiences - Poster #768

Presenter: Sarah Scruton, Nova Scotia Health

Other Authors: Stephen Sallows, Julee Pauling, Yelena Aizenberg, Cynthia Kendell, Anthony Reiman, Amy Trottier, Alison Wallace, Robin Urquhart, John Thoms

ABSTRACT_ID#3018

ABSTRACT

Background: Significant developments in the treatment of advanced cancer have led to unprecedented improvements in survival, introducing a new population of Canadians living long term with their disease. However, most existing supports are tailored to those receiving curative intent treatment, and we do not have an understanding of the unique needs people with advanced cancer face or how best to support them. **Methods:** 25 interviews will take place with individuals diagnosed with advanced cancer to qualitatively explore their experiences, unique challenges, and their thoughts on developing supports to meet their needs. Workshops with patients and healthcare providers will be held to co-design a resource or support to be tested in a future study. **Results:** To date, 20 interviews have been completed. Preliminary analyses have identified numerous unmet and unique needs among individuals undergoing long-term treatment. These include being unable to return to work due to lasting treatment effects resulting in financial and emotional strain, limited availability of long-term support, difficulties with meaning-making, barriers to accessing care, and concerns about future treatment options when current therapies fail. Peer support, exercise programs, and family/friend support were mentioned as facilitators to needs being met. Participants emphasized their desire for future interventions focused on navigation, resource libraries, and assistance managing day-to-day activities. **Discussion:** The findings of this study will provide the information needed to co-design interventions to better support individuals living long-term with advanced cancer. This work highlights gaps in existing services, which will be turned into meaningful supports during the upcoming co-design workshops.

Real-world treatment patterns and survival of patients with hormone receptor-positive (HR+) / human epidermal growth factor receptor 2 negative (HER2-) metastatic breast cancer (mBC) in Alberta, Canada -

Poster #769

Presenter: Winson Y. Cheung, University of Calgary

Other Authors: Simran Shokar, Anna Parackal, Zhor Senhaji Mouhri, Jan-Willem Henning

ABSTRACT_ID#3022

ABSTRACT

BACKGROUND: The treatment landscape for patients with HR+/HER2- mBC has evolved in recent years. Few Canadian population-based studies have investigated treatment patterns and overall survival (OS) in this patient population. **Methods:** For this retrospective cohort study, data for women aged ≥ 18 years diagnosed with de novo stage IV HR+/HER2- mBC between 2015-2021 were derived from population-level databases in Alberta, Canada. Patients were followed until December 31, 2022. **Results:** The study included 681 patients, of whom 611 initiated therapy: 40.9% initiated first line only (1L), 24.7% advanced to second line (2L) only, and 34.4% advanced to third line (3L) or later. The most frequent 1L therapies during the study period were aromatase inhibitors (AI) as single endocrine therapy (ET; 41.9%) and CDK4/6 inhibitors (CDK4/6i)+ET (28.3%), in 2L were CDK4/6i+ET (29.1%) and capecitabine monotherapy (14.4%), and in 3L was capecitabine monotherapy (21.9%). ET was given as 1L only to 45.2%, 1L+2L to 26.0%, and $\geq 3L$ to 23.6%. Median (IQR) time to next treatment (TTNT) was 7.7 (2.8–17.9) months for 1L. For 2L, TTNT was 5.8 (2.8–13.6) months and for 3L was 6.5 (3.8–12.3) months. Median OS (95% CI) was 39.6 (35.8–45.0) months from the initiation of 1L, 31.8 (27.6–37.9) months from 2L initiation, and 23.4 (19.7–27.2) months from 3L initiation. **Conclusions:** This study highlights historically poor long-term survival among patients with HR+/HER2- mBC. Future research should seek to evaluate the impact on clinical outcomes of recently approved targeted therapies and biomarker-directed treatments in 1L and beyond.

LAY SUMMARY

This is among the first Canadian population-based study to investigate treatment patterns and overall survival (OS) in patients with breast cancer that has spread from its original site (metastatic) with hormone receptors for estrogen and/or progesterone and cells with a low level of a protein called HER2. First-line therapy (mostly hormone therapy [aromatase inhibitors] or cyclin-dependent kinase [CDK4] inhibitors) was started in 90% of patients, of whom 59% received second-line therapy (mostly a mix of CDK4 inhibitors, aromatase inhibitors, and chemotherapies), and 34% received third-line therapy. Survival rates decreased with each new line of therapy. Historically, long-term survival has been poor in patients with HR+/HER2- mBC. This study shows the importance of having more effective targeted therapies as second- and third-line options to improve patient outcomes.

Prevalence of multimorbidity and associated risk of mental health problems in adults with cancer: A systematic review and meta-analysis - Poster #770

Presenter: Vagner Tavares, University of Calgary

Other Authors: Lais Cezario, Winson Cheung, Colleen Cuthbert

ABSTRACT_ID#2705

ABSTRACT

Background: Adults with cancer and multimorbidity are less likely to receive benefits from cancer treatment. This systematic review and meta-analysis examined the prevalence of multimorbidity (physical and/or mental health conditions) and risk factors for developing a mental health condition amongst adults with cancer. Methods: We systematically searched major databases (n=4) from inception to November 16, 2024 including studies reporting multimorbidity prevalence and risk factors. We calculated effect sizes reported as proportion rates (%) and risk factors expressed as log OR or HR, including 95% confidence intervals (95%CI). Results: Of 7,292 studies, 130 were selected for full-text review. After applying the inclusion criteria, 29 studies (333,882 participants; 68% female) were included in the meta-analysis. The overall prevalence of multimorbidity was 10% in adults with cancer. The prevalence of mental health (e.g., depression) conditions was 7.1%, while physical health (e.g., diabetes) conditions reached 12%. Males showed increased prevalence. Adults with cancer with physical health conditions had a higher risk of a mental health condition when compared to those without physical health conditions (log OR = 0.34; $p < 0.0001$). Metastasis and treatment type did not affect mental health risk. Sensitivity analysis using the trim-and-fill method adjusted estimates. Publication bias and funnel plot asymmetry were detected in some analyses. In general, there was high heterogeneity and publication bias. Conclusion: This meta-analysis highlights that approximately 10% of adults with cancer experience multimorbidity. Although this prevalence is lower than that observed in the general population, it is noteworthy that 1/10 cancer patients may face complex healthcare needs.

LAY SUMMARY

Multimorbidity is common in cancer patients: About 1 in 10 adults with cancer also had multiple health conditions, with 7.1% experiencing mental health issues (e.g., depression) and 12% having physical health conditions (e.g., diabetes). Physical health problems increase mental health risks: Cancer patients with physical health conditions were significantly more likely to also experience mental health issues compared to those without such conditions. Metastasis and treatment type did not modify mental health risk: The presence of metastasis nor the type of cancer treatment was associated with meaningful changes in the risk of mental health conditions.

Survival of Patients Following Recurrence After Curative-Intent Surgery for Non-Small Cell Lung Cancer - Poster #771

Presenter: Yixian Chen, BC Cancer Research Institute

Other Authors: Tiffany Chang, Pianpian Cao, Cheryl Ho, Rafael Meza

ABSTRACT_ID#3285

ABSTRACT

Background: For patients with non-small cell lung cancer (NSCLC) treated with curative-intent surgery, recurrence remains common, yet survival following recurrence is not well described in population-based cohorts. This study aimed to characterize post-recurrence survival using real-world data sources. **Methods:** Using data from the British Columbia Cancer Registry, we conducted a retrospective cohort study that included patients with NSCLC diagnosed between 2005 and 2021 who had recurrence following curative-intent surgery. A trained clinical research assistant reviewed pathology and imaging reports to determine recurrence timing and location—considered the gold standard. We used Kaplan-Meier method to estimate post-recurrence survival and log-rank tests to compare survival by recurrence location, age, sex, and characteristics of primary lung cancer (diagnosis year, stage, and histology). **Results:** Median survival ranged from 6 to 13 months across clinical factors. Distant-only recurrence was associated with the poorest survival (2-year survival: 7.7%), followed by local-only (22.2%) and combined local and distant recurrence (25.4%) ($p=0.011$). Older age (70-79 vs 40-69, $p=0.015$) was also associated with shorter post-recurrence survival. Sex, diagnosis year, stage, and histology were not significantly associated with survival. **Conclusion:** This study, combined with medical chart data, provides reliable estimates of post-recurrence survival following surgery in NSCLC patients. Future analyses will include patients without recurrence to identify predictors of recurrence and expand assessment of clinical characteristics and treatments. These findings can support follow-up care and individualized strategies for patients at risk of recurrence.

LAY SUMMARY

We built a detailed dataset using manual chart review, allowing more accurate tracking of the location and timing of the cancer's recurrence. Patients whose cancer came back only in the lungs lived longer than those whose cancer spread to other parts of the body. Older people and those with distant-only recurrence had shorter survival after recurrence. This study lays the groundwork for future research to examine which treatments are most effective and which patients are at higher risk. Next, we will include people whose cancer did not come back, so we can understand what factors predict recurrence after surgery. We also plan to analyze more detailed characteristics of both the original tumors and the recurrences, helping doctors better tailor care more precisely.

The Role Tyrosine Kinase Receptors (RTK) and RTK Inhibitors (TKI) Play in Breast Cancer-Related Lymphedema - Poster #772

Presenter: Mimi Vignjevic, University of Alberta

Other Authors:

ABSTRACT_ID#2539

ABSTRACT

Lymphedema is a cancer survivorship disease characterized by arm and leg swelling, causing pain, infection, and reduced quality of life. Cancer-treating surgery, radiation, and chemotherapy damage lymph vessels and nodes, impairing lymphangiogenesis and vessel repair. Many cancers overexpress epithelial growth factor receptor (EGFR) family members and are treated with tyrosine kinase inhibitors (TKIs), like lapatinib, erlotinib, and gefitinib, to block receptor activation and progression. The role of EGFR TKIs in lymphedema is unknown. Primary human dermal lymphatic endothelial cells (HDLEC) and human umbilical vein endothelial cells (HUVEC) were treated with varying EGFR TKI concentrations over 72 hours. Cell death was assessed using trypan blue and Annexin-V/7-AAD assays. We found that EGFR TKIs increase apoptosis in HDLEC and HUVEC in a dose and time dependent manner. EGFR expression, downstream signaling molecules AKT and MAPK, and anti-apoptotic proteins BCL-2 and MCL-1 were then measured by western blot after lapatinib treatment. Lapatinib treatment reduced AKT, MAPK, BCL-2, and MCL-1 expression levels in both HDLECs and HUVECs. We then determined whether lapatinib induces mitochondrial dysfunction using flow cytometry. Results showed that increased mitochondrial reactive oxygen species (ROS) and decreased mitochondrial membrane potential after lapatinib treatment. These findings suggest that EGFR TKIs induce cell death in lymphatic cells through induction of apoptosis and inhibition of survival signaling pathways. This study also provides crucial insights into how lymphedema may develop in cancer patients after targeted therapy.

LAY SUMMARY

1. Lymphedema is a poorly researched cancer survivorship disease that occurs after damage from cancer-treating surgery, radiation, and chemotherapy.
2. Lapatinib, erlotinib, and gefitinib are tyrosine kinase inhibitors that target the EGFR family of membrane receptors in cancers.
3. The role of the EGFR family receptors and TKIs in lymphedema development is unknown and requires further investigation.

Development of an Interprofessional Online Chronic Pain Management Program for Survivors of Breast Cancer: A feasibility study of the “I Can Manage Pain After Cancer” (ICM-PAC) Program - Poster #773

Presenter: Karen Zhang, Juravinski Cancer Centre/McMaster University

Other Authors: Greg Tippin, Jonathan Yen, Som Mukherjee, Silva Darrouj, Peter Ellis, Deb Evans, Kathleen Gallagher, Greg Pond, Jonathan Sussman, Veronica Wong, Denise Bryant-Lukosius

ABSTRACT_ID#3318

ABSTRACT

Background: Chronic pain following breast cancer treatment is common and significantly impacts quality of life. While the biopsychosocial model is the gold standard for pain management, its application has not been evaluated in cancer survivorship. This study evaluated the feasibility of the “I Can Manage Pain After Cancer” (ICM-PAC) online interdisciplinary program for breast cancer survivors. Methods: Participants were recruited from a regional cancer centre in Southcentral Ontario, Canada. Eligible individuals had stage I–III breast cancer, completed surgery and chemoradiation, and reported bothersome pain for over three months. The six-session ICM-PAC program was co-facilitated by professionals in nursing, psychology, physiotherapy, and occupational therapy. Feasibility criteria included: recruitment of ≥ 5 patients/month, consent rate $\geq 60\%$, and $\geq 75\%$ of participants attending at least three sessions. Standardized measures assessed patient acceptability, emotional distress, and pain. Results: Forty-seven patients were recruited over a period of 8 months. Of these, 37 met eligibility criteria and 23 survivors were enrolled (consent rate: 62.2%). Approximately 75% of participants attended at least half of the sessions, and the attrition rate was 13%. Participants rated the program as highly acceptable (mean = 4.11 on a 1–5 scale). Analyses of pre- and post-intervention pain and emotional distress outcomes are ongoing. Conclusion: The ICM-PAC program demonstrated strong feasibility, exceeding predefined success criteria. Trends in the pre-post analyses of emotional distress and pain outcomes will be discussed. The current pilot findings support the development of a randomized controlled trial to evaluate the program’s efficacy in improving pain outcomes among breast cancer survivors.

LAY SUMMARY

A novel online pain management program for breast cancer survivors was developed and delivered by professionals in oncology, nursing, psychology, occupational therapy, and physiotherapy. Patient uptake of the online interdisciplinary pain management program was high, suggesting a critical need for this type of program in breast cancer survivorship. Implications for the development of a randomized controlled trial to examine the efficacy of the online interdisciplinary program for pain management among breast cancer survivors will be reviewed.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Exploring Molecular and Tumour Immune Microenvironment Alterations in Pathogenesis of Triple-Negative Breast Cancer Using Spatial Proteomics - Poster #774

Presenter: Anayra Goncalves Santiago, University of Western Ontario

Other Authors: Vasudeva Bhat, Emily Goebel, Carl Postenka, Muriel Brackstone, Alison Allan, Armen Parsyan

ABSTRACT_ID#2532

ABSTRACT

Introduction: Triple-negative breast cancer (TNBC) is one of the most aggressive and lethal breast cancer subtypes. The interplay between cancer cells and the immune tumour microenvironment (iTME) is a major contributor to metastatic progression to the axillary lymph nodes (ALN). Understanding this process may reveal novel biomarkers and therapeutic targets to improve patient outcomes. We hypothesize that region-specific alterations in tumour cells and the surrounding iTME within heterogeneous TNBC tissues contribute to ALN metastasis. Materials and Methods: Samples of primary tumours, metastatic ALN and normal breast tissues from 50 treatment-naïve TNBC patients (with ALN metastases and no distant metastases) were selected to create tissue microarrays. Spatial proteomics using GeoMx Digital Spatial Profiler with nCounter immuno-oncology panels (Bruker Spatial Biology, USA) was performed after identifying tumour and immune cell regions, using fluorescent antibodies to PanCK (tumour cells) and CD45 (immune cells), respectively. Results and Discussion: Bioinformatic analysis revealed differential protein expression in PanCK+ cells in primary breast tumours and ALNs versus normal tissue, including upregulation of S6, p53, BCLXL (B-cell lymphoma extra large), phospho-MEK1 (S217/S221), PARP (poly ADP-ribose polymerase), Histone H3, and phospho-AKT (S473), suggesting involvement of these proteins in TNBC development and progression. Ongoing analyses is underway to identify additional molecular and iTME alterations for downstream validation. Knowledge gained from this study may support the discovery of novel biomarker and the development of targeted therapeutic strategies in TNBC.

LAY SUMMARY

Tumour cells from both primary breast tumours and metastatic axillary lymph nodes show distinct protein patterns compared to normal breast tissue. Changes in proteins related to the tumour's immune environment could help identify new biomarkers and lead to the development of targeted treatment strategies for triple-negative breast cancer patients.

Determining kinase activation states in chronic lymphocytic leukemia (CLL) through multiplexed targeted mass spectrometry - Poster #775

Presenter: Havva Afshari, University of Manitoba

Other Authors: Victor Spicer, Prasanta Paul, Oleg Krokhin, Thomas Klonisch, Versha Banerji, Sachin Katyal, René P Zahedi

ABSTRACT_ID#2653

ABSTRACT

Introduction: Dysregulated kinases represent a common and effective target in cancer treatment. Phosphoproteomics can decipher signaling networks, but determining specific kinase dysregulation remains challenging. Many kinases are activated by phosphorylation of their 'T-loop', which can be sensitively measured using targeted mass spectrometry (parallel reaction monitoring, PRM). Chronic lymphocytic leukemia (CLL) is incurable even with treatment. BTK inhibitors are dosed daily and costly, often-times have off-target effects, whereby many patients develop resistance. We aim to identify novel and more specific CLL drug targets by profiling kinase activation using T-loop phosphorylation PRM assays. Methods: In-house proteome data on 57 CLL patient samples and MEC1 cells was used to develop PRM assays for kinases, T-loop phosphopeptides, and relevant signaling proteins on an Orbitrap Exploris 480. To identify critical kinases and signaling molecules in complementary discovery data, we developed a novel machine learning-based symbolic architecture that reveals causal structures across 'omics data through deformation-aware convergence. We evaluated our two-pronged approach using MEC1 cells treated with BTK inhibitors ibrutinib and acalabrutinib, which differ in specificity and on/off-target profiles. Results: We identified 317 protein kinases and 92 phosphatases. PRM assays were developed for phosphorylated/non-phosphorylated T-loop peptides representing >100 kinases, and >20 adaptor proteins and phosphatases. Compared to controls, our assay confirmed known and revealed novel off-target effects of these drugs. Conclusion: Our approach using a translational CLL model enables multi-pronged phenotyping of drug responses in a variety of cancers. Integration of PRM with discovery/machine learning approaches may personalize profiling of signaling pathways in patient samples.

LAY SUMMARY

Many cancers, like chronic lymphocytic leukemia (CLL), happen because a communication signal within a cell gets stuck in the "on" position. These signals are controlled by proteins called 'kinases', that act like on/off switches of many processes inside cells. If kinases themselves are too active, they can cause cancer. Because of this, many cancer drugs target kinases – our body has over 500 of them. CLL is the most common blood cancer in the elderly. Current treatments for CLL block one key kinase, but these drugs are expensive and in many patients they stop working over time. We want to find out which other kinases are too active in individual patients, to improve current therapies. Unfortunately, there is no simple and reliable test to check which kinase switches are wrongfully flipped on. We are developing a lab test using a 'mass spectrometry', a technology that lets us measure the activity of over 100 kinases directly in patients samples. We use artificial intelligence to look for complex patterns in our results. The goal is that our lab test will help doctors to match patients with the best treatments and track how well those treatments are working.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Mapping pan-cancer chromosome 4p loss synthetic lethal interaction network - Poster #776

Presenter: Anastasia Gherghi, Concordia University

Other Authors: Alain Pacis, Fatemeh Gholizadeh, Mehri Hajiaghayi, Rohan Dandage, Rachel Wong, Peter Darlington, Guillaume Bourque, Elena Kuzmin

ABSTRACT_ID#3299

ABSTRACT

Chromosome-4p is recurrently deleted in multiple solid tumors, associated with poor survival, and occurs early in cancer evolution conferring a proliferative advantage in basal-breast cancer. However, its functional role in other cancers remains elusive. Here, we use an integrated approach to map the chr4p genetic interaction network across solid cancers identifying targetable vulnerabilities. Focusing on cancer types with evolutionary early chr4p-loss, including esophageal adenocarcinoma, lung squamous cell carcinoma, stomach adenocarcinoma and bladder urothelial carcinoma, TCGA copy number data analysis reveals chr4p-loss affecting large fraction of chromosome arm without minimally deleted region, suggesting broad deletion, driven by loss of multiple genes. Chr4p-deletion is functionally significant, correlating with poor survival. Differential gene expression analysis reveals chr4p-loss association with reduced expression of ~65–74% of genes on chr4p and transcriptome-wide upregulation of cell cycle, microtubule cytoskeleton organization, DNA replication and downregulation of genes involved in immune system activation, indicating misregulation of these processes. Preliminary co-culture experiments using natural killer (NK) and basal-breast cancer cell models with varying chr4p copy number status indicate that chr4p-loss cells confer 2-fold resistance to NK-mediated cytotoxicity, highlighting potential immune evasion mechanism driven by this loss. DepMap genome-wide pooled CRISPR-Cas9 screens analysis identified chr4p-specific genetic interactions with cancer type-specific interactions being 4-fold more common than shared, suggesting tissue-specific selective pressures acting on chr4p-loss. Genetic interactions are enriched for genes annotated to signal transduction, cell cycle, metabolism and DNA replication, with a subset validated using individual gRNA. This study sheds light on targeted therapies for solid cancers harboring large chromosomal deletions.

LAY SUMMARY

Chromosome 4p (chr4p) is frequently deleted in many solid tumors, correlating with poor survival and occurring early in cancer development. In basal breast cancer, it confers a proliferative advantage, but its broader functional role remains unclear. Here, we use an integrated multi-omic approach to identify genetic vulnerabilities associated with chr4p-loss across solid tumors. Focusing on cancer types with early chr4p-loss, namely esophageal, lung squamous, stomach, and bladder cancers, analysis of TCGA copy number data reveals broad deletions lacking a minimally deleted region, suggesting loss of multiple genes. Transcriptomic analysis shows chr4p-loss reduces expression of ~65–74% of genes on the arm and induces global upregulation of cell cycle, DNA replication, and cytoskeleton genes, with concurrent downregulation of immune activation pathways. Preliminary co-culture assays with NK cells show chr4p-loss breast cancer cells are ~2-fold more resistant to immune-mediated killing, indicating potential immune evasion. DepMap CRISPR-Cas9 screens reveal chr4p-specific genetic interactions, with tissue-specific effects occurring four times more often than shared ones. These interactions are enriched in signaling, metabolism, replication, and cell cycle pathways, and a subset has been experimentally validated. Ultimately, our study aims to uncover novel targeted therapies for solid tumors that are currently managed primarily through chemotherapy or surgery.

Systems Biology, Genomics and Proteomics

Sunday, November 2, 2025

Unraveling coordinated multilayer mechanisms to understand and treat glioblastoma - Poster #777

Presenter: Ziyue Cheng, McMaster University

Other Authors: Alberto Torrez, Yuhan Liao, Mikhail Salnikov, Kabir Siraj, Cole Nickason, Fuad Chowdhury, Sheila Singh, Hong Han

ABSTRACT_ID#2721

ABSTRACT

Brain tumors are in desperate need of transformative therapeutic strategies. Glioblastoma (GBM) is the most common and lethal primary malignancy affecting the adult nervous system. It displays immense genetic and cellular tumor heterogeneity and possesses an inherently immunosuppressive tumor microenvironment (TME). The GBM ecosystem, comprising heterogeneous tumor cells and diverse immune, brain, and other cell populations in the TME, evolve rapidly through time and therapy, resulting in therapeutic failure and distinct tumor and associated TME at recurrence. This highlights the urgent need to expand systematic single-cell characterization efforts of patient-matched tumor samples and patient-derived models to better illustrate the spatiotemporal mechanisms underlying GBM progression. Here, we employed systematic single-cell omics analysis to reveal coordinated multilayer gene regulatory networks, including expression, splicing, and polyadenylation, that control GBM progression and recurrence. Notably, we identified novel cellular and molecular mechanisms that drive tumor and glia cell interactions, which subsequently impact key GBM states and treatment responses. Co-culture models coupled with single-cell profiling were further applied to characterize pathways critical for tumor and glia interactions, including cell adhesion, locomotion, and extracellular matrix organization. Our study highlights previously underappreciated mechanisms in driving aggressive GBM phenotypes and provides a tractable model to investigate microenvironmental influences on tumor progression. It will support the timely development of new multilayer biomarkers, regulators, and candidate therapeutic targets urgently required for GBM. The framework and methodologies we build here are highly translatable to investigate diverse cancer types and models.

LAY SUMMARY

Mapping how tumor interacts with brain and immune cells to advance understanding of GBM complexity and progression. Single-cell characterization of patient samples and co-culture models to uncover new ways tumor cells communicate with their surrounding environment. Analysis of coordinated gene regulatory programs and cell-cell interactions to discover biomarkers and therapeutic targets.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Multi-omic Plasma Profiling to Identify Inflammatory Biomarkers Associated with Lung Cancer Recurrence - Poster #778

Presenter: Dagem Chernet, University of Manitoba

Other Authors: Simone Da Silva Rosa, Evan Gauvin, Jay Kormish, Amir Ravandi, Rene Zahedi, Christopher Pascoe, Biniam Kidane

ABSTRACT_ID#3348

ABSTRACT

Rationale: Curative lung resection surgery can increase the risk of infectious postoperative pulmonary complications (PPCs) which are linked with early cancer recurrence. Inflammatory lipid and protein biomarkers linking lung injury to cancer recurrence have not yet been identified. **Objective:** To identify perioperative inflammatory biomarkers that play a role in cancer recurrence and that may be detectable as early as the post-operative period. **Methods:** This was a case-control study nested within a larger prospective cohort study, which recruited eligible consecutive patients undergoing lung surgery with intraoperative pre- and post-surgery arterial plasma collection. Ten lung cancer patients who developed PPCs were identified from within this cohort for LC-MS/MS lipidomics and multiplex cytokine analysis. Seven patients who developed cancer recurrence within two years and seven matched control patients (no recurrence) were included for label-free DIA mass spectrometry proteomics. **Results:** Two lipid clusters were observed across pre-surgery samples based on PCA. Differential abundance analysis comparing these clusters revealed eight significantly increased lipid classes. Pathway analysis of these lipids indicated “inflammatory mediator regulation of TRP channels” as the top significant KEGG pathway. Multiplex cytokine analysis revealed an upregulation of IL-1 α in cluster-2 vs cluster-1. Comparing pre-surgery samples of cancer recurrence patients vs matched controls, proteomic analysis showed a significant downregulation of Lipocalin-1; Putative lipocalin 1-like protein 1. **Conclusion:** Our study connects lipidomic, cytokine and proteomic profiling to elucidate inflammatory pathways contributing to PPCs and cancer recurrence. Our pilot study offers a promising direction towards uncovering tumour-agnostic inflammatory biomarkers non-invasively to predict and detect cancer recurrence.

LAY SUMMARY

Treatment-induced inflammation, along with pre-existing chronic inflammation, can contribute to a tumor microenvironment that promotes cancer progression and recurrence. Pre-surgery plasma lipidomic and cytokine profiling of clusters of ten lung cancer patients who developed postoperative pulmonary complications highlighted “inflammatory mediator regulation of transient receptor protein (TRP) channels” as top enriched KEGG pathway and upregulation of interleukin-1 α , respectively. Lipocalin-1; Putative lipocalin 1-like protein 1 was downregulated in seven patients who developed cancer recurrence within two years compared to seven matched controls with no recurrence.

Systems Biology, Genomics and Proteomics

Sunday, November 2, 2025

The proteasome activator, PA28ab, interacts with KIF11 to promote stemness and therapeutic resistance in glioblastoma - Poster #779

Presenter: Kyle Heemskerk, Charbonneau Cancer Institute, University of Calgary

Other Authors: Ravinder Bahia, Samir Assaf, Orsolya Cseh, Fatima Hamood, Gayatri Dronamraju, Xiaoguang Hao, Rozina Hassam, Artee Luchman, Samuel Weiss

ABSTRACT_ID#2789

ABSTRACT

Proteasome activators canonically bind and regulate the proteasome core particle. However, efforts to inhibit the core particle have failed in clinical trials for glioblastoma (GBM). PA28ab is known to bind the core particle and process peptides for MHC class I antigen presentation. Here, we report a non-canonical role for PA28ab in regulating the proteome of GBM stem cells (GSCs). We developed PA28ab knockout (KO) and overexpressing GSCs and found that PA28ab promoted stem cell features of GSCs without altering in vitro growth. PA28ab KO led to decreased sphere formation and improved overall survival in orthotopic xenografts. Transcriptomic and proteomic characterization of PA28ab KO GSCs revealed alterations of endomembrane system and synaptic trafficking machinery. PA28ab pull-down followed by mass spectrometry revealed undescribed interactors of PA28ab in GSCs. We found that PA28ab interacted with KIF11, a kinesin motor protein known to regulate GSC function and normal neuronal growth and migration. Interaction of PA28ab with KIF11 stabilized KIF11 and promoted resistance to KIF11 inhibitors in GSCs, suggesting that PA28ab can non-canonically regulate the stability of specific proteins. Immuno-peptidomic characterization of PA28ab KO GSCs revealed that its role in peptide processing is conserved in GSCs. However, PA28ab knockdown in a syngeneic immunocompetent model of GBM showed a survival benefit, highlighting that its role in peptide processing may be secondary to tumor promotion in GBM. Our findings suggest that PA28ab has cellular roles beyond peptide processing for MHC class I antigen presentation and has potential as a therapeutic target in GBM.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Discovery of Adrenomedullin 2 as a driver of intratumoral cancer cell invasion, intravasation, and metastasis

- Poster #780

Presenter: Seyedeh Sara Karimian, University of Alberta

Other Authors: Konstantin Stoletov, Alice Amitrano, Bishwa Ranjan Si, Varinder Madhav Verma, Gane Ka-Shu Wong, Konstantinos Konstantopoulos, John D Lewis

ABSTRACT_ID#2313

ABSTRACT

Cancer cell intravasation is the key step of metastasis, and the factors that drive this process are poorly known. Intravasation requires both the metastatic cancer cells capable of entering the vasculature and weakened intratumoral blood vessels that serve as “intravasation ports.” Using high-resolution intravital confocal time-lapse microscopy, we discovered that within the primary tumour mass, cancer cells that display increased ATF2 (ATF2^{high}) activity localize to the perivascular regions where they robustly invade and intravasate. ATF2^{high} cells preferably intravasate into the large, torturous tumour-draining veins that serve as preferable ports of cancer cell intravasation. Comparative RNAseq analysis of ATF2^{high} and ATF2^{low} cancer cells revealed that the human Adrenomedullin 2 peptide hormone (ADM2) is significantly upregulated in highly invasive ATF2^{high} cell population. shRNA-mediated knockdown of ADM2 expression drastically abrogated intratumoral cancer cell invasion and intravasation. Importantly, ADM2-deficient tumours showed significantly diminished vasculature size, depriving the metastatic cancer cells of intravasation-competent tumour vasculature. Moreover, ADM2 knockdown inhibited the formation of invasive metastatic lesions, establishing the requirement of ADM2 peptide hormone throughout the metastatic cascade. Our findings identify Adrenomedullin 2 as a novel pluripotent anti-metastasis target that can simultaneously block tumour angiogenesis and cancer cell invasion, intravasation and invasive lesion formation.

LAY SUMMARY

Cancer intravasation—the entry of cancer cells into blood vessels—is a key step in cancer spreading to other parts of the body. Currently, there are no treatments that specifically target this process. Our team developed a cutting-edge imaging system that lets us watch cancer cells invade and enter blood vessels in real time and with high resolution. Using this system and fluorescent markers, we found that cancer cells with high activity of the ATF2 protein cluster around blood vessels and are solely responsible for all intravasation events. These events mostly occur in large (~50 µm) veins deep within the tumour, which we call “intravasation ports.” This pattern was seen in several human cancers, including fibrosarcoma, head and neck, and prostate cancer. To better understand these ATF2-positive cells, we separated them from ATF2-negative ones and compared their gene expression. We discovered that the ADM2 gene is expressed three times more in ATF2-positive cells. When we knock down ADM2 levels, the cancer cells can no longer invade tissue or form intravasation ports. These findings suggest that ADM2 plays a critical role in cancer cell intravasation, and we propose to further study ADM2 as a potential new target for anti-metastatic cancer therapies.

Systems Biology, Genomics and Proteomics

Sunday, November 2, 2025

Single cell profiling of PR subclones defines high-risk disease in multiple myeloma - Poster #781

Presenter: Noemie Leblay, University of Calgary

Other Authors: Sungwoo Ahn, Alexandra Poos, Mehmet Samur, Sheri Skerget, Holly Lee, Mansour Poorebrahim, David Jung, Sacha Benaoudia, Elie Barakat, Daniel Penaherrera, Herve Avet Loiseau, Jonathan J. Keats, Nikhil Munshi, Niels Weinhold, Nizar J. Bahlis, Paola Neri

ABSTRACT_ID#3076

ABSTRACT

Multiple myeloma (MM) is the second most common hematologic malignancy characterized by the expansion of malignant plasma cells in the bone marrow (BM). Despite therapeutic advances, MM remains incurable, with a subset of patients defined as high-risk (HR) showing poor outcomes. Identification of HR MM remains challenging due to the lack of prognostic and predictive biomarkers. We here evaluated a new method for identifying HR MM based on the presence of highly proliferative (PR) cells and characterized their transcriptomic signature for potential therapeutic targets. Using the 10x GemCode system, we analyzed the single-cell transcriptomics profile of BM plasma cells obtained from 58 newly diagnosed (ND) and 78 relapse/refractory (RR) MM patients. A PR signature ($\geq 5\%$ of MM cells) was found in 27% of ND and 50% of RR cases, including in standard-risk patients. A low PFS was observed in all patients carrying PR subclones (ND: 30 mos vs 43 mos, $p=0.004$, RR: 63 mos vs 105 mos, $p=0.009$). Of note, PR subclones persisted overtime, and in 44% of patients PR cells enrichment was observed at progression, consistent with Darwinian clonal evolution and therapeutic pressure. Pathway enrichment analysis of PR cells revealed enrichment of MTORC1, MYC targets, and DNA repair pathways. Among the DEGs identified, high ENO1 (alpha-enolase) expression was associated with poor PFS, suggesting its role in HR. The presence of PR cells identifies HR disease and correlates with poor survival at diagnosis and progression. Their identification can improve disease prognosis and characterizing their molecular signature may offer therapeutic possibilities.

LAY SUMMARY

Multiple myeloma (MM) is the second most common blood cancer characterized by the presence of cancerous plasma cells in the bone marrow. It is an incurable disease, and some patients have a more aggressive form of MM, called high risk, that responds poorly to treatment and is associated with shorter survival. In this study we have developed a method that can identify these high-risk cancer cells in newly diagnosed and relapsed MM patients. We have shown that these cells persist overtime and their presence is associated with poor outcomes. Furthermore, we have investigated the unique features of these high-risk cancer cells and identified specific signals that can be potentially targeted to generate more effective treatments for these patients.

HPV integration may alter the epigenetic landscape and immunogenicity of extrachromosomal DNAs in cervical cancer - Poster #782

Presenter: Signe MacLennan, University of British Columbia

Other Authors: Michelle Ng, Vanessa Porter, Pawan Pandoh, Diane Trinh, Robin Coope, Yongjun Zhao, Marco Marra

ABSTRACT_ID#2909

ABSTRACT

Cervical cancer, a human papillomavirus (HPV) driven malignancy, remains the fourth deadliest cancer in females. Approximately 25% of primary cervical cancer samples are predicted to contain circular amplicons called extrachromosomal DNAs (ecDNAs). Often harbouring oncogenes, ecDNAs are known cancer drivers. However, neither ecDNAs containing only human DNA (i.e. human-only ecDNAs) or ecDNAs containing both HPV and human DNA (i.e. HPV-human hybrid ecDNAs) are well characterized in terms of their epigenetic regulation. Here we report that 30% of cervical cancer samples from a Ugandan cohort contained one or more predicted ecDNAs. Human-only ecDNAs (74% of the total) were larger on average and contained more human genes, whereas HPV-human hybrid ecDNAs (26%) more often contained epigenetically active enhancers, more structural variants (SVs), and invariably bore the HPV oncogenes E6 and E7. Samples containing HPV-human hybrid ecDNAs trended towards having higher T-cell infiltration scores, particularly when the ecDNAs were present at high copy number. We also found that E6 expression was increased and E2 expression was decreased among HPV-human hybrid ecDNA-containing samples, suggesting that HPV-human hybrid ecDNAs represent a form of HPV integration that promotes higher HPV oncogene expression. Although ecDNAs contained few allelic differentially methylated regions (aDMRs), we found a few examples of aDMRs overlapping gene promoters within ecDNA loci. Overall, our study in cervical cancer found notable differences in structural and epigenetic features between ecDNAs containing HPV DNA compared to those without. Our study may hint at whether HPV integration might relate to inferred T cell abundance.

LAY SUMMARY

1. Many cancers contain circular DNA structures called extrachromosomal DNAs (ecDNAs) that promote cancer development and progression by carrying cancer-promoting genes called oncogenes. Some ecDNAs in cervical cancer contain parts of the human papillomavirus (HPV) genome (i.e. called HPV-human hybrid ecDNAs), but prior to our study it was unknown how HPV-human hybrid ecDNAs differ from ecDNAs containing only human DNA (i.e. human-only ecDNAs). We found that HPV-human hybrid ecDNAs are distinct genomically (structurally) and epigenomically (in terms of regulation) compared to ecDNAs containing only human DNA. In particular, HPV-human hybrid ecDNAs are smaller, tend to bear more active enhancers, and contain a higher density of structural variants (SVs).
2. Long-read haplotype phasing can be used to assign ecDNA-specific methylation patterns, which may influence which genes on ecDNAs are expressed (including cancer-promoting oncogenes). We found that methylation is sometimes altered surrounding HPV integrants on ecDNAs. ecDNAs are also not enriched in allelic differentially methylated regions (aDMRs) relative to the rest of the genome.

Rising Circulating Cell-Free Tumour DNA During the Trajectory of Prostate Cancer Patients Reveals Genomic Alterations Related to Progression - Poster #783

Presenter: Samira Rahimirad, McGill University

Other Authors: Eleonora Scarlata, Lucie Hamel, Seta Derderian, Ginette McKercher, Fadi Brimo, Fred Saad, Armen Aprikian, Simone Chevalier

ABSTRACT_ID#2663

ABSTRACT

Background: Although prostate cancer (PCa) has a high survival rate, several patients progress to late stages. Indicators of progression and therapy response/resistance are key for evaluating treatment efficacy. Longitudinal liquid biopsies (blood) are non-invasive and may be meaningful to monitor progression and reveal underlying mechanisms. We aimed to identify genomic alterations in aggressive PCa primary tumours and track them in patients' blood to capture changes emerging during progression. **Methods:** Banked fresh frozen prostate tissues from six radical prostatectomy cases with lethal PCa were processed to identify tumour foci and macro-dissect cores of high cellularity (>75%) for DNA extraction. Their serial plasma collections (<4 mL) were used to isolate cell-free DNA (cfDNA), next sequenced along with tumour and matched blood DNAs. **Results:** Already established and novel copy number variations (CNVs), mostly deletions, were found in primary tumours of the six patients. Longitudinal cfDNA analysis revealed rising circulating tumour DNA (ctDNA) fractions when patients reached the metastatic and late stages. Alterations in ctDNAs were associated with emerging neuroendocrine and stem cell subtypes. Although genomic amplifications were rare in primary tumours, they were common during progression. Furthermore, the clonality of ctDNA alterations were related to treatment resistance over time. **Conclusion:** The identification of novel genomic changes in tumour DNA and ctDNAs in advanced disease is enhancing our knowledge of PCa severity. Findings on ctDNA underscore the significance of longitudinal liquid biopsy for monitoring disease progression and could contribute to the development of novel diagnostic tests and more effective treatments impacting on PCa lethality.

LAY SUMMARY

The sequencing of serial cell-free DNA from blood of prostate cancer patients is worth adding to clinical tests (PSA) and imaging to monitor disease progression. The genomic profile of each patient's primary tumour is unique at time of radical prostatectomy and remains patient's specific in their liquid biopsies, even if modified later during progression. These findings highlight the importance of integrating blood tumour DNA into Precision Oncology.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Hyperpolarized light: a proteomic analysis of MCF7 and MCF10A cell lines in vitro - Poster #784

Presenter: Barbara Truglia, University of Alberta

Other Authors: Yassen Tabana, Atef Mannaa, Richard Fahlman, Bart De Wever, Maral Aminpour, Jack Tuszynski

ABSTRACT_ID#2770

ABSTRACT

Breast cancer therapies require alternatives to minimize treatment side effects. Hyperpolarized light therapy (HPLT) is an emerging, non-invasive approach that delivers highly organized, broad-spectrum polarized light to biological tissues. This study explores the effects of HPLT on breast cancer (MCF7) and non-tumorigenic breast epithelial (MCF10A) cell lines using a comprehensive proteomic analysis. Cells were exposed to hyperpolarized light (350–3400 nm, 2.4 J/cm² per minute) for 10, 20, or 30 minutes, and protein expression profiles were assessed 24 hours post-treatment using data-independent acquisition mass spectrometry. Significant changes in protein expression were observed, particularly after 20 minutes of exposure. In breast cancer cells, HPLT promoted the upregulation of proteins associated with cytoskeletal organization and actin filament dynamics, suggesting enhanced cellular structural remodeling. In contrast, non-tumorigenic cells showed a marked downregulation of proteins linked to extracellular matrix structure, signaling pathways, and tissue elasticity. These differential responses highlight the selective impact of HPLT on cancerous versus healthy breast cells, potentially influencing cellular architecture and communication in ways relevant to tumor progression and tissue homeostasis. The findings support the potential of HPLT to modulate key cellular pathways without the damaging effects associated with conventional therapies. By providing new insights into the molecular mechanisms underlying light-based interventions, this research establishes a foundation for the development of more targeted, less toxic therapeutic strategies for breast cancer and possibly other malignancies. Further studies are warranted to optimize treatment parameters and evaluate the clinical applicability of HPLT in oncology.

LAY SUMMARY

Effects of light-based electromagnetic fields in terms of morphology alterations, energy production and metabolic shifts addressing modifications in specific proteins such as actin and microtubules (cytoplasm) or mitochondria optimization of protocols for less invasive and harmful treatments based on light electromagnetic fields.

Systems Biology, Genomics and Proteomics

Monday, November 3, 2025

Accelerated Tumor Evolution with Multimodal CRISPR Technologies - Poster #786

Presenter: Vimal Keerthi, Université de Montréal

Other Authors: Sebastian Merker, Gabriela Flores-Vargas, Xiaozhen Wen, David Sharon, Jeffrey Sullivan, Aishwarya Jayaraman, Benjamin Haley

ABSTRACT_ID#2491

ABSTRACT

Personalized therapies targeting oncogenic KRAS and EGFR mutants have significantly advanced non-small cell lung cancer (NSCLC) treatment. However, acquired drug resistance remains a critical challenge. While genome sequencing can retrospectively identify resistance mutations, this approach relies on patient sampling post-treatment failure, potentially missing the full spectrum of resistance-promoting alterations. To accelerate tumor evolution and identify drug resistance modifiers, we're deploying massive scale CRISPR-activation (CRISPRa) and CRISPR mutagenic base-editing (CRISPR-mBE) perturbation technologies. Our CRISPRa system utilizes a custom, genome-scale lentiviral guide RNA library for targeted upregulation of gene expression, simulating oncogenic amplifications. We will apply this platform across multiple lung cancer models, in the presence or absence of MAPK pathway inhibitors, to uncover gain-of-function effects impacting drug sensitization. Our parallel CRISPR-mBE approach systematically enables in situ saturation mutagenesis across all coding regions of ~150 MAPK pathway genes implicated in cancer progression and drug resistance. A proof-of-concept screen to discover EGFR inhibitor resistance in a NSCLC line recovered anticipated variants, including KRAS Q61 and A146 mutants, and we are applying this platform across a broader range of drugs and cell types. Top hits with robust effect sizes, clinical implications, and novel biological relevance will be prioritized for validation and uncharacterized candidates will be examined to elucidate novel resistance mechanisms and genetic network associations. These complementary CRISPR approaches enable us to elucidate diverse mechanisms of drug resistance, construct a comprehensive "resistome" database of resistance-driving variants/over-expression effects, guide targeted therapy development, and establish prognostic biomarkers for predicting resistance to MAPK pathway inhibitors.

LAY SUMMARY

- We're accelerating tumor evolution in-vitro. By using advanced CRISPR perturbation technologies, we can reconstruct ways cancer cells evolve and become resistant to drugs on an accelerated timescale, before it arises in patients. This lets us understand, predict and potentially prevent treatment failure.
- We're building a map of resistance pathways. Through massive-scale genetic screens, we're identifying the genes and mutations that help cancer cells survive targeted therapies, such as those aimed at KRAS and EGFR mutations in lung cancer. This work can guide precise cancer treatments. The goal is to create a "resistome" that not only helps doctors choose effective drug combinations but also informs the development of next-generation therapies, including the possibility of cancer vaccines tailored to individual tumor mutations.

Outlining The Role of Hypoxia in Bladder Cancer Dedifferentiation and Progression - Poster #788

Presenter: Andrew Garven, Queens University

Other Authors: Hamid Ghaedi, Julia Vassalakis, Lynne Postovit, Amber Simpson, David Berman

ABSTRACT_ID#2865

ABSTRACT

Introduction: Bladder cancer (BC) arises from the urothelium, comprising basal progenitor cells and their differentiated luminal progeny. Early-stage non-muscle invasive BC (NMIBC) typically exhibits a luminal phenotype and are often responsive to therapy. In contrast, the aggressive basal subtype emerges predominantly in muscle-invasive BC (MIBC), with greater metastatic potential and worse clinical outcomes. This study investigates mechanisms driving the transition from luminal NMIBC to basal MIBC. **Methods:** To investigate transcriptional differences between subtypes, we analyzed RNA-seq data from 419 MIBC tumours in TCGA-BLCA and flow-sorted SW780 cells, comparing luminal (GATA3?) and basal (CK5?) populations using differential expression and pathway enrichment analyses. Single-cell trajectory analysis reconstructed lineage relationships and traced the emergence of basal phenotypes from luminal precursors. Oxidative stress was modeled by culturing SW780 cells under hypoxic conditions, followed by quantification of differentiation markers, hypoxia-associated genes, and cellular stress indicators via qPCR. **Results:** Oxidative stress pathways, including hypoxia and cellular stress responses, were upregulated in basal versus luminal samples from TCGA-BLCA and SW780 cells. Single-cell analysis showed luminal-to-basal transitions marked by transcriptional changes linked to stress and dedifferentiation. Hypoxic culture of SW780 cells increased HIF1 α , LOX, and eIF2 α , while inducing a basal phenotype (higher KRT5, lower GATA3). **Conclusion:** Lineage plasticity, driven in part by hypoxia and oxidative stress, enables luminal BC cells to acquire basal-like features, promoting progression to MIBC. **Impact:** Elucidating mechanisms of luminal-to-basal transition provide insights into the development of lethal BC, supporting earlier detection of high-risk cases and guiding the development of novel therapies.

LAY SUMMARY

1. **Bladder Cancer's Dangerous Disguise** Early-stage bladder cancers are predominantly made up of a less aggressive cell identity called luminal. Over time, however, some of these tumours shift toward a more dangerous cellular identity known as basal, which spreads more quickly and is harder to treat. This shape-shifting ability, known as plasticity, helps explain how certain early bladder cancers become life-threatening.
2. **Low Oxygen Levels Can Trigger Plasticity in Bladder Cancer** When exposed to low-oxygen (hypoxic) conditions bladder cancer cells experience significant stress that alters their behaviour. In response, these cells begin to lose their original luminal identity and adopt features of the more aggressive basal type. This shift suggests that oxygen deprivation in the tumour environment is an active driver of harmful cellular plasticity.
3. **Catching the Shift Before It's Too Late** Understanding how and why bladder cancer cells evolve from less aggressive to more dangerous forms offer critical insight into disease progression. Tracking this transformation may reveal early warning signs that a tumour is becoming high-risk. This knowledge could lead to earlier diagnosis and support the development of therapies aimed at interrupting this dangerous cellular shift.

Optimization of a Pan-Membrane Marker for 9-Plex Multiplex Immunofluorescence Using Opal-TSA Technology: Addressing Challenges of Stripping, Cross-Reactivity, and Blocking - Poster #790

Presenter: Julia Schuurmans, Queen's University

Other Authors: Logan Engle, Andrew McLellan, Eman Radwan, Michael Fotheringham, Mide Olanrewaju, Ben Green, Katherine Brewer, Janis Taube, Tricia Cottrell

ABSTRACT_ID#2650

ABSTRACT

Background Multiplex immunofluorescence (mIF) enables spatial profiling of the tumour microenvironment (TME), but extraction of single-cell data requires precise cell segmentation. This is often suboptimal for large, irregular tumour cells, leading to inaccurate quantification and missed membrane-localized proteins (e.g., PD-L1). AI-based segmentation algorithms leverage membrane and nuclear signals for accurate performance, but inconsistent membrane staining limits their application. This study aimed to optimize a pan-membrane marker cocktail for consistent membrane delineation in a 9-marker mIF panel. Methods A CD44, CD45, and ATPase antibody cocktail was designed with board-certified pathologists (JT/TRC) to label tumour and lymphocyte membranes. Antibody staining behavior was assessed by gold-standard IHC. Fluorescent signal optimization involved titration of primary, secondary, and Opal-TSA reagents. Once optimized in single-plex, the cocktail was incorporated into a 9-plex mIF panel including PD-1, PD-L1, FoxP3, MPO, CD8, CD163, pan-cytokeratin (pan-CK), and DAPI. mIF validity was defined as <5% difference in positive staining vs IHC (N = 4 NSCLC/MESO cases). Results Pan-membrane was optimized in single-plex at dilute concentrations (CD44: 0.066 µg/µL; CD45: 0.156 µg/µL; ATPase: 0.007 µg/µL). Cross-reactivity from pan-CK was resolved by adjusting the staining order. Membranous PD-L1/PD-1 partially blocking the cocktail was merged into the segmentation algorithm input to compensate for signal interference. This combination yielded more accurate cell boundaries than pan-CK-dependant segmentation (32.5% ±10% increase in mean cell area). Positive pixel-based validation showed ±4% concordance between mIF and chromogenic IHC. Conclusion Our approach demonstrates a validated strategy for improving membrane delineation in mIF—critical for TME spatial analysis and AI-assisted segmentation.

LAY SUMMARY

Our goal was to address the gap between multiplex immunofluorescence (mIF) image optimization and accurate single-cell data extraction caused by faulty outlining of single-cells in the complex tumour microenvironment. Specifically, we focus improving the outlining of large irregularly shaped tumour cells – where detection and accurate quantification of membrane protein expression is critical for clinical decision making (e.g. PD-L1). In this study, we optimized a "pan-membrane" marker, made up of a mix of three membrane-targeting antibodies using TSA-Opal technology. We present resolution of common difficult technical problems, like unwanted background signal (cross-reactivity) and interference between markers, ensuring membrane-specific staining. Our results show that the inclusion of a pan-membrane marker on large tumour cells in mIF—rather than relying on cytoplasmic ones—greatly improves the accuracy of cell membrane detection by AI-based image analysis tools like Cellpose, without custom model training. We provide a step-by-step workflow, including troubleshooting tips, to help other researchers successfully add a pan-membrane stain to their multiplex immunofluorescence (mIF) panels using Opal-TSA technology—improving the accuracy of extracted single-cell data.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Effect of mtDNA and gDNA on Immune Cell Activation in Colorectal Cancer Subtypes - Poster #791

Presenter: Angie Chen, University of Alberta

Other Authors: David Doell, Siwon Choi, Shayla Mosley, Courtney Mowat, Kristi Baker

ABSTRACT_ID#2333

ABSTRACT

There are two subtypes of colorectal cancer (CRC) known as microsatellite instable (MSI) and chromosome instable (CIN). Patients with MSI CRC have greater survival compared to patients with CIN CRC due to increased activation of cGAS/STING, which is a cytosolic DNA sensing pair that detects the presence of leaked mitochondrial DNA (mtDNA) or genomic DNA (gDNA). MSI CRCs commonly have a mutation in an important mitochondrial protein which induces mtDNA leakage upon dysfunction, therefore we hypothesize that MSI mtDNA will have a more potent cGAS/STING response leading to greater immune activation. We tested this using dendritic cells (DCs) and T cells isolated from C57BL/6 mice along with mtDNA and gDNA isolated from MSI and CIN variants of the mouse MC38 CRC cell line. DCs were treated with varying ratios of mtDNA:gDNA, resulting in increased mRNA expression of cGAS/STING response genes after MSI mtDNA treatment. This was suppressed in STING knock-out DCs as seen with flow cytometry measurements of immune factor expression. Cocultures of mtDNA or gDNA treated DCs with either CD8+ or CD4+ T cells showed varying levels of T cell activation with flow cytometry. CD8+ T cells had greater activation during coculture with mtDNA-treated DCs, indicating a DNA type specific response, while CD4+ T cells had greater activation during cocultures with MSI DNA-treated DCs, indicating a CRC subtype specific response. These results show the importance of mtDNA in the activation of immune cells in response to CRC and present new potential adjuvant therapies for patients with CIN CRC.

LAY SUMMARY

Colorectal cancer (CRC) contains two subtypes called MSI and CIN. MSI patients have a better prognosis compared to CIN patients due to stronger natural immunity potentially caused by the activation of an immune pathway. This immune pathway can be activated by DNA which normally resides in specific cell compartments such as the nucleus or mitochondria, the energy producing component of the cell. This DNA is called mitochondrial DNA and has a bacterial origin. We hypothesize that MSI mitochondrial DNA will trigger the immune pathway more strongly compared to DNA from the nucleus. Results show that treating dendritic cells, which are immune cells vital for triggering the anti-tumour response, with mitochondrial DNA originating from MSI colorectal cancer subtypes causes a greater immune pathway activation compared to treatment with nuclear DNA. Dendritic cells treated with mitochondrial DNA from MSI CRC can strengthen T cell response, which are responsible for killing tumour cells. Currently, we are investigating how mitochondrial DNA enhances immune activation through these immune pathways. Our goal is to better understand why MSI CRC have a stronger immunity than patients with CIN CRC so that we can one day utilize these mechanisms to develop new immunotherapy treatments for CIN CRC patients.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Mechanistic Insights into von Willebrand Factor and Endothelial Interplay in Solid Tumor Metastasis: A Scoping Review - Poster #792

Presenter: Abdelrahman Ahmed, Department of Biomedical and Molecular Sciences, Queen's University

Other Authors: Jordan Harry, Abdulrahman M. Yaish, Monzer Assar, Isabell Kearn, Maha Othman

ABSTRACT_ID#3254

ABSTRACT

von Willebrand Factor (vWF) is a critical endothelial protein, consistently linked to cancer progression and metastasis. However, the underlying mechanisms remain unclear. No study has synthesized the multiple mechanisms of vWF in metastasis into a unified framework, particularly in relation to other key regulators such as VEGF and MMPs. Thus, this scoping review aims to compile existing literature to facilitate the understanding of vWF's role in cancer metastasis, with a focus on endothelial modulation. Following PRISMA guidelines, a comprehensive search was conducted using MEDLINE, Embase, and Web of Science using a strategy developed with the following concepts: solid tumours, endothelium, vWF, and cancer metastasis. Inclusion criteria included primary peer-reviewed in-vitro, in-vivo studies, and randomized controlled trials involving adult (≥ 18) patients diagnosed with metastatic solid cancer. Patients having bleeding/coagulation disorders or using anticoagulants were excluded. Each study was reviewed by two independent reviewers, with conflicts resolved through group consensus. The search yielded 1761 studies on May 4, 2025. Following removal of 798 duplicates, 963 studies were identified for Title and Abstract screening. To date, 675 studies were screened and 164 studies moved to full-text review. Data extraction will include relevant elements such as: cancer type, metastasis location, patients' demographics and treatments, any endothelial protein with a proven role in metastasis and vWF-related mechanisms. This scoping review will provide a comprehensive overview of current evidence linking vWF-mediated endothelium changes to metastatic processes in cancer. The findings will help enhance understanding of cancer metastasis and potential therapy resistance, informing novel therapeutic strategies.

LAY SUMMARY

vWF is an important hemostatic protein involved with endothelial integrity as well as cancer metastasis.. vWF's exact mechanistic pathways involving the endothelium during cancer metastasis are not well investigated, especially in the context of other important regulators like VEGF, kinases, and MMPs. Unraveling the different mechanisms and pathways involving vWF's interactions with endothelium and other mechanistic pathway should clarify tumour biological pathways and inform future research and therapeutic exploration.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

A *Drosophila* tumor model reveals non-apoptotic role of initiator caspase in driving hypoxia-mediated tumor overgrowth - Poster #794

Presenter: Abhishek Sharma, University of Calgary

Other Authors: Savraj Grewal

ABSTRACT_ID#3335

ABSTRACT

Solid tumors are often associated with low oxygen (hypoxic) microenvironments. While hypoxia damages normal cells, tumor cells carry genetic mutations allowing them to thrive under hypoxic conditions. As a result, hypoxia is often associated with aggressive cancers and poor prognosis, making it important to understand how hypoxia impacts tumor biology. *Drosophila* imaginal discs provide a powerful *in vivo* system to model tumor biology. These epithelial tissues resemble mammalian epithelia, and their growth depends on several conserved oncogene and tumor suppressor pathways. Using this system, we generated cell clones mutant for the tumor suppressors *tsc1* and *foxo* and found that they exhibit tumor-like overgrowth. Interestingly, hypoxia elevated ribosome and protein synthesis in these cell clones and dramatically amplified their overgrowth phenotype—growth that came at the expense of neighboring wild-type tissue. These hypoxic tumors also showed increased caspase activation and apoptosis. Blocking apoptosis at the level of effector caspases further exacerbated tumor overgrowth, suggesting that apoptosis functions to limit tumor growth. Strikingly however, blocking the upstream initiator caspase *Dronc* not only prevented apoptosis but also blunted the increased ribosome and protein biosynthesis and reversed the hypoxic tumor overgrowth. These findings reveal that *Dronc* caspase functions in dual roles: promoting tumor-suppressive apoptosis while simultaneously driving tumor-promoting biosynthetic programs under hypoxic conditions. We are currently exploring the mechanisms by which *Dronc* mediates these effects. Given the conservation of caspase pathways across species, our findings may reveal fundamental principles governing how hypoxic stress shapes tumor behavior in human cancers.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

The Molecular Mechanisms of Vessel Co-option by Liver Metastases - Poster #795

Presenter: Amatzia Gantz, McGill University

Other Authors: Stephanie K Petrillo, Anthoula Lazaris, Peter Metrakos

ABSTRACT_ID#2497

ABSTRACT

Colorectal cancer liver metastases (CRCLM) represent a critical clinical challenge, with poor prognosis particularly in patients exhibiting replacement histological growth pattern (RHGP). CRCLM presents with two major histological pathologies: i. RHGP tumors infiltrate the liver parenchyma and obtain their blood supply via co-opting existing liver vasculature and remain largely unresponsive to current standard of care and ii. desmoplastic subtype (DHGP) which is angiogenically driven, that respond to current treatment. RHGP is characterized by cancer cell infiltration into the liver parenchyma, suggesting a unique tumor-microenvironment interaction. Current therapies fail to address patients with RHGP lesions, presenting the need for subtype-specific therapeutic strategies. This study investigates the mechanisms underlying RHGP lesion formation, focusing on tumor-hepatocyte interactions. Co-culture systems were established using GFP-labeled immortalized human hepatocytes (IHH) and mCherry-labeled colorectal cancer cell lines (HT29, COLO320). Our findings reveal cell-in-cell events, with cancer cells infiltrating hepatocytes and then exiting. These interactions may reflect a novel mechanism of colorectal cancer cells. Cell-in-cell events were visualized using Incucyte live imaging and confocal microscopy. These events appear to be temporary, some resulting with no visual effect on either of the cell types. This work provides foundational insights into RHGP-specific biology and mechanism, supporting the development of novel biomarkers to potentially develop tailored treatment strategies for CRCLM.

LAY SUMMARY

Colorectal cancer spreads to the liver where it forms metastases. There are limited treatment options for these patients. There are two main ways these cancer cells grow in the liver. One type, called replacement histological growth pattern (RHGP), does not respond to current treatment because cancer cells blend into normal liver tissue and use its existing blood supply. The other type, desmoplastic (DHGP), does not infiltrate and thus responds better to current therapies. This study explored how RHGP tumors form and interact with liver tissue. Using fluorescent markers, we observed a novel phenomenon, where cancer cells enter and exit liver cells - an interaction called "cell-in-cell." In most events observed, these events didn't appear to harm either cell type but may reflect a unique behavior of RHGP tumors. The findings could lead to better detection tools and new treatments tailored for this deadly cancer type.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Mapping Macrophage Dynamics During the Transition from Normal Oviduct to Tumour Microenvironment - Poster #796

Presenter: Alexandra Viscotchi, McGill University

Other Authors: Dardan Konjusha, Yojiro Yamanaka

ABSTRACT_ID#2778

ABSTRACT

Most tissue resident macrophages (TRMs) originate from embryonic macrophages and are different from monocyte-derived macrophages (MDMs), which originate from definitive hematopoietic cells in the bone marrow. TRMs are derived from erythro-myeloid progenitors in the yolk sac, which differentiate into tissue-specific subtypes as they colonize embryonic tissues. TRMs are thought to have self-renewal abilities but can also be replenished by MDMs. MDMs are continuously renewed from hematopoietic stem cells. Both TRMs and MDMs are known to coexist in many tissues, but their distribution and roles are not well understood in many sites, such as the female reproductive tract. The oviduct (fallopian tube in humans), particularly the distal portion, is thought to be the site of origin for high-grade serous ovarian carcinoma (HGSOC), the most lethal form of ovarian cancer. However, the precise role of TRMs and MDMs in the early steps of HGSOC progression remains unknown. I hypothesize that TRMs are the first responders to oncogenic insults in epithelial cells and are key mediators controlling local communication with epithelial cells and recruiting circulating immune populations; together, they contribute to the transition from a normal tissue environment to a tumour microenvironment. To test this, we use IL7RCre;tdTomato:CD68-GFP mice to visualize TRMs in red and MDMs in green. We introduce oncogenic mutations into distal oviduct epithelial cells using in vivo electroporation, CRISPR/Cas9, and piggybac-based overexpression. Preliminary data at 1–4 weeks post-electroporation shows an increase in tdTomato+ and GFP+ cells, possibly indicating TRM activation and recruitment of MDMs to the site of oncogenic insult.

LAY SUMMARY

Tissue residential macrophages may be the first responders to mutations in epithelial cells and may recruit macrophages derived from circulating monocytes. Together, they may contribute to the transition from normal to tumour microenvironment.

TGF- β Inhibition as a Strategy to Target Vessel Co-option and Immune Evasion in Colorectal Cancer Liver Metastases - Poster #797

Presenter: Anastasia Tsatoumas, McGill University

Other Authors: Audrey Kapelanski-Lamoureux, Amatzia Gantz, Stephanie K Petrillo, Anthoula Lazaris, Peter Metrakos

ABSTRACT_ID#2488

ABSTRACT

Colorectal cancer (CRC) is the third most commonly diagnosed cancer and a leading cause of cancer-related death, primarily due to liver metastases (LM), which affect over 50% of patients and account for 90% of CRC-related mortality. Surgical resection offers the only potential cure, but only 15–20% of patients are eligible. Current therapies are non-specific and overlook the biological heterogeneity of colorectal cancer liver metastases (CRCLM). Two major CRCLM subtypes exist: desmoplastic lesions, which rely on angiogenesis, and replacement lesions, which co-opt liver vasculature, resist anti-angiogenic therapy, and correlate with poor prognosis. TGF- β signaling promotes vessel co-option via epithelial-to-mesenchymal transition (EMT), the TGF-RUNX1-TSP1 loop, and by fostering immune exclusion. We hypothesize that TGF- β signaling from tumor-associated macrophages (TAMs), hepatocytes, and tumor cells cooperatively drives replacement-type CRCLM by promoting vessel co-option and immune suppression. To test this, we targeted TGF- β signaling from all three sources and selectively depleted TAMs, key TGF- β sources. CRCLM was modeled in C57BL/6 mice via intrasplenic injection of MC38 cells. Mice were randomized into control, TGF- β inhibitor, standard-of-care (SOC: chemotherapy + anti-VEGF), and SOC + TGF- β inhibitor. A separate cohort was treated with anti-CSF1R to deplete TAMs or IgG control. Tumor histology, survival, and immune infiltration were analyzed. Both SOC and TGF- β inhibition modestly improved survival, but only TGF β -treated tumors showed necrosis and stromal remodeling. Anti-CSF1R did not improve survival. Notably, anti-TGF- β or anti-CSF1R increased CD8+ T-cell infiltration, supporting TGF- β 's role in vessel co-option and immune exclusion. Targeting this pathway may enhance subtype-specific immunotherapy in replacement-type CRCLM.

LAY SUMMARY

1. **Liver Metastases Differ in How They Grow and Respond to Treatment:** Two major subtypes of CRCLM exist. Desmoplastic lesions grow by forming new blood vessels (angiogenesis) and can respond to anti-angiogenic therapies. In contrast, replacement-type lesions co-opt existing liver blood vessels, making them resistant to current treatments and linked to worse outcomes. Recognizing these differences is crucial for developing more precise therapies.
2. **TGF- β Signaling Drives Aggressive Tumor Behavior and Immune Suppression:** Our research shows that TGF- β , produced by tumor-associated macrophages (TAMs), liver cells, and cancer cells, plays a key role in enabling replacement-type lesions to hijack liver vessels and avoid immune detection. This signaling supports vessel co-option and keeps immune cells, especially CD8+ T cells, out of the tumor, helping it grow unchecked.
3. **Blocking TGF- β Reactivates Immune Attack in Resistant Tumors:** In our mouse model, TGF- β inhibition led to tumor damage, structural remodeling, and increased CD8+ T-cell infiltration—effects not seen with standard treatment alone. Depleting TAMs also boosted immune infiltration but didn't improve survival on its own. These findings suggest that targeting TGF- β could enhance immunotherapy specifically for replacement-type CRCLM.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Investigating the effects of alternative energy sources on the metabolism of colorectal cancer CRC with different DNA repair subtypes - Poster #798

Presenter: Shahad Al-Imarah, University of Alberta

Other Authors: Jenilee Woodfield, Melinda Wuest, Frank Wuest, Kristi Baker

ABSTRACT_ID#2348

ABSTRACT

Colorectal cancer (CRC) is classified into different DNA repair subtypes that are categorised into microsatellite instability (MSI) and chromosomal instability (CIN). Studies have shown that MSI tumours tend to exhibit a better prognosis. Due to the substantial energy demands of cancer cells, cancer cells consume alternative energy sources including glutamine (GLN) and short chain fatty acids such as butyrate. The efficacy of cancer cells DNA repair is highly dependent on their metabolic processes. Therefore, we hypothesize that MSI and CIN CRC subtypes utilise different metabolic pathways due to their different DNA repair requirements. To investigate these differences, we exposed samples of CRC subtypes to different levels of glutamine and BUT then analysed their response using Seahorse analyser assays, glutamine radiotracer uptake, metabolomics, Western Blot and real time PCR analysis. Our results show significant effects of low glutamine conditions on CRC cells' metabolism, growth and DNA repair even in conditions where glucose is abundant. Both CRC subtypes expressed higher levels of DNA double strand breaks under low glutamine. The Cancer Genome Atlas shows a significant difference in the expression of glutamine modulators between CRC subtypes including the glutamate/cysteine antiporter (SLC7A11). In collaboration with the Wuest lab, we have tested the CRC subtypes' uptake of glutamate derivative radiotracer [18F] FSPG which is up taken via SLC7A11. Early results showed different patterns of uptake between the subtypes using in vitro and in vivo models. This study may pave the way for enhancing precision and efficacy of treatments and diagnostic tools for CRC patients.

LAY SUMMARY

Colorectal cancer (CRC) is grouped into two main subtypes based on how they repair DNA: Microsatellite instability (MSI) and Chromosomal instability (CIN). MSI-type cancers usually have a better outcome than CIN-type. Cancer cells need a lot of energy, so they use other fuel sources like glutamine (GLN) and butyrate (a short-chain fatty acid). How well cancer cells fix their DNA damage depends on how they use energy. The study's goal was to see if MSI and CIN cancers use energy differently due to their unique DNA repair needs. We grew both CRC subtypes in conditions with different amounts of glutamine and butyrate and used different lab tests to measure how the cells responded. Low glutamine levels affected both CRC subtypes' cell growth, metabolism, and DNA repair, even when glucose was available. Data from The Cancer Genome Atlas showed differences in genes related to glutamine use especially the SLC7A11 gene. Working with our collaborators we have tested how CRC cells took up a glutamate-based imaging tracer, [18F] FSPG, which enters cells through SLC7A11. Early results showed different uptake patterns in MSI vs. CIN cancers. This research could lead to better-targeted treatments and diagnostic tools for CRC patients.

Evaluation of Tissue Platinum Concentration and Immune Cell Population Outcomes after Liver Resection for Metastatic Colorectal Cancer - Poster #799

Presenter: Angela MY Chan, Alberta Health Services

Other Authors: Alexandar S Watson, Md Mahsin, Konstantin Koro, Kata Osz, Holly Wilson, Paola Neri, Wen-Jiang Zhang, Eric X Chen, Nicholas Bosma, Omar Khan, Richard Lee-Ying, Patricia Tang

ABSTRACT_ID#2760

ABSTRACT

Background: Colorectal cancer is the second-leading cause of cancer deaths in Canadian men and the third among women. In oligometastatic colorectal cancer to the liver, surgical resection with curative intent remains possible. Perioperative platinum (oxaliplatin)-based chemotherapy (PC) has shown to provide a disease-free survival (DFS) benefit, although overall survival (OS) benefit remains controversial. Tissue platinum concentrations ([P]) have been associated with improved responses in non-small cell lung cancer and gastric cancer. The predictive value of [P] in oligometastatic colorectal cancer remains unknown. Additionally, PC is known to be immunomodulatory; examination of the hepatic immune tumor microenvironment (iTME) post-PC may provide mechanistic insights and biomarkers of response to help guide clinicians post-hepatectomy. Methods: Employing a state-of-the-art multiplex immunofluorescence (mIF) assay and digital image analysis, we explored the relationship between [P] and the immune landscape. Fifty-four specimens of resected colorectal cancer liver metastases after pre-operative PC had [P] determined with high-performance liquid chromatography mass spectrometry. Tissue microarray was constructed, and state-of-the-art multiplex immunofluorescence (mIF) assay was employed, enabling the simultaneous detection T cells, regulatory T cells, macrophages, and tumor cells in the iTME. Results: By Pearson correlation analyses, there was a moderate association between platinum concentration and levels of cytotoxic T cells, macrophages and PDL1, suggestive of increased cytotoxic inflammation after platinum chemotherapy. Survival analyses are ongoing. Conclusion: This study serves as a proof of concept for integrating spatial immune profiling with tissue pharmacokinetics and can be applied to other tumor types.

LAY SUMMARY

- Cancer surveillance – Combining tissue imaging and measuring drug concentration in tumor tissue may help predict recurrence risk and provide guidance in follow-up care for patients with metastatic colorectal cancer.
- Personalized medicine – Measuring platinum levels together with immune profiling may identify patients more likely to benefit from chemotherapy and may be linked to better survival.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

ANGPTL3, ApoCIII, leptin, and triglycerides are elevated in men with metastatic prostate cancer compared to those with localized Gleason 8 or 9 cancers - Poster #801

Presenter: Anne Gangloff, Laval University

Other Authors: Gabriel Boulay, France-Hélène Joncas, Chloé Castonguay, Jonatan Blais, Frederic Calon, Frederic Pouliot, Vincent Fradet, Alain Bergeron

ABSTRACT_ID#2959

ABSTRACT

Background: Prostate cancer (PCa) cells thrive on abundant lipids, particularly cholesterol, which they utilize to synthesize androgens, thus promoting castration resistance. We hypothesized that metastatic PCa (mPCa) is associated with increased circulating hyperlipidemic factors. Therefore, our objective was to compare circulating PCSK9, ANGPTL3, Leptin and ApoCIII levels in men with mPCa versus localized Gleason 8 or 9 PCa (lPCa). Method: Circulating levels of Lp(a), lipids (total cholesterol, triglycerides, HDL-cholesterol, ApoB), free and total PSA [Roche Cobas analytical platform], LDL and non-HDL cholesterol [calculated], PCSK9, ANGPTL3, ApoCIII and leptin [ELISA] were measured in men diagnosed with mPCa, men with lPCa, and a control group of men at risk of PCa (n=35/group). Analyses of covariance (Gaussian variables) and generalized linear models (non-Gaussian variables) were conducted using JMP Pro 17.2 and SAS 9.4 with age and BMI as covariates in the models. Results: PSA levels increased with disease severity (mPCa 214 +/-395, lPCa 16 +/-15, control 5 +/-3 µg/L). No changes in PCSK9 or Lp(a) levels were observed between groups. Triglycerides (mPCa 2.32 +/-1.17, lPCa 1.47 +/-0.65, control 1.70 +/-1.17 mmol/L), ApoCIII (mPCa 160 +/-97, lPCa 115 +/-58, control 111 +/-56 µg/mL), ANGPTL3 (mPCa 57 +/-27, lPCa 43 +/-24, control 42 +/-20 ng/mL), and leptin (mPCa 17.7 +/-17.8, lPCa 8.2 +/-7.9, control 9.6 +/-9.1 ng/mL) were increased in mPCa. Conclusion: Metastatic PCa was associated with increased levels of hyperlipidemic factors as compared to lPCa or control men. Lipid metabolic rewiring occurs at the metastatic stage in prostate cancer.

LAY SUMMARY

Cholesterol and lipids are essential to prostate cancer progression. In this study, increases in triglycerides, leptin, apo CIII, and ANGPTL3 were only observed at the metastatic stage. These levels were not altered in Gleason 8 or 9 localized prostate cancer compared to control men.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Diet-Driven Vulnerabilities: Targeting Fatty Acid Sensitivity in Burkitt's Lymphoma - Poster #802

Presenter: Audrey L. Collin, Université de Montréal

Other Authors: Léa Bourgignon, Julianna Blagih

ABSTRACT_ID#2481

ABSTRACT

Burkitt's lymphoma (BL) is an aggressive B-cell non-Hodgkin lymphoma associated with genetic mutations, infections, such as the Epstein-Barr virus (EBV), and immunosuppression. To support rapid proliferation, lymphoma cells reprogram their cellular metabolism by increasing glucose and amino acid uptake. Another energy source is fatty acids (FA), comprising saturated fatty acids ranging from short, medium, and long single-bond chains, and unsaturated fatty acids (one or more double bonds). FA originate from dietary sources and the tumour microenvironment, including adipocytes, which play an unknown stromal role in BL. We show that BL cells display metabolic heterogeneity independent of EBV infection status. We tested if BL cells respond differently to medium- and long-chain FAs in cell culture. Despite variability in FA uptake between cell lines, as well as the ability to take up FA from adipocytes, proliferation assays identified three response phenotypes: sensitive, intermediate, and resistant. Consistent with increased FA sensitivity, we observed elevated mitochondrial reactive oxygen species and lipid peroxidation — hallmarks of oxidative stress. Elevated expression of ACSS2 and ACSL1, acyl-CoA synthetases that fuel mitochondrial metabolism, emerged as biomarkers of FAs tolerance. These data suggest a metabolic vulnerability of BL cells to FAs, which can be harnessed through diet. In the transplantable EμMyc lymphoma model, congenic recipient mice were fed diets containing medium-chain (coconut oil), long-chain (palm oil and cocoa butter), or high saturated fats and compared to chow diet. Our preliminary observations suggest that dietary interventions could regulate BL growth, laying the groundwork for considering diet in modulating BL progression.

LAY SUMMARY

Burkitt's lymphoma (BL) is an aggressive cancer of the immune system. While it usually relies on sugar and amino acids for energy, our study found that fatty acids (FAs), especially those from diet or fat cells in the tumor environment, can also feed these cancer cells. BL cells show different levels of sensitivity to FAs. Some are sensitive by them, while others are more resistant. We also found that two specific enzymes, ACSS2 and ACSL1, appear to help cells tolerate FAs and could serve as biomarkers. In mice with lymphoma, changing the diet to include different types of FAs (like coconut oil or palm oil) influenced tumor growth. This opens the door to new ideas about using nutrition as part of cancer care.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Characterization of the tumor microenvironment in pulmonary adenocarcinoma: a new insight into the biology of Spread Through the Alveolar Space - Poster #803

Presenter: Arnaud Driussi, Université Laval

Other Authors: Fabien C. Lamaze, Mark Sorin, Sébastien Renaut, Victoria Saavedra Armeno, Nathalie Gaudreault, Andréanne Gagné, Dominique Boudreau, William Enlow, Yohan Bossé, Logan Walsh, Philippe Joubert

ABSTRACT_ID#3151

ABSTRACT

Introduction: In lung adenocarcinoma (LUAD), Spread Through Air Spaces (STAS) corresponds to the presence of cancer cells within the lung airways beyond the boundaries of the tumor, and is associated with local recurrence and poor prognosis. However, the mechanisms underlying this dissemination remain unknown. Since STAS may represent a mode of tumor spread, understanding its biology is crucial. We hypothesize that STAS results from an alteration in cellular interactions and signaling within the tumor microenvironment (TME). Our objective is to identify TME and transcriptomic features associated with STAS. **Method:** Tissue microarrays comprising 398 tumors were constructed and the TME was characterized using a 35-antibody panel on the Hyperion mass cytometry imaging platform. Following cell type identification, cellular interaction parameters (cellular neighborhoods, CNs) were evaluated and compared between STAS(-) and STAS(+) groups. In parallel, tumor mRNA from 487 patients was sequenced and gene set enrichment analyses (GSEA) were performed to compare the expression of canonical signaling pathways between the groups. Finally, spatial transcriptomics was conducted on 49 tumor samples to localize STAS-associated signatures within the tissue. **Results:** CN analysis indicated a higher frequency of tumor cell clusters and a lower frequency of neutrophil cluster. GSEA identified alterations suggesting extracellular matrix remodeling. This signature was primarily localized to epithelial regions. **Conclusion:** These results highlight a distinct TME and cellular signaling profile in STAS(+) LUAD. Since STAS represents a form of tumor dissemination, identifying the characteristics associated with STAS will enhance our understanding of the biological mechanisms underlying tumor spread in LUAD.

LAY SUMMARY

In some lung cancers, cancer cells can spread beyond the main tumor into nearby air spaces, a pattern called Spread Through Air Spaces (STAS). STAS is linked to higher risk of recurrence and worse outcomes. By analyzing the tumor microenvironment (the surrounding cells and molecules), we found that STAS-positive tumors have more clusters of cancer cells and fewer clusters of immune cells called neutrophils. This suggests changes in how cells interact within these tumors. We also discovered changes indicating a remodeling of the tissue structure around the tumor. These changes were mainly found in areas of the tumor that are in contact with lung airways.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Mapping the tumor-immune landscape in prostate cancer bone metastases - Poster #805

Presenter: Bitá Mojtabehzadeh, University of British Columbia

Other Authors: Felipe Eltit, Raphaële Charest-Morin, Katy Milne, Kassandra Sewell, Nicolette Fonseca, Michael Cox

ABSTRACT_ID#3313

ABSTRACT

The most prevalent complication of advanced prostate cancer (PC) is bone metastases (BM), occurring in >80% of patients, and causing pain, fractures, and spinal cord compression. PCBM present with a distribution of bone density and woven bone accumulation differences (PMID:39224570). How the distinct cancer phenotypes spatially segregate with bone pathology and immune cells in the tumour microenvironment (TME) remains poorly understood. Hypothesis: PCBM tumour phenotypes are heterogeneous, with subtypes causing recurrent bone pathologies, and TME immune and stromal cell distribution differences that may guide intervention strategies. Fourteen PCBM vertebral biopsies collected during spinal decompression surgeries at Vancouver General Hospital were decalcified and assessed for tissue morphology, collagen alignment, and PC phenotype using histology and immunohistochemistry. A 15-marker Akoya PhenoCycler Fusion 2.0 multiplexed immunofluorescence panel was optimized for bone tissue, and used to map TME cell landscape and spatial distribution identified by biomarker expression using established digital imaging tools. Initial results show that PCBM tumor populations present with varying tumor morphologies and biomarkers (PSMA, androgen receptor, synaptophysin) between and within lesions. PC nests localize within the inter-trabecular space, with surrounding vasculature (CD31+) and macrophage/monocyte lineage cells (CD68+). PC cells also present adhered on the endosteum, contacting osteoblasts and CD68+ cells. Multiplexed immunofluorescence-based characterization of PCBM tumour-immune cell interactions as they relate to respective cell characteristics may identify vulnerabilities to help overcome therapeutic resistance.

LAY SUMMARY

Bone metastases are the most prevalent complication of prostate cancer, the most diagnosed male malignancy, with more than >80% of patients developing it. The primary site of bone metastasis is the spine, ribs, and hips, causing changes in bone organization and density, resulting in bone fragility, immense pain, high risk of fractures, and compression of the spinal cord. We know that prostate cancer causes a spectrum of bone lesions; this study aims to characterize the types of prostate cancer presentation across these lesions. A key feature of prostate cancer is a lack of immune response at the site of the tumour. This project evaluates the immune cell variations across prostate cancer bone metastasis tumour subtypes. This study will ultimately help guide the development of new therapeutics for prostate cancer bone metastasis and inform treatment plans for patients. Our results have shown presentation of prostate cancer cells in the bone marrow space, as well as adhered to the surface of the bone. We have identified differing populations of prostate cancer subtypes in the bone in close contact with bone cells. Immune cells have been identified but need to be further subtyped in this space.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Defining the role of thermogenesis in breast tumor development - Poster #806

Presenter: Caroline Knorn, McGill University

Other Authors: Caitlynn Mirabelli, Valerie Sabourin, Elias Maldonado, Jakub Bunk, Lawrence Kazak, Josie Ursini-Siegel

ABSTRACT_ID#2801

ABSTRACT

Breast cancer is the most commonly diagnosed cancer in women worldwide. Despite recent developments in treatment, advanced breast cancer is still considered to be incurable. Breast tumors are incredibly complex due to a variety of factors, including the ability to alter their metabolic processes. For cancer to undergo these metabolic changes, it relies heavily on genetic reprogramming as well as crosstalk between the tumor cells and their local environment. In the case of breast cancer, this local environment is primarily composed of adipose tissue. Adipose tissue is integral to both energy storage and thermal insulation, and is comprised of two distinct types of fat cells: white and brown adipocytes. Notably, brown fat cells play a unique role in regulating thermogenesis, a process by which the body produces heat to maintain its temperature. Upon the activation of thermogenesis, these cells absorb various nutrients from the bloodstream, including sugars and fatty acids, ultimately impacting systemic nutrient levels. However, the exact relationship between thermogenesis and breast cancer development is still unclear. The focus of this project is to explore how thermogenesis is connected to the metabolic reprogramming and development of breast cancer. We will develop a genetically altered mouse model in which thermogenesis can be selectively deactivated in brown adipose tissue. This project is significant because it will allow us to gain an understanding of how modulations in thermogenesis and, in turn, nutrient availability can alter tumor development and progression.

LAY SUMMARY

Chronic induction of thermogenesis appears to potentially impact tumor onset in the context of an aggressive Her2+ luminal B breast cancer cell line. Absence of thermogenesis through a UCP1/CKB double-knockout in brown adipose tissue is potentially tumor promoting in the context of a luminal B breast cancer cell line.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Repurposing angiotensin receptor blockers to target myofibroblast CAFs - Poster #807

Presenter: Che-Min Lee, BC Cancer Research Centre

Other Authors: Nikita Telkar, Rocky Shi, Michael Hall, Kiersten Thomas, Meredith Clark, Ryan Urban, Sarah Hamilton, Wan Lam, Kevin Bennewith

ABSTRACT_ID#2921

ABSTRACT

Cancer associated fibroblasts (CAFs) exist across various solid tumours, with myofibroblast-like CAFs (myCAFs) shown to be pro-tumorigenic and associated with poor cancer therapy efficacy. Currently there are no myCAF targeting drugs that are approved for clinical use. Previously, in a murine tumour model treated with blood pressure medication, telmisartan, we see a decrease in tumoural collagen and improved radiation response (Wadsworth et al., 2020, Cancer Letters). Interestingly, telmisartan did not eliminate CAFs, but decreased expression of a myofibroblast marker, α SMA within the myCAF subset. This indicated that telmisartan specifically inhibits myCAFs. To understand CAF heterogeneity and response to telmisartan, we conducted scRNA-seq (single cell RNA-sequencing) in the same murine tumour model with telmisartan treatment. The scRNA-seq analysis revealed changes in cell identities within the myCAF populations following telmisartan treatment resulting in an inflammatory state arising in the myCAFs population and less fibrillar collagens and ECM-related myCAFs within the telmisartan-treated cells. To see if cancer patients on telmisartan, or other angiotensin receptor blockers (ARB), had improved patient outcomes, we conducted a retrospective study on a cohort of oropharyngeal squamous cell carcinoma patients in British Columbia. When comparing the ARB users to a propensity score matched group of patients not on antihypertensives, we found that ARB users had significantly improved overall survival, disease-specific survival, and locoregional control (Lee et al., 2025, JNCI). Overall, this work shows that telmisartan can effectively target myCAFs and telmisartan may be a useful adjuvant therapy in the clinic to ameliorate the effects of existing cancer therapeutic.

LAY SUMMARY

Solid cancer tumours contain non-cancerous cells that impact cancer therapy efficacy. These include cancer associated fibroblasts, or CAFs. One type of CAF, called myCAFs, are like the construction workers of the tumour that help build the tumour. When myCAFs are too active and lay down too much material (such as collagen), this can cause tumour stiffness. Stiff tumours respond poorly to cancer therapy as stiffness prevents drugs from reaching and killing cancer cells. Currently, we do not have any drugs that can make myCAFs less active. One possible candidate for targeting myCAFs is the a commonly prescribed blood pressure drug family called angiotensin receptor blockers (ARBs). We show in various experiments that one drug in the ARB family, called telmisartan (brand name Micardis), can target myCAFs to decrease collagen in tumours not by killing myCAFs but by dampening myCAF activity in a mouse model of cancer. Additionally, we did a study on existing data from head and neck cancer patients who were on ARBs prior to their cancer diagnosis and found that ARB users had higher survival outcomes. Overall, we show that telmisartan may a useful drug to use alongside current cancer therapies to improve cancer patient survival outcomes.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Exploring the relationship between mRNA quality control and cancer immunogenicity - Poster #808

Presenter: Dana Peters, University of Alberta

Other Authors: Paul LaPointe, Matthew Stiegelmar, Isabelle Sirois, Kristi Baker

ABSTRACT_ID#3221

ABSTRACT

Immunotherapies have come to the forefront of cancer research in recent years due to their efficacy in late stage and metastatic cancers. However, due to a lack of immunogenicity, some patients fail to respond to these treatments. To diversify the repertoire of antigens presented on cancer cells, we inhibit nonsense-mediated decay (NMD), an mRNA quality control mechanism that removes transcripts containing premature termination codons (PTCs). Since PTCs can arise from frameshift mutations, it is expected that inhibiting this mechanism will increase the production of novel antigens and therefore strengthen the immune response to cancer. In this study, we knock down NMD in breast cancer cells using siRNAs for several proteins involved in NMD, PTC-readthrough compounds, and drugs neutralizing NMD proteins. Using immunopeptidome profiling of the major histocompatibility complex I (MHC I), we determine that cells with a reduced capacity for NMD display a different set of antigens than the control group. Further, antigens matching sequences derived from the +2 and +3 reading frames were detected in the immunopeptidome. Such peptides would be unlikely to be associated with tolerance and should in theory result in an increase in immunogenicity. Overall, the data implies that the NMD pathway could be used as a novel immunotherapy target, and could be especially effective when used in conjunction with other existing therapies that further enhance the immune response to cancer.

LAY SUMMARY

Quality control systems shield cancer cells from the immune system Inhibiting quality control mechanisms may enhance the efficacy of cancer immunotherapies

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Integrative single cell analysis of CD8+ T-cells across early and advanced oral cancers reveal signatures of anti-tumour activity - Poster #809

Presenter: Constance Li, University of Calgary

Other Authors: Hong Sheng Quah, Camille Arcinas, Lisda Suteja, Fui Teen Chong, Hui Sun Leong, Darren Toh, Cliburn Chan, Gopalakrishna Iyer

ABSTRACT_ID#3047

ABSTRACT

Tumour-infiltrating lymphocytes play a critical role in anti-tumour immunity, but their function is frequently suppressed in the tumour microenvironment. Cytotoxic T lymphocytes (CD8+ T-cells) are of particular interest due to their primary function of identifying and eliminating tumour cells. Indeed, key immunotherapies rely on immune checkpoint blockade (anti-PD-1, anti-CTLA-4) or engineered cells (CAR-T) to stimulate anti-tumour cytotoxicity. While the dynamics of tumour-infiltrating CD8+ T-cell populations are well-described, less is known about nuances in those dynamics through metastatic disease. Moreover, the identification of CD8+ T-cells with anti-tumour activity is a specialized endeavour with high materials and sample costs. There is subsequently a need for additional high accuracy methods of identifying tumour-targeting CD8+ T-cells. We investigated CD8+ T-cell dynamics in oral cavity squamous cell cancers through primary, nodal, and distant metastatic disease. We generated matched single cell RNA (scRNA-seq) and T-cell receptor (scTCR-seq) profiles in 40,356 CD8+ T-cells purified from 28 resected and biopsied tissues of 17 patients. Using molecular profiles describing multiple disease stages, we traced CD8+ T-cell differentiation through cancer progression and treatment in both inter- and intra-patient analysis, and describe sub-populations of CD8+ T-cell states. We analysed patterns of T-cell receptor diversity and tracked clonal expansion between tumour, lymph and recurrence tissues. Critically, we integrated this cross-sample, multi-omics profiling to identify tumour-targeting CD8+ T-cells, and developed a 215-gene random forest classifier to predict tumour targeting based on gene expression. Together, this work deepens our understanding of cytotoxic T lymphocyte dynamics and will help in accelerating advancements in immunotherapy development.

LAY SUMMARY

Single cell sequencing reveals the variable landscape of tumour-infiltrating CD8+ T-cells between primary site, lymph node, and recurrent site tissues. Clonotype tracking between multiple tissues from the same patient shows the majority of expanded CD8+ T-cell clones in primary tumour are shared with metastatic tissues (nodal and/or distant). The expansion of unshared clonotypes is detected in metastatic tumours following immune checkpoint blockade, suggesting response to new neoantigens. Clonally expanded CD8+ T-cells that are also shared between primary and metastatic tumours are likely tumour-targeting. Using a TCR-seq-derived definition to label tumour-targeting CD8+ T-cells, we trained a random forest to classify them based on matched scRNA-seq data. Our random forest model achieves an overall classification accuracy of 79%, with 76% precision and 51% recall on the internal hold out test set. While there are no external datasets with suitable scRNA-seq and scTCR-seq to form a labelled validation set, our classifier has a detection prevalence of up to 15% in external scRNA-seq data, which is within range of detection seen in our data. Additional work is needed to validate this single cell-based classifier and adapt its use for bulk RNA data.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

The Impact of platelet-derived microparticles on hypoxia-induced EMT in breast cancer - Poster #810

Presenter: Isabelle Violette, Atlantic Cancer Research Institute

Other Authors: Vanessa Veilleux, Gilles Robichaud

ABSTRACT_ID#2386

ABSTRACT

Metastasis is the leading cause of mortality in patients with breast cancer. As tumors grow rapidly, angiogenesis fails to keep pace resulting in insufficient oxygen supply, a condition known as hypoxia. Tumor hypoxia is considered as a major driver of the epithelial-to-mesenchymal transition (EMT), which promotes malignant processes (migration and invasion), metastasis and disease progression. Recent studies suggest that hypoxia and EMT potentially share common signaling pathways. Additionally, our team has recently reported that platelet-derived microparticles (PMPs), which are small extracellular vesicles released from activated platelets, interact and internalize into breast cancer cells and release their biological cargo. Our preliminary data also show that these interactions induce breast cancer aggressivity (migration and invasion) and hypoxic gene expression profiles in normal oxygen levels (normoxia). The objective of our study was to investigate the impact of PMPs on hypoxia-induced EMT in breast cancer to gain a better understanding of PMPs and the tumor environment. Breast cancer cell lines were therefore co-cultured with PMPs and treated under various hypoxic conditions followed by the analysis of EMT-related gene expression and hypoxic signaling using RT-qPCR. In addition, PMPs regulated aggressive cancer cell behavior during hypoxia. Our findings demonstrate that PMPs exacerbate hypoxia-induced EMT signaling thus suggesting an important role for PMPs in tumor malignancy. This research provides new insights into the mechanisms regulating the tumor environment and malignancy. Our findings also present new avenues (i.e., PMP blockade) for the development of more effective and strategic therapeutic interventions against breast cancer progression.

LAY SUMMARY

PMPs synergistically contribute to breast cancer aggressivity in hypoxic conditions. We map new mechanisms driving breast cancer aggressivity which could be targeted by new strategic therapeutic interventions.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Understanding the impact of the p66ShcA/PKR axis on the evolution of a pro-tumorigenic immune microenvironment in breast cancer - Poster #811

Presenter: Emily McIsaac, McGill University

Other Authors: Elias Maldonado, Rachel La Selva, Steven Hebert, John Heath, Ryuhjin Ahn, Claudia L Kleinman, Josie Ursini-Siegel

ABSTRACT_ID#2501

ABSTRACT

Breast cancer is a complex heterogenous disease affecting 1 in 8 women in Canada, with a low survival rate for aggressive cases. Cells of the tumor immune microenvironment (TIME) have been highlighted as key components of disease, play complex roles in tumor progression. One factor influencing the immune response in tumors are local stress responses, such as oxidative stress and the integrated stress response. Our lab has shown p66ShcA, PKR, and eIF2 α to play oncogenic roles by promoting tumor progression and metastasis. Previous data generated in the lab has also shown distinct inflammatory responses in these aggressive tumor models. Together, this data provides evidence that stress responses are important in modulating the TIME. My project aims to define the specific immune landscapes promoting pro-tumorigenic inflammation and tumor progression. This will be done by performing flow cytometry and CODEX multiplex staining to get a general, and spatial overview of immune populations present in the tumors. My project objectives will improve upon our current understanding of how stress responses influence inflammation and breast cancer progression.

LAY SUMMARY

Chronic levels of oxidative stress increases tumor onset, progression, and metastasis contributing to tumor aggressiveness and progression. The integrated stress response plays an oncogenic role by promoting tumor progression and metastasis. Altered oxidative stress and integrated stress response induce a change in immune populations recruited to the site of the tumor.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Development and Characterization of a Patient-Derived Organoid Model of NSCLC Brain Metastasis - Poster #812

Presenter: Emily Niu, University of Calgary

Other Authors: Maryam Al-Witry, Robbie McTaggart-Cowan, Erin Lee, Hyein Jang, Karen Blote, Martha Hughes, Shrivani Pirahas, John Kelly

ABSTRACT_ID#3251

ABSTRACT

Non-small cell lung cancer (NSCLC) is the leading cause of death in Canada, where 30,000 people are diagnosed per year. NSCLC has a propensity to spread to the brain and brain metastases are typically incurable, highlighting an urgent need for new treatment options. Much of what we know of cancer come from cultured cell model studies. Unfortunately, cell models do not completely reflect tumor behavior due to their uniformness, so other models are needed. Patient-derived organoids (PDOs) are in vitro, 3-dimensional (3D) structures consisting of self-organizing, organ-specific cell types derived from a patient's tumor tissue. They can be established in a short time and has been shown to mimic key features of a patient's tumor. They have many uses, but most importantly, may help in developing personalized treatment regimens. Perhaps due to the difficulty in accessing metastatic tumor tissues, there are limited PDO models of NSCLC BMs. In this project, we established PDOs from NSCLC BMs and studied their growth and metabolic properties. We found that these PDOs tend to grow variably, show a limited degree of growth after cryopreservation, and grow better after cryopreservation in hypoxia. Phenotypic characterization shows retention of key NSCLC and cancer stem cell markers, with the growth property of NSCLC BM PDOs tending to correlate with increased glycolytic activity. Additionally, we found NSCLC cells showed response to metabolic inhibitors. Overall, this study has enhanced our understanding of NSCLC BM PDO development and growth characteristics that can be used to further interrogate this disease.

LAY SUMMARY

Patient-derived organoids (PDOs) are tiny, patient-derived, three-dimensional tissue constructs which grow quickly in a culture dish and can mimic tumor tissues, even over long-term culture. Due to the challenge of obtaining brain metastases tissues from patients with non-small cell lung cancer (NSCLC), there have been exceedingly few PDOs models of NSCLC BMs. Our work aims to create a PDO model of NSCLC BMs. Key messages: From our work, we found that these NSCLC BM PDOs generally resembled the tissue from which they were derived from. These PDOs maintain adenocarcinoma, cancer stem cell, and proliferation ability of their parent tumors. NSCLC BM PDOs show key differences in their growth: two samples grew infinitely, six samples grew finitely, and two samples failed to grow. NSCLC BM PDOs show distinct metabolic differences across patient samples, with infinitely growing NSCLC BM cell lines showing increased proliferation, utilizing both glycolysis and oxidative metabolism pathways.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Role of CLAUDIN-1 in Drug Adaptation in HER2+ Brain Metastatic Breast Cancer (BCBM) Cells - Poster #813

Presenter: Adline Fernando, University of Manitoba

Other Authors: Danyyl Ippolitov, Sean Robertson, Olivia Wilkins, Victor Spicer, Rene Zahedi, Sabine Hombach-Klonisch, Thomas Klonisch

ABSTRACT_ID#2969

ABSTRACT

Introduction: HER2+ breast cancer (BC) frequently metastasizes to the brain, where effective therapy is limited by the blood-brain barrier and rapid emergence of drug resistance. We have identified the covalent HER inhibitor Pozotinib as highly efficacious in reducing tumor burden in the brain. However, the molecular mechanisms underlying treatment adaptation remain unclear. Claudin-1 (CLDN1), a tight junction protein with emerging roles in oncogenic signaling, remains poorly characterized in BC brain metastasis (BCBM). Methods: We generated a patient-derived HER2+ brain metastasis model and pozotinib-resistant (PozR) cells. Single cell RNAseq and total-/phospho-proteomic analysis was performed on sensitive and PozR cells. Western blot, quantitative PCR and immunofluorescence were used to validate differences in gene and protein expression and localization. CRISPR/Cas shRNA and siRNA methodology allowed for knockdown (KD) of CLDN1 and HER3. Functional assays included cell migration, viability and colony formation assays. Results: CLDN1 was one of the most upregulated proteins, showing an increase of 17-fold in total protein and 60-fold for phospho-CLDN1Y210 by quantitative (phospho-)proteomic analysis, and validated by immunoblotting. Super-resolution microscopy showed membrane localization of pCLDN1Y210 in PozR cells. scRNAseq data revealed that acute Poz exposure (24 h) in sensitive cells already caused a shift towards a CLDN1^{high} subpopulation that was abundant in PozR cells. CLDN1 KD significantly reduced migration. siHER3 silencing and SRC inhibition reduced CLDN1 expression and phosphorylation. CLDN7 silencing increased CLDN1 transcripts and protein levels in PozR cells indicating compensatory regulation. Summary: Our data position CLDN1 as a dynamically regulated and functionally relevant protein in drug-adapted HER2+ BCBM.

LAY SUMMARY

Breast cancer that spreads to the brain is very hard to treat, especially when it stops responding to drugs. In our research, we looked at why some brain tumor cells become resistant to a drug called Pozotinib. We found that a protein called Claudin-1 becomes much more active in these resistant cells, especially in a switched-on form that moves to the cell surface. This increase happened quickly, even after just one day of drug exposure. When we reduced Claudin-1, the resistant cells moved around less, which suggests it helps cancer cells spread. We also found that blocking other cancer signals like HER3 or SRC lowered Claudin-1 levels. Another protein, Claudin-7, also played a role. When Claudin-7 was reduced, Claudin-1 went up. These results show that Claudin-1 is closely controlled by different cancer pathways and may help tumors adapt when treatment stops working. Understanding how these proteins interact could reveal new ways to stop cancer cells from becoming drug resistant and spreading further, especially in patients with few treatment options.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Advancing In Vitro Tumour Vascular Models: Culture Optimization and Morphological Analysis of Microvascular Networks - Poster #814

Presenter: Edmond Young, University of Toronto

Other Authors: Han Shao

ABSTRACT_ID#2850

ABSTRACT

Understanding microvascular networks (MVNs) is critical in cancer biology, as tumour progression, metastasis, and therapeutic response are influenced by the structure and function of the tumour vasculature. Recently, microfluidic systems have emerged to enable the formation of perfusable MVNs in vitro by facilitating self-assembly of endothelial cells into MVNs through supporting fibroblasts, angiogenic factors and mechanical cues. However, optimal culture conditions for generating physiologically relevant MVNs are not known, and the resulting differences in MVN morphology across protocols are not well defined. To optimize MVNs for cancer research, we studied effects of fibroblast concentration, fibroblast conditioned media, angiogenic factors, and luminal flow on the morphology, perfusability, and permeability of MVNs cultured in a microfluidic system. To quantitatively assess MVN morphology, we defined novel void-based metrics (i.e., metrics of the empty spaces between vessel branches) in addition to traditional vessel-based metrics. Overall, we observed that high fibroblast concentration increased MVN growth rate and led to excessive vessel fusion. MVNs cultured without fibroblasts but with either fibroblast conditioned media or angiogenic factors developed patch-like morphology instead of physiological thin vessel branches. Thus, fibroblasts are crucial for MVN development and cannot be substituted by fibroblast conditioned media or angiogenic factors. Void-based metrics provide an efficient alternative to traditional branch-based metrics that yields higher information density per metric while reducing data volume for machine learning and AI-assisted image analyses. The optimal MVNs cultured in this study have potential to be adapted into a 3D microfluidic tumour model for studying non-angiogenic tumour vascularization.

LAY SUMMARY

- Microfluidic devices facilitate the formation of 3D perfusable microvascular networks that can be exploited to study tumour-vascular interactions.
- Fibroblasts at optimal concentrations are crucial for supporting microvascular network formation and long-term stability.
- A combination of traditional vessel-based metrics and novel void-based metrics provides a rich quantitative approach to describing network morphology.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Perfusable microfluidic tumour model with encircling 3D microvascular network for studying tumour cell infiltration and vessel co-option - Poster #816

Presenter: Edmond Young, University of Toronto

Other Authors: Han Shao

ABSTRACT_ID#2851

ABSTRACT

Understanding tumour vascularization is crucial for developing treatments to suppress tumour growth. While anti-VEGF therapies aim to block tumour angiogenesis, their clinical efficacy is limited due to treatment resistance. Tumours in a vasculature-rich microenvironment can bypass angiogenesis by hijacking existing blood vessels through vessel co-option, a process linked to increased tumour cell motility and metastasis. However, the molecular mechanisms driving vessel co-option remain poorly understood. To investigate tumour-vascular interactions, we developed a novel microfluidic 3D vascularized tumour model consisting of a central tumour compartment surrounded by a fibrin-gel-laden microvascular network (MVN) that encircles the tumour. The MVN, formed by co-culturing endothelial cells and fibroblasts in a fibrin matrix, was flanked by two perfusable side channels to support vessel maturation. After MVN formation, lung or breast cancer cells were seeded into the tumour compartment. We assessed tumour-induced vascular changes by perfusing fluorescent dextran to measure vessel permeability and quantifying angiogenic sprouts directed toward the tumour. Confocal microscopy was used to evaluate tumour cell infiltration, vessel co-option, and infiltration distances. Future experiments will deliver the anti-VEGF drug bevacizumab to study resistance mechanisms, including vessel co-option and drug-induced tumour cell motility. This microfluidic platform provides a physiologically relevant environment to examine dynamic tumour-vascular interactions and to uncover how tumours adapt their vascularization strategies under anti-angiogenic pressure.

LAY SUMMARY

- A novel microfluidic system was created that mimics tumour-vascular interactions by incorporating a central tumour surrounded by a perfusable, fibroblast-supported microvascular network.
- The model enables detailed analysis of vessel permeability, angiogenic sprouting, tumour cell infiltration, and vessel co-option using fluorescent dextran perfusion and confocal microscopy.
- Future use of the model for drug testing will allow investigation into how tumours resist anti-VEGF therapy via vessel co-option and increased cell motility, addressing a gap in understanding drug resistance.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Transcriptional and Functional RNA Epitranscriptome in Cancer - Poster #817

Presenter: Housheng Hansen He, Princess Margaret Cancer Centre, University Health Network

Other Authors: Xin Xu, Shiyang Wang, Yujuan Wang, Yong Zeng, Julie Livingstone, Rupert Hugh-White, Helen Zhu, Ming Tsao, Paul Boutros

ABSTRACT_ID#2619

ABSTRACT

N6-methyladenosine (m6A) is the most abundant internal RNA modification and a key regulator of RNA stability, splicing, and translation. Despite its biological significance, comprehensive and functional mapping of m6A in patient tumors has been hindered by input limitations. To overcome this, we developed a low-input m6A MeRIP-seq protocol optimized for nanogram-scale RNA, enabling robust epitranscriptomic profiling in clinical specimens. Using this platform, we analyzed 162 localized prostate tumors and 51 lung adenocarcinomas with matched genomic, transcriptomic, proteomic, and clinical data. We identified over 30,000 high-confidence m6A sites, spanning 30–40% of expressed genes. Prostate and lung cancers exhibited global hypermethylation and hypomethylation, respectively, with tumor-specific m6A patterns shaped by germline variants, somatic mutations, and microenvironmental factors. In prostate cancer, global and site-specific m6A patterns were linked to hypoxia and aggressive phenotypes. In lung cancer, we identified 652 differentially methylated transcripts, including EML4, a hypermethylated metastasis driver, and pharmacologic inhibition of m6A suppressed EML4 protein and metastasis in vivo. To systematically identify functional m6A sites, we conducted a high-throughput RNA base-editing screen targeting 2,120 sites using a 12,000 sgRNA library. This revealed hundreds of essential epitranscriptomic regulators of tumor growth. Together, our study presents the first large-scale functional atlas of m6A modifications in primary tumors, defines conserved and cancer-specific m6A programs in prostate and lung cancers, and highlights RNA epitranscriptomic vulnerabilities as novel targets for cancer therapy.

LAY SUMMARY

We developed a sensitive technique to map m6A RNA modifications using very small amounts of tumor tissue, making it possible to study patient samples. Prostate tumors tend to have more m6A marks (hypermethylation), while lung tumors often have fewer (hypomethylation). These RNA changes are linked to inherited traits, tumor mutations, and environmental factors like low oxygen. By editing thousands of m6A sites, we found many that are critical for tumor growth. One such gene, EML4, helps cancer spread and can be turned off with a drug that blocks m6A, pointing to new treatment strategies.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

NOTUM Depletion Restores CD8+ T Cell-Mediated Removal of Colorectal Cancer Cells - Poster #819

Presenter: Julian Chua, University of Calgary

Other Authors: Parnika Sakthivel, Arshdeep Kaur, Elleine Allapitan, Oliver Bathe, Parham Minoo, Arshad Ayyaz

ABSTRACT_ID#2540

ABSTRACT

Colorectal cancer (CRC) is expected to be the fourth most diagnosed cancer and is currently the 2nd leading cause of cancer related deaths in men and 3rd in women in Canada. While immunotherapy has presented promising results against microsatellite high (MSI-H) CRC, the more common subtype, microsatellite stable (MSS) CRC which represents nearly 85% of all CRC cases, is unresponsive to immunotherapy. In this study (Chua et al., 2025 bioRxiv) we aim to elucidate mechanisms contributing to this immunotherapy resistant phenotype. Through comparative single-cell genomic analysis of 160 primary human CRC tumors across 5 datasets I identified a novel cell type unique to MSS CRCs. These cells, which we named WNT inhibitory cancer cells (WICs), evade cytotoxic T cell-mediated recognition by expressing immunotherapy resistant genes such as Notum and Apccdd1. To test the impact of these cells, WICs were ablated using an inducible iCaspase9 system under the control of the Apccdd1 promotor in AKP cOVA mouse derived organoids. This was followed with an adoptive cell therapy model using OT-1 T cells, primed for cOVA. Ablation of this WICs with the addition of adoptive cell therapy treatment significantly increased cancer cell death in AKP cOVA organoids in comparison to organoids containing WICs. Importantly, we have identified NOTUM as a key modulator which inhibits CD8+ T cell-mediated elimination of CRC cells. This project thus reveals a novel cell type unique to immunotherapy resistant type of bowel cancers, identifying new therapeutic targets to improve immunotherapy outcomes for the majority of CRC patients.

LAY SUMMARY

1. Identification of a previously unknown cancer cell type called WNT inhibitory cancer cells (WICs), that is only found in immunotherapy-resistant MSS CRC tumors. These cells enhance tumor immunotherapy resistance by expressing WNT inhibitory genes like Notum and Apccdd1.
2. Selective ablation of WICs with adoptive cell therapy in intestinal cancer organoids significantly increased cell death in comparison. This suggests that removing these resistant cells could make immunotherapies more effective against MSS CRC.
3. NOTUM is a key modulator, which inhibits CD8+ T cell-mediated elimination of colorectal cancer cells.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Lysyl Oxidase Promotes Mesenchymal-to-Epithelial Transitions in Triple Negative Breast Cancer Cells Which May Be Associated with Metastatic Tumour Formation - Poster #820

Presenter: Grant Kelly, Memorial University of Newfoundland

Other Authors: Eric Mensah, Shahbin Hossain, Alicia Vilorio-Petit, Sherri Christian

ABSTRACT_ID#2825

ABSTRACT

Obesity is a major risk factor for the development and progression of breast cancer. One important facet of obesity is the crosstalk between adipocytes and breast cancer cells. We have previously shown that co-culture of breast cancer cells with adipocytes results in a mesenchymal-to-epithelial transition (MET). MET is a pivotal step in the latter stages of metastasis, enabling migratory cancer cells to colonize distal tissue. We previously identified that the adipocyte-secreted protein LOX has the potential to cause the observed MET. LOX is an amine oxidase that stiffens the extracellular matrix. It is secreted as a proprotein and must be activated through cleavage by either BMP1 or ADAMTS2. However, the mechanism underlying the regulation of MET by LOX is not understood. We have found that chemical or genetic inhibition of LOX suppresses MET in breast cancer cells, suggesting a critical role for LOX, BMP1 and/or ADAMTS2 in MET. To determine this, we will use CRISPR/Cas9 gene silencing and chemical inhibition to determine if BMP1 or ADAMTS2 are also necessary for MET. Furthermore, transcriptomic changes in adipocytes and breast cancer cells in co-culture will be analyzed using single cell RNA-seq. Moreover, the role of LOX in promoting metastasis in vivo will be analyzed. Better understanding the role and effects of LOX, BMP1, and ADAMTS2 in breast cancer MET will provide foundational understanding of the influence obesity has on tumour metastasis. Future work may uncover novel therapies to prevent metastasis in high-risk patients before it occurs.

LAY SUMMARY

1. Better understanding of how obesity influences metastasis.
2. New candidates for therapeutics in high metastatic risk patients
3. Clearer picture of the environment surrounding metastatic tumours.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Gene expression profiling of primary and metastatic tumors following different PARP inhibitor sequencing strategies with carboplatin in a triple-negative breast cancer xenograft model - Poster #821

Presenter: Kenan Aloss, Centre de Recherche de Centre hospitalier de l'Université de Montréal (CRCHUM)

Other Authors: Shu Feng Zhou, Mallory I Frederick, Saima Hassan

ABSTRACT_ID#2740

ABSTRACT

Introduction: Triple-negative breast cancer (TNBC) lacks overexpression of hormone receptors and HER2, limiting therapeutic options. Despite the improved outcomes for patients with early TNBC, patients benefit from only one targeted therapeutic agent: PARP inhibitors (PARPi). PARPi target PARP-1/2, inducing synthetic lethality in BRCA1/2-mutant contexts, or via PARP-DNA trapping. While PARPi such as talazoparib and olaparib have shown improved outcomes in BRCA-mutant TNBC, pre-clinical efficacy has also been demonstrated amongst BRCA-wild type tumors. We have demonstrated that the sequential combination of talazoparib-first, followed by carboplatin (Seq T->C) markedly inhibited migration, invasion, and lung metastasis. Therefore, we wanted to investigate the mechanism of efficacy of the sequential combination approach. Methods: We performed RNAseq gene expression analysis on the metastatic lung and primary tumors from MDAMB231 orthotopic xenograft mouse model, to compare concomitant talazoparib and carboplatin (Conc T+C), Seq T->C, and carboplatin-first followed by talazoparib (Seq C->T) approaches. Results: Principal component analysis showed that Seq C->T was grouped closely with Conc T+C in the primary tumor and metastatic lung. Seq T->C was associated with a downregulation of the homologous recombination, DNA replication, and mismatch repair pathways in the metastatic lung. Both Seq C->T and Conc T+C upregulated pathways, related to epithelial-mesenchymal transition, angiogenesis, and inflammatory response in the primary tumor. Conclusion: The Seq T->C approach exclusively downregulated DNA damage response pathways at the metastatic site. The Seq C->T and Conc T+C approaches both upregulated pathways associated with treatment resistance. Therefore, the Seq T->C strategy may be effective to inhibit distant metastasis

LAY SUMMARY

Treatment of triple-negative breast cancer (TNBC) is challenging due to the lack of targeted therapies. Currently, PARP inhibitors (PARPi), such as talazoparib and olaparib, are the only class of targeted therapy that benefit TNBC patients. We previously demonstrated that administering talazoparib first, followed by carboplatin, significantly reduced cancer migration and lung metastasis in a TNBC mouse model compared to other combination strategies. In this study, we investigated the mechanism behind this enhanced efficacy. We found that the sequence of talazoparib followed by carboplatin inhibited DNA repair pathways in metastatic lungs. These findings suggest that this treatment sequence may be a promising strategy to inhibit the development of distant metastasis.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Modeling the Tumor Microenvironment: A 3D Co-culture Platform to Study Phenotypic and Epigenetic Reprogramming in Lung Cancer - Poster #822

Presenter: Harshini Renganathan, Universite de Montreal

ABSTRACT_ID#3300

ABSTRACT

Lung cancer remains the leading cause of cancer-related mortality in Canada, with non-small cell lung cancer (NSCLC) accounting for the majority of cases. Despite ongoing therapeutic advances, NSCLC patients with advanced disease still face a five-year survival rate below 5%. A major obstacle in drug development is the limited predictive power of conventional two-dimensional (2D) culture systems, which fail to replicate the complexity of the tumor microenvironment (TME). Our study aims to establish physiologically relevant 3D co-culture spheroid models using NSCLC cells (A549) and lung fibroblasts (MRC-5) to better understand tumor-stroma interactions, with a focus on transcriptomic, phenotypic, and epigenetic reprogramming. We optimize co-culture conditions using media mix of F12K and EMEM for the 3D spheroid growth. Spheroid architecture is tracked over time via fluorescence imaging, while cell viability and proliferation are assessed by live/dead staining, Ki-67 immunostaining, and flow cytometry. We evaluate tumor and CAF markers (CEACAM6, α -SMA, COL1A1, FAP) and epigenetic regulators (DNMT1, TET1, H3K27me3, H3K9ac) using Western blot and confocal microscopy. Paracrine signaling is profiled via multiplex ELISA to profile a series of cytokines. Single-cell ATAC-seq and spatial RNA-seq are employed to resolve chromatin state changes during 3D co-culture and tumor-stroma crosstalk, respectively. Finally, drug screening with natural and epigenetic compounds is performed in 3D monoculture and 3D coculture to decipher treatment targeting both cell types. This project delivers a novel 3D platform for studying NSCLC tumor microenvironment and stromal contributions, especially CAFs (cancer-associated fibroblasts), with the potential to improve translational drug discovery and precision oncology strategies.

LAY SUMMARY

We created a 3D lung cancer model that more closely mimics how tumors grow and behave in the human body by combining cancer cells with supportive cells called fibroblasts. This helps us study how these cells "talk" to each other and influence tumor growth and drug resistance. We found that these supportive fibroblasts change their behavior in 3D conditions, becoming more aggressive and promoting cancer progression by modifying the surrounding environment and activating cancer-related genes and epigenetic changes (how genes are turned on/off without changing DNA). This model helps identify better drug targets by showing how cancer and fibroblast interactions affect treatment response. We will be testing natural and epigenetic drugs in this model and comparing results with real patient data to identify treatments that could be more effective in the clinic.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Characterization of Two Novel Immune Competent, Murine Models of Soft Tissue Sarcoma - Poster #823

Presenter: Kurt Hildebrand, University of Calgary

Other Authors: Carolina Salazar Arcila, Franz Zemp, Kiran Narta, Ted Verhey, Hyojin Song, Arvind Singla, Lisa Difranceso, Ashley Flaman, Mara Caragea, Roman Krawetz, Douglas Mahoney, Sorana Morrissy, Michael Monument

ABSTRACT_ID#3123

ABSTRACT

Soft tissue sarcomas (STS) are mesenchymal lineage tumours that are resistant to systemic therapies. Unfortunately, metastatic STS is fatal, thus in desperate need of novel treatments. To achieve a stronger understanding of STS biology and therapeutic interventions, pre-clinical mouse models are essential. Herein, we developed two mouse models on a C57Bl/6 background; 1) Trp53fl/fl; Ptenfl/fl; KrasG12D/+ mice, and 2) Trp53fl/fl; Ptenfl/fl mice. Tumours were induced by performing a subperiosteal scratch to the proximal-lateral tibia followed by injection of a CMV-driven cre-recombinase plasmid (lenti-cre). One female inducible primary tumour from each group was processed into a cell line and engrafted right hindlimb C57Bl/6 mice to develop syngeneic tumours. Inducible and syngeneic tumours were harvested at a volume of 500-800mm³ to be assessed by flow cytometry, histology, and bulk RNA sequencing. Trp53fl/fl; Ptenfl/fl; KrasG12D/+ inducible tumours developed in 30-40-days, whereas Trp53fl/fl; Ptenfl/fl tumours developed between 100-350 days. Syngeneic tumours from both models developed in 2-3-weeks. Inducible and syngeneic tumours from both novel models resembled undifferentiated pleomorphic sarcoma (UPS) based on histology. However, assessing sarcoma diagnostics using bulk RNA sequencing yielded more heterogeneous results. In assessing immune infiltrates, flow cytometry revealed that myeloid cells and macrophages are the most prevalent immune cells across all models, with a small proportion of lymphocytes. Lastly, the syngeneic models did not respond to anti-PD1 immune checkpoint blockade. These novel murine models of STS recapitulate the attributes of the human disease, providing immune competent models to study STS biology and novel therapies.

LAY SUMMARY

- To achieve a stronger understanding of tumour biology and therapeutic interventions, pre-clinical mouse models are essential.
- We developed two novel murine models of STS on a C57Bl/6 background; Trp53fl/fl; Ptenfl/fl; KrasG12D/+ and Trp53fl/fl; Ptenfl/fl.
- Cre-recombinase induced tumours and syngeneic tumours recapitulate the characteristics of human STS, such as rapid growth, histological features, abundance of myeloid cells, few lymphocytes, and poor response to anti-PD1 immunotherapy. These provide two novel models to study STS biology and novel therapies.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

The Influence of Interleukin-6 on Macrophage and Tumor Activity in Different Colorectal Cancer Subtypes - Poster #824

Presenter: Taylor Holtom, University of Alberta

Other Authors: Kayleigh Morris, Kristi Baker

ABSTRACT_ID#2522

ABSTRACT

The two most common subtypes of colorectal cancer (CRC) are microsatellite instable (MSI) and chromosome instable (CIN). The MSI subtype promotes a more immunogenic microenvironment relative to the CIN subtype. Key contributors to this environmental shift are macrophages - innate immune cells involved in the initial response to cellular damage. M1 macrophages secrete and express more tumor-inhibiting factors than their M2 counterparts, but whether their activities change in different CRC types is unclear. We focused on how interleukin-6 (IL-6), a pro-inflammatory cytokine produced by CRCs and macrophages, may change tumor-macrophage interactions. Previous data indicated CIN CRCs produce more IL-6 than MSI CRCs. We hypothesized that IL-6 differentially regulates tumor-macrophage interactions in MSI and CINs CRCs. We first assessed if IL-6 changed the phenotype of bone marrow derived macrophages (BMDMs). We next used multiple in vitro models to test if IL-6 changed macrophage migration towards CRCs. We finally isolated macrophage and CRC proteins from co-cultures to identify IL-6-dependent signaling pathways. We found that IL-6 is sufficient to induce an M2 phenotype in otherwise unpolarized (M0) macrophages. We noted that blocking IL-6 increased the migration of all macrophage subtypes towards CRCs. Finally, we saw that IL-6 regulates the phosphorylation of STAT3 in CIN CRCs and M2 and M0 macrophages. Overall, we found that CIN CRC-derived IL-6 changes macrophage function and activity to support sustained growth of the tumor. These findings aid in further characterization of the differential immune response to CRC subtypes and the dynamics that may help or hinder therapeutic approaches.

LAY SUMMARY

- Interleukin-6 (IL-6) is a small protein that contributes to variable immune responses in patients with colorectal cancer based on its local concentration.
- Macrophages are a type of white blood cell involved in the immune response to cancer. They can be conditioned to act in tumor-promoting ways by IL-6.
- IL-6 regulates a pathway in a certain colorectal cancer subtype that promotes cancer cell survival.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Immunophenotyping of longitudinal peripheral blood mononuclear cells from patients with glioblastoma to distinguish pseudoprogression from true progression - Poster #825

Presenter: Lauren Westhaver, Dalhousie University

Other Authors: Kathleen Attwood, Jeremy Roy, Adrienne Weeks

ABSTRACT_ID#2708

ABSTRACT

Background Glioblastoma (high-grade glioma; GBM) is the most common and aggressive primary brain tumour, with a uniformly fatal prognosis. Despite aggressive treatment, the disease inevitably recurs. Following treatment, patients undergo repeat magnetic resonance imaging (MRI) to monitor for tumour progression. In a subset of patients, treatment- or immune-related effects can mimic tumour progression on MRI, showing an apparent increase in tumour size without actual growth – a phenomenon known as pseudoprogression. This creates diagnostic uncertainty, delaying treatment decisions or even prompting the premature discontinuation of effective therapy. There are no current imaging modalities that can definitively diagnose pseudoprogression. Therefore, there is an urgent need for minimally invasive monitoring techniques to reduce delays in care and enhance patient quality of life. **Methods** We designed and optimized two 28-parameter immune-profiling antibody panels to categorize immune states in patient blood, throughout the clinical journey. These high-parameter panels will allow for simultaneous assessment of phenotype and function of immune populations. Data are analyzed using dimensionality reduction and unsupervised clustering. **Results** We have established the largest longitudinal blood collection for brain tumour patients in Atlantic Canada. We have begun profiling the immune landscape of our GBM patient cohort using our multiparametric antibody panels. This resource enables immune system characterization, allowing us to track immune status in patients at any stage of disease. **Conclusion** Understanding changes in immune system profiles in patients undergoing treatment for GBM is crucial for improving diagnostic accuracy and reducing delays in care, thereby improving quality of life for patients with GBM.

LAY SUMMARY

- Pseudoprogression remains a major diagnostic challenge in glioblastoma, often leading to treatment delays or discontinuation of otherwise effective therapies, due to limitations in current imaging modalities.
- Longitudinal immune profiling of PBMCs using high-parameter flow cytometry may reveal distinct immunologic signatures associated with true progression versus pseudoprogression.
- This approach may support development of minimally invasive immune-based biomarkers to improve diagnostic accuracy and inform clinical decision-making.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Targeting the Senescent Microenvironment in Oral Carcinogenesis : Pathways to Prevention - Poster #826

Presenter: Jessica Bourbonnais, CRCHUM, Université de Montréal

Other Authors: Jade Montpetit, Guillaume Cardin, Alicia Pellerin-Viger, Francis Rodier, Apostolos Christopoulos

ABSTRACT_ID#3213

ABSTRACT

Oral cavity squamous cell carcinoma (OSCC) is the most common type of head and neck cancer. Oral carcinogenesis involves many steps of transformation within the epithelial cells themselves and the surrounding microenvironment. Although the genetic landscape surrounding the varying stages of transformed mucosal epithelial cells has been well documented, there is a lack of understanding when it comes to the role of the microenvironment. Specifically, the role of senescent cells in oral carcinogenesis and cancer progression has yet to be well defined. Senescence is a stable state of cell proliferation arrest that can be triggered by genotoxic stress. Senescent cells produce molecules that promote tumour cell proliferation, migration, and invasion, potentially accelerating cancer progression. High levels of p53 have been detected in the oral mucosa surrounding OSCC, suggesting the presence of senescent cells. My project aims to determine the role of senescence in oral carcinogenesis. Our co-cultures of H2B-GFP-labeled OSCC cell lines with senescent fibroblasts or epithelial cells show increased proliferation rates compared to our controls. We plan to assess the migration and invasion capabilities of co-cultures of OSCC and senescent cells, as well as to characterize cellular transformations that occur in OSCC cells following exposure to senescent cells. In addition, we plan to launch an unbiased CRISPR-Cas12 screen to identify the mechanisms of interaction between OSCC and senescent cells.

LAY SUMMARY

- We're studying how "senescent cells" — stressed or damaged cells that have stopped dividing — might actually help oral cancer grow and spread. These cells can release molecules that support tumor growth, and we've seen signs of them in tissue near oral cancers.
- When we grow oral cancer cells together with senescent cells in the lab, the cancer cells grow faster. This suggests that senescent cells may play an active role in helping tumors progress.
- Next, we'll test whether senescent cells also help cancer cells move and invade other tissues. We'll also use a CRISPR gene-editing screen to discover exactly how these two types of cells interact.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Impact of MAG-EPA supplementation on the prostate tumor microenvironment - Poster #827

Presenter: Lucie Leclair, Laval University

Other Authors: Gabriel Lachance, Oscar Molina, H       Hovington, Yves Fradet, Karine Robitaille, Vincent Fradet

ABSTRACT_ID#2747

ABSTRACT

Chronic inflammation is increasingly recognized as a significant factor in the development and progression of prostate cancer. In a randomized, controlled phase IIb clinical trial we evaluated the effect of supplementation with eicosapentaenoic acid (MAG-EPA), an anti-inflammatory omega (w)-3, in prostate cancer patients undergoing radical prostatectomy. A total of 130 patients received 3 g/day of MAG-EPA or a placebo for 6–8 weeks before surgery and were followed for one year. MAG-EPA supplementation led to a significant reduction in cancer aggressiveness between biopsy and surgery (termed downgrade) and a lower rate of biochemical recurrence. This study explores the impact of MAG-EPA on the prostate tumor microenvironment and its association with observed clinical outcomes. Fatty acids were profiled in prostate tissue collected during prostatectomy. Tissue microarrays were constructed from tumor and adjacent normal tissues. Immune cell populations were characterized using immunohistochemistry and multiplex immunofluorescence. Fatty acid profiling revealed increased levels of w3-EPA and -DPA, and decreased levels of w6-adrenic and -Osbond acids in the prostate of MAG-EPA supplemented patients. Spatial analyses revealed a greater number and larger lymphocyte aggregates in MAG-EPA-supplemented patients who experienced downgrade ($p=0.03$). These aggregates were enriched in FoxP3+CD8+ lymphocytes ($p<0.001$). These results suggest that MAG-EPA induces a favorable reorganization of immune cells within the microenvironment. Our findings support the potential of MAG-EPA as a complementary therapeutic approach that could delay or avoid invasive standard treatments affecting patients' quality of life. Further analyses of interactions between lipid metabolism and immune modulation will help uncover the antitumor mechanisms involved.

LAY SUMMARY

Omega-3 supplementation could reduce cancer aggressiveness: MAG-EPA, an anti-inflammatory omega-3, taken before prostate surgery was linked to a reduction in cancer aggressiveness between biopsy and surgery. Improved immune response: patients who took daily supplement of MAG-EPA had more and larger clusters of immune cells in their tumors, which can be associated with better cancer control by the immune system. Delay invasive treatments: this approach could help reduce the need for aggressive treatments, improving quality of life for prostate cancer patients.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Investigating the role of hypoxia-induced integrin expression on organotropic breast cancer metastasis - Poster #828

Presenter: Jeri Spilberg, University of Western Ontario

Other Authors: David Susman, Alison Allan

ABSTRACT_ID#2601

ABSTRACT

Breast cancer is the second leading cause of death in Canadian women, with metastasis accounting for >90% of breast cancer-related deaths. Common metastatic sites include the lung, liver, brain, and bone which is influenced by molecular traits of cancer cells and their microenvironments. In the current study, we hypothesize that breast cancer cells with different organotropic tendencies exhibit distinct patterns of hypoxia-induced integrin expression, leading to enhanced metastatic behavior. Preliminary proteomic analysis revealed that in response to hypoxia (1% O₂), 3 integrins (ITGα5, ITGαV, and ITGβ4) were increased in parental MDA-MB-231 TNBC cells, whereas 7 integrins (ITGα1, ITGα2, ITGα3, ITGα6, ITGβ1, ITGβ3, and ITGβ5) were increased in bone-seeking 231-BoM variant cells. Integrin profiles of parental MDA-MB-231 cells and their bone (231-BoM), lung (231-LM2), and brain (231-BR) metastatic variants are now being assessed under normoxic (20% O₂) and hypoxic (1% O₂) conditions using CHEMICON® Alpha/Beta Integrin-Mediated Cell Adhesion Arrays. To date, we have observed that parental 231 cells demonstrate upregulation of ITGα5, ITGα3, ITGβ1, and ITGβ4 under hypoxia. Notably, all integrins followed the hypoxia-induced upregulation patterns predicted by proteomic analysis. However, ITGα3 and ITGβ1 showed significant upregulation in array experiments, a finding not previously identified in the earlier analyses. Ongoing work is aimed at understanding hypoxia-mediated changes in organ-specific MDA-MB-231 variants. Investigating integrin expression across different metastatic organotropism under hypoxic conditions will deepen our understanding of how cancer cells adapt to hostile environments. This knowledge has the potential to identify novel biomarkers or biotherapeutics for preventing and treating breast cancer metastasis.

LAY SUMMARY

Hypoxia Alters Integrin Expression in an Organotropic-Specific Manner: Breast cancer cells with different organ-specific metastatic tendencies (bone, lung, brain) exhibit distinct integrin expression patterns in response to hypoxia, which may contribute to enhanced metastatic behavior. **Parental and Bone-Seeking Cells Respond Differently to Hypoxia:** Proteomic and array analyses revealed that hypoxia induces specific integrins in parental MDA-MB-231 and bone-seeking (231-BoM) cells, with ITGα3 and ITGβ1 newly identified as significantly upregulated in parental MDA-MB-231 cells. **Therapeutic Potential in Targeting Integrins:** Understanding hypoxia-driven integrin changes across metastatic variants may uncover novel biomarkers or therapeutic targets to prevent or treat breast cancer metastasis

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

The correlation between high-fat diet and physical activity during the progression of prostate cancer - Poster #829

Presenter: Mina Masoudnia, CRCHUM

Other Authors: Mina Masoudnia, Amine Lounis, Benjamin Peant, Murthy Madiraju, Fred Saad

ABSTRACT_ID#2360

ABSTRACT

Background: One of the strategies to arrest PC progression could be manipulating risk factors, including diet and physical activity (PA). We have previously shown that PA could slow the growth of LNCaP cancer cells in mice on a high-fat diet (HFD). However, the underlying mechanisms are yet to be discovered. Recently, the interaction of gut microbiota and prostate cancer cells in response to diet has been shown. However, the extent to which microbiota composition could affect PC progression and how PA, diet and ADT could interplay in the progression of PC remains unclear. We hypothesize that PA influences PC development and progression by enhancing therapeutic response and may counteract the adverse effects of HFD on PC tumor growth. **Methods and results:** Using NRG mouse models to follow the effect of HFD and PA on the metabolism of cancer cells via Metabolomics, we have shown PA could decrease the growth of PC cells in mice after castration and treatment with Enzalutamide. We are also currently running a pilot with the same setup to follow the growth of tumor and microbiota shift. **Conclusion:** Based on our preliminary findings, we expect that physical activity positively impacts prostate cancer progression and improves responses to ADT. We are currently investigating the modulation of gut microbiota composition in response to diet and PA and its potential contribution to the growth of PC. Investigating the mechanisms through which PA could execute its effect could potentially lead to the identification of new biomarkers and therapeutic regimes.

LAY SUMMARY

Prostate cancer, High fat diet, Physical activity

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Modeling Tumour Invasion and CAR-T Efficacy Using the Chick Chorioallantoic Membrane - Poster #830

Presenter: Ling Li, UBC, BCCHR

Other Authors: Tariq Bhat, Mahlegaha Ghavami, Juliana Vieira, Maryam Aletaha, Haniyeh Ghaffari-Nazari, Gregor S.D. Reid, Chinten James Lim

ABSTRACT_ID#2686

ABSTRACT

The chorioallantoic membrane (CAM) of fertilized chicken eggs is a highly vascularized membranous structure that supports embryonic development of amniotes. As a naturally immunodeficient model, CAM facilitates rapid xenografting of human tumours that can be implanted topically on the vascular bed, or injected intravenously for direct dissemination into the vascular network. We explore the utility of CAM as a fast and scalable alternative in vivo host for modeling studies of tumour cell invasion and for assessing the efficacy of chimeric antigen receptor (CAR) T-cell therapy. We used previously derived MDA-MB-231 cell lines exhibiting differential in vitro (Transwell) and in vivo orthotopic (murine) invasion ability for CAM model validation studies of tumor invasion. Following topical implantation onto intact CAM, xenografted wildtype tumours exhibited local invasion across the CAM epithelium at day 5, while integrin loss of function variants exhibited reduced local invasion. In sum, the CAM model recapitulated the differential invasion potential in an expedited fashion. The disialoganglioside GD2 is expressed and therapeutically targeted in neuroblastoma. We generated GD2-directed CAR-T cells and tested their anti-tumour effects against GD2-positive and GD2-null tumours. CAR-T cells were administered either intravenously or topically onto grafted tumors. The anti-tumor effect was assessed by comparing fluorescence and/or bioluminescent signals from the Luc-GFP tagged tumours, with CAR-T infiltration identified by histological examination. We conclude that the chick CAM model constitute a useful and economical alternative for pre-clinical research in oncology to accelerate in vivo tumour studies.

LAY SUMMARY

This study shows that the chick chorioallantoic membrane (CAM) model can be used as a fast and scalable alternative pre-clinical tool to model tumour cell invasion and to assess the efficacy of chimeric antigen receptor (CAR) T-cell therapy. The CAM model recapitulated the differential in vitro and in vivo (murine) invasion potentials. The CAM model can assess the anti-tumour efficacy of CART-cells in an expedited fashion.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Genomic instability regulates STING mediated anti-tumor immunity through distinctive cytosolic DNA structures - Poster #831

Presenter: Shayla Mosley, University of Alberta

Other Authors: Natalie Lapa, Thomas Catley, Courtney Mowat, Felix Meier-Stephenson, Vanessa Meier-Stephenson, Alice Pyne, Kristi Baker

ABSTRACT_ID#2916

ABSTRACT

Cancer genomes are characterized by instability, displaying significant levels of DNA damage and mutation due to DNA repair defects. In colorectal cancer (CRC), mismatch repair defects define the microsatellite instable (MSI) subtype, with stronger immunogenicity and better prognosis than the more common chromosomally instable (CIN) subtype. The mechanisms behind these differences remain unknown. The STING pathway detects foreign DNA in the cytosol, leading to induction of innate and adaptive immune responses. In cancer, STING also recognizes self cytosolic DNA (cyDNA) generated through endogenous or treatment-induced DNA damage. Our lab previously identified STING as a key player in the increased cytotoxic T-cell infiltration characteristic of MSI CRCs, however cyDNA quantity was not higher than in CIN CRCs. Therefore, we aim to characterize the cyDNA present in the CRC subtypes and understand how they differentially affect anti-tumor immunity. Using MC38 CRC cells with mutations modeling MSI or CIN, we found equal concentrations of MSI cyDNA more strongly activate STING, and thereby cytotoxic T-cells, than CIN cyDNA. Similar results are observed with cyDNA from CRC patient organoids. Sequencing identified patterns prevalent in strongly stimulatory cyDNAs, including microsatellites which enhance STING and cytotoxic T-cell activation. Additionally, we observe increased dendritic cell and cytotoxic T-cell activation in CIN tumors exposed to microsatellite DNA. Preliminary data suggests micronuclei and secondary-DNA structures may exert similar effects. This project will further our understanding of immune differences between MSI and CIN CRCs, allowing future development of therapeutic strategies, such as STING agonists, to improve anti-tumor immunity in CIN patients.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

3D Tumoroid Model for Studying Macrophage Polarization and Therapeutic Strategies - Poster #832

Presenter: Reza Javan, University of Saskatchewan

Other Authors: Michael Dean Chamberlain, Breanne Bevelander, Taylor Dzikowski, Parnaz Soori

ABSTRACT_ID#2434

ABSTRACT

Breast cancer affects millions of women worldwide, but treatment often fails due to the complexity of the tumour microenvironment (TME). Immune cells within the TME, particularly monocytes, play a crucial role in cancer progression and drug resistance. After recruitment, monocytes can differentiate into M2-like tumour-associated macrophages (TAMs), which support tumour growth and contribute to therapeutic failure. Traditional 2D culture systems fail to replicate the complexity of the TME, prompting the development of more physiologically relevant models. Our lab has established a novel 3D collagen-based microtissue model that better mimics the natural extracellular matrix and tumour structure. This model enables investigation of monocyte infiltration and polarization into M2-like macrophages. Recent data show that microtissues release higher levels of key cytokines involved in monocyte recruitment and TAM development, such as MCP-1, IL-10, IL-13, and VEGF, compared to 2D cultures. These findings highlight the model's potential to study immune-tumour interactions more accurately. Our research aims to understand monocyte-to-macrophage polarization and to explore macrophage repolarization strategies using dsRNA-loaded nanoparticles. By repolarizing macrophages within the microtissue model, we aim to counteract immune suppression and enhance anti-tumour immunity. This approach may offer a novel immunotherapeutic strategy to overcome drug resistance in breast cancer.

LAY SUMMARY

- We developed a 3D breast cancer model that better mimics real tumours by using a collagen-based structure, allowing us to study how immune cells interact with cancer in a more natural environment.
- Our model shows that breast tumours can attract immune cells called monocytes, which then change into tumour-supporting cells (macrophages) that help cancer grow and resist treatment.
- We are exploring ways to "reprogram" these immune cells using special nanoparticles, with the goal of turning them against the tumour and improving responses to treatment.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

D-serine facilitates aggressive migration and stemness of recurrent glioblastoma cells by interacting with host endothelial cells - Poster #833

Presenter: Ryan Mota, University of Manitoba

Other Authors: Ping Lu, Emma Martell, Alejandra Llanes Cuesta, Lisa Liang, Sheila K. Singh, Tanveer Sharif, Christopher M. Anderson

ABSTRACT_ID#2919

ABSTRACT

Introduction: Glioblastoma (GBM) cells infiltrate deep brain structures by exploiting perivascular pathways, utilizing stem-like properties and malignant traits to access nutrient-rich environments that support tumor expansion. Our findings suggest that N-methyl-D-aspartate receptors (NMDARs) in brain Endothelial Cells (ECs) play a key role in transducing signals initiated by parenchymal cells, facilitating recurrent GBM progression. Hypothesis: D-serine enhances GBM migration and stemness by interacting with host endothelial cells, thereby promoting recurrent tumor aggressiveness. Methods: Patient-derived recurrent GBM cells and human cerebral microvascular ECs were co-cultured in a transwell system to assess the role of D-serine in GBM migration and stemness. Pharmacological inhibitors, CRISPR/Cas9-mediated gene silencing, and enzymatic modulation of D-serine were employed to determine the contribution of GBM-derived D-serine and endothelial NMDARs (eNMDARs) to tumorigenesis. Xenograft experiments employed gene silencing or pharmacological inhibitors of Serine Racemase (SRR) to test the effects of recurrent tumorigenesis. Results: In silico analysis of patient datasets showed increased SRR transcripts in recurrent GBM tissue which led to a poorer patient outcome. In vitro, ECs potentiate GBM migration and stemness in co-culture. A low D-serine microenvironment was established and led to a significant reduction in GBM migration and stemness markers. NMDAR antagonism and eNMDAR silencing further mitigated these effects. In vivo, SRR silencing, or inhibition, reduced tumor burden at 4 weeks post-injection and extended survival in mice. Conclusion: Our findings demonstrate that brain ECs enhance GBM malignancy through D-serine-mediated activation of eNMDARs. This pathway supports GBM migration and stemness, presenting a novel therapeutic target for recurrent GBM treatment.

LAY SUMMARY

Brain blood vessel cells help drive glioblastoma (GBM) spread. We found that endothelial cells (which line blood vessels in the brain) play an active role in helping GBM cells migrate and grow by a D-serine-mediated signalling loop. D-serine boosts tumor stem-like behavior by interacting with endothelial cells. D-serine, produced by GBM cells, makes the tumors more aggressive by enhancing their ability to migrate and act like stem cells through the release of pro-tumor molecules from endothelial cells. These aggressive properties are known to cause the cancerous cells to recur and be resistant to therapy. Blocking D-serine or its targets slows tumor growth. When we reduced D-serine levels or blocked its receptor (NMDAR) in endothelial cells using drugs or gene editing, GBM cells became less invasive in lab and animal models. These approaches also reduced tumor size and improved survival in mice.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Investigating metabolic dependencies during the evolution of glioblastoma - Poster #834

Presenter: Maryam Al-Witry, University of Calgary

Other Authors: Robert McTaggart Cowan, Camille Rozon, HyeIn Jang, Emilie Niu, Karen Blote, Martha Hughes, Shrivani Pirahas, John Kelly

ABSTRACT_ID#2866

ABSTRACT

Glioblastoma (GBM) is the most common and aggressive form of brain cancer in adults, with a five-year survival rate of less than 7%. GBMs are difficult to treat and often recur within a year of diagnosis. Despite many attempts, treatment options have remained stagnant over decades. Clinical trials have yet to reveal an effective therapy for GBMs. This stems, in part, from an incomplete understanding of tumour biology, and lack of appropriate preclinical models to capture patient specific tumour heterogeneity. There is an urgent need for novel approaches to identify new therapies. Patient-derived organoids (PDOs) have emerged as important models, maintaining the phenotypic and genetic features of the parent tumours. PDOs are used in precision medicine to explore new therapeutic applications. Altered metabolism is a hallmark of GBM and is necessary for the growth and survival of GBMs. Here, we aim to develop and characterize matched newly diagnosed and recurrent GBM (nd-rGBM) organoids to identify phenotypic, growth and metabolic properties of GBMs. Our data indicates that nd-rGBM organoids displayed similar features such as cells expressing cycling (Ki67), stem- or progenitor-like (NES, SOX2, OLIG2) and glial (GFAP) markers compared to their parent tumours. Notably, we observed distinct growth between matched nd-rGBM pairs, with slow-growing GBMs exhibiting increased oxidative phosphorylation (OxPhos). This is important because different metabolic subtypes of GBMs are linked to varied prognoses. We therefore anticipate our work may lead to identification of new insights into the tumour evolution and can be further exploited to identify new therapeutic strategies.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Understanding the Interaction Between Macrophages and Tumor Cells in the Brain Tumor Microenvironment to Enhance Anti-Tumor Immune Responses - Poster #835

Presenter: Mallak Salla, University of Alberta, Cross Cancer Institute

ABSTRACT_ID#2521

ABSTRACT

Glioblastoma (GBM) is an aggressive primary brain tumor characterized by profound immune suppression within the tumor microenvironment. Among immune cells, macrophages are the most abundant in the GBM microenvironment and are generally expected to possess intrinsic anti-tumor functions. However, these functions are often suppressed by tumor cells. The extensive intratumoral heterogeneity of both macrophages and GBM cells—at single-cell and spatial levels—makes understanding the mechanisms driving their communication a significant challenge. To address this limitation, we developed a novel platform that integrates single-cell RNA sequencing, spatial transcriptomics, and spatial CRISPR screening to investigate macrophage–GBM interactions while preserving the single-cell spatial heterogeneity of the GBM microenvironment. Using published single-cell and spatial transcriptomics datasets, we first identified distinct subsets of macrophages and GBM cells, as well as their anatomical locations. We then analyzed interactions between different macrophage subsets—including monocyte-derived macrophages—and GBM cells, identifying key signaling networks that mediate their communication. Building on these observations, we designed a spatial CRISPR library targeting 50 genes that are highly expressed in tumor cells and have known or potential druggable properties. We aim to use this CRISPR screen to identify genes whose inhibition can reprogram macrophages toward an anti-tumor phenotype and suppress tumor growth in a rodent GBM model.

LAY SUMMARY

Glioblastoma (GBM) suppresses the immune system: We found that glioblastoma, an aggressive brain cancer, creates a complex tumor microenvironment that weakens the body's natural immune response. GBM has developed ways to suppress the anti-tumor functions of the immune cell macrophages. Advanced technology reveals how immune and tumor cells communicate: By combining single-cell RNA sequencing, spatial transcriptomics, and a new CRISPR screening method, we were able to map where different tumor and immune cells are located within the microenvironment, and how they talk to each other to suppress immune responses. Targeting tumor genes to reprogram immune cells: Through the advanced technology techniques utilized in this research, we were able to identify tumor genes that seem to influence immune suppression. We are now testing whether blocking these genes can reprogram macrophages activating them to fight the tumor cells. This discovery could lead to more effective treatments for GBM.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Evaluating novel therapeutic approaches to inhibit the Androgen Receptor (AR) and AKT pathways in metastatic Castration-Resistant Prostate Cancer (mCRPC) - Poster #836

Presenter: Mehdi Haghi, McGill University

Other Authors: Jennifer Maxwell, Sadaf Solati, Soumaya Labidi, April Rose

ABSTRACT_ID#3090

ABSTRACT

Background: Androgen receptor pathway inhibitors (ARPIs) (e.g., enzalutamide) combined with androgen deprivation therapy (ADT) improve survival in metastatic prostate cancer. However, AR alterations (amplification, mutations, splice variants) drive resistance to ARPIs, giving rise to mCRPC. Next-generation ARPIs (ngARPIs) aim to target these alterations. We found AR alterations frequently co-exist with AKT pathway alterations in mCRPC. Therefore, we interrogated the efficacy of combining ngARPIs with AKT inhibitors in preclinical mCRPC models. Methods: Functional genomics (CRISPR Competition) assays assessed AR gene essentiality. ARPI-resistant cells were generated by growing 22Rv1 and LnCAP cells in enzalutamide for several months. We investigated the effects of ARPIs (enzalutamide) and ngARPIs (masofaniten, ARV-110) on CRPC cell lines (22Rv1, C4-2B) alone and with the AKT inhibitor, capivasertib. Immunoblotting assessed pathway activation, and Bliss synergy assays evaluated combinatorial effects on cell viability. Results: AR gene knockout significantly reduced 22Rv1 cell viability. ARV-110 (10–1000 nM) induced dose-dependent AR degradation and decreased PSA/KLK3 levels in 22Rv1 and C4-2B. Increasing masofaniten doses (10–40 μ M) also reduced PSA/KLK3. At the highest ngARPI doses, cleaved PARP levels increased, indicating enhanced apoptosis, while pAKT levels increased, suggesting activation of compensatory survival signaling. Combining capivasertib with masofaniten and ARV-110 further reduced cell viability and gave Bliss Synergy Scores of 4 and –1.6, respectively, indicating additive benefit. Conclusion: AR signaling is essential in CRPC cells. Dual AR and AKT targeting may represent a promising strategy to overcome ARPI resistance. Ongoing studies will assess AR gene essentiality and drug synergy in ARPI-resistant CRPC models, informing future clinical strategies.

LAY SUMMARY

Prostate cancer can become resistant to common treatments when the androgen receptor (AR) signaling pathway is altered, creating challenges for patient care. Our laboratory tests show that combining AR-targeting drugs with AKT pathway blockers can more effectively reduce cancer cell survival and growth compared to using AR-targeting drugs alone. This dual-targeting approach may offer a new strategy for treating patients whose prostate cancer has become resistant to current therapies.

Evaluating the potential interplay between senescence induction by chemotherapy and immune modulation

- Poster #837

Presenter: Mylène Laflamme, CR-HMR

Other Authors: Paul Minaya Ferruzo, Romain Villot, Elpida Bokou, Audrey L. Collin, Karine Boulay, Léa Bourguignon, Frédérick-Antoine Mallette, Julianna Blagih

ABSTRACT_ID#3150

ABSTRACT

The compromised immune system of cancer patients undergoing chemotherapy, as well as cancer survivors, is widely recognised. However, the mechanisms by which chemotherapy affects long-term immune suppression remain poorly understood, limiting our capacity to preserve it during treatment. While chemotherapy effectively eradicates cancer cells, it can also induce cellular senescence, in both cancerous and normal cells, a phenotype in which cells cease to grow and exhibit a senescence-associated secretory phenotype (SASP). The SASP may have immune-altering properties. Therefore, we hypothesise that the SASP can partly mediate chemo-induced chronic immune modulation. To test this, we challenged C57BL/6 mice with a single dose of doxorubicin – a frontline therapy for multiple cancers – and immunophenotyped various immune compartments (thymus, lymph nodes, spleen, bone marrow, adipose tissue, and liver) along with plasma inflammatory markers on days 7, 14, 21 and 28 following chemotherapeutic treatment via flow cytometry and ELISA. Interestingly, we observed a significant reduction in CD8 and CD4 T cell frequencies even at later time points in various immune compartments, alongside a weakened proliferative capacity when tested *ex vivo*. Moreover, while we noted lower circulating SASP levels, we detected localised SASP induction within tissues. In line with this, conditioned media from senescent fibroblasts significantly reduced the proliferation and differentiation of CD8 T cells in cell culture. These data suggest an opportunity to reduce SASP production in response to chemotherapy, potentially alleviating its immunosuppressive effects.

LAY SUMMARY

- **Chemotherapy:** While chemotherapy is effective at eliminating cancer cells, it is also known to weaken the immune system in both patients undergoing treatment and survivors. In this study, we used doxorubicin, a common chemotherapeutic agent, to assess its impact on the immune system in mice.
- **Cellular senescence:** Chemotherapy can induce cellular senescence, a state in which cells stop dividing and adopt a senescence-associated secretory phenotype (SASP). Although circulating SASP levels were low, localized SASP expression was detected in tissues, suggesting it may play a role in tissues locally and create persistent side effects.
- **Immune modulation:** Doxorubicin treatment led a sustained reduction in CD4+ and CD8+ T cell frequencies across multiple immune compartments (thymus, spleen, bone marrow, etc.), as well as reduced proliferative capacity *ex vivo*. In vitro, conditioned media from senescent fibroblasts significantly impaired CD8+ T cell proliferation and differentiation, indicating that SASP factors can directly suppress immune responses.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Targeting Lysosomes in the Chronic Lymphocytic Leukemia (CLL) Microenvironment - Poster #838

Presenter: Madhumita Manivannan, University of Alberta

Other Authors: Nirav Patel, Spencer Gibson

ABSTRACT_ID#2355

ABSTRACT

Chronic lymphocytic leukemia (CLL) cells exploit lysosomal pathways and the tumor microenvironment to evade apoptosis, contributing to therapeutic resistance. Lysosomes support cancer cell survival by regulating autophagy and preventing activation of cell death pathways, thereby promoting resistance to therapy. Pimozide, an FDA-approved antipsychotic used for Tourette's syndrome, is a lysosomotropic agent that disrupts lysosomal integrity and presents a promising strategy to overcome drug resistance in CLL. In this study, we investigated the effects of pimozide alone and in combination with venetoclax, a BCL-2 inhibitor, on primary CLL cells. Pimozide monotherapy induced significant lysosomal membrane permeabilization (LMP), release of lysosomal proteases and inhibits autophagy, a cell survival mechanism. This was accompanied by elevated reactive oxygen species (ROS) levels, particularly mitochondrial ROS, as detected using DHE and MitoSox staining. When combined with venetoclax, pimozide further increased ROS production, leading to synergistic apoptotic cell death. This combination therapy effectively overcame the protective effects of the microenvironment, including IL-4/CD40L stimulation and co-culture with HS-5 stromal cells. Importantly, T cells were not affected, supporting the selective cytotoxicity of the treatment. Pimozide was also effective in samples from relapsed CLL patients. These results demonstrate that pimozide synergizes with venetoclax to target both lysosomal and mitochondrial pathways, disrupting survival signals that protect CLL cells. Given its established safety profile as an FDA-approved drug, pimozide holds strong potential for repurposing in CLL therapy. Future studies will utilize 3D bone marrow spheroid models to assess the impact of this combination on microenvironmental resistance and support clinical translation.

LAY SUMMARY

We're repurposing a common drug to fight leukemia: A medication called pimozide, usually used to treat Tourette's syndrome, is showing promise in killing cancer cells in chronic lymphocytic leukemia (CLL). It works by damaging parts of the cancer cell called lysosomes, tiny "recycling centers" that help the cell survive under stress. It works even better when paired with existing treatment: When we combined pimozide with venetoclax (a drug already used for CLL), they worked together to kill cancer cells more effectively, even when those cells were in a protective environment that usually helps them resist treatment. This combination also worked in relapsed patients, whose disease had returned after earlier treatment. It's tough on cancer, gentle on healthy cells: Encouragingly, the treatment mostly left healthy immune cells alone, suggesting it could be a safer and more targeted therapy for people with CLL.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Spatially resolved proteomics to investigate immune cell-tumor cell interactions in breast ductal carcinoma in-situ upgrade - Poster #839

Presenter: Puneet Arora, University of British Columbia

Other Authors: Sylvia Lam, Zhao Yang Chen, Anthony Speranza, Alan Nichol, Zuzana Kos, Peter Watson, Calum MacAulay, Martial Guillaud

ABSTRACT_ID#3118

ABSTRACT

Ductal carcinoma in-situ (DCIS) is a pre-invasive form of breast cancer that is characterized by limited proliferation of epithelial cells lining the breast ducts. In Canada, the current standard of care is to perform partial mastectomy because there is a risk of 25-60% DCIS cases to progress to invasive ductal carcinoma (IDC). DCIS subtypes exhibit distinct tumour microenvironment (TME) and proteomic profiles and are associated with varying clinical outcomes. The quantity, and spatial distribution of lymphoid aggregates of tumour-infiltrating lymphocytes (TILs) can predict upgrade to IDC. For instance, the immune cells near tumour cells may not be "activated" to kill the cancer, however, with current technology, this remains unknown. We hypothesized that cell-cell interactions and the spatial organization of the TME would enable us to identify distinct immune signatures that would predict upgrade of low-grade DCIS to IDC. Our approach utilizes spatial proteomics (SP) and cell sociology analysis to visualize protein interactions and immune cell activity across different breast regions. We aim to perform SP analysis using 50 samples from patients with DCIS (n = 25) and DCIS adjacent to IDC (n = 25) using the Lunaphore COMET platform to target immune cells and tumour cells. Data analysis will be performed using QuPath and an in-house cell sociology platform to quantify the TME. We anticipate that SP will distinguish between the TME of DCIS lesions that upgrade to IDC and those that remain indolent. This study has the potential to improve DCIS risk stratification and guide personalized treatment options.

LAY SUMMARY

We aim to analyze the spatial arrangement of immune cells in pure DCIS and in DCIS cases with nearby invasive ductal carcinoma (IDC) to understand whether differences in immune cell location and interaction with tumour cells relate to risk of upgrade. It is possible that in cases where DCIS upgrades to IDC, the immune cells are not properly "activated" to eliminate abnormal cells. By comparing the presence or absence of active immune cells between the two groups, we aim to identify immune-related markers that could help predict which cases are more likely to upgrade from DCIS to IDC. The results of this study will be used to develop more precise diagnostic tools, reducing unnecessary surgeries while ensuring high-risk patients receive the care they need.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

State-aware spatial phenotyping of primary and recurrent glioblastoma - Poster #840

Presenter: Ana Nikolic, University of Calgary

Other Authors: Amy Banks, Jamie McNicol, Shalina Ousman, Michael van Vliet, Andrew Chojnacki, Margaret Kelly, Jennifer Chan

ABSTRACT_ID#3102

ABSTRACT

Glioblastoma (GBM) is the most common malignant adult brain tumour, and its aggressive behaviour and limited treatment options make it an important clinical challenge. These tumours have a host of microenvironmental interactions, including ones with macrophages and non-tumour neurons and glia. Interactions between malignant cells and their surrounding microenvironment contribute to treatment resistance and poor patient outcomes, with median survival of under two years despite aggressive multimodal therapy. Recent studies have shown that glioblastoma tumour cells can exist in multiple distinct cell states, with unique properties and markers that influence their response to therapy. Cellular plasticity allows GBM tumours to adapt to treatment pressures and environmental changes. It remains unclear how these tumours change at recurrence and how different parts of the tumour, such as the leading edge, may evolve spatially and temporally throughout disease progression. We use an emerging multiplex imaging technology (Multiplex Ion Beam Imaging – MIBI) to map GBM cell states and their microenvironmental interactions in multiple tumour regions in a cohort of pathology specimens from over 100 primary and recurrent tumours. This high-resolution spatial profiling approach enables simultaneous detection of multiple protein markers while preserving tissue architecture. We characterize these phenotypes and neighbourhoods in conjunction with matched H&E sections, providing new insights into relationships between conventional histology, cell state, and microenvironment evolution in this disease.

LAY SUMMARY

- We were able to use new technologies to better subtype single GBM brain tumour cells in tissue.
- We can see how GBM cells are arranged throughout different areas of the tumor and how they change over time.
- This work can help identify new markers to help guide treatment decisions for GBM patients.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Extracellular Vesicles as Mediators of Tumor–Microenvironment Crosstalk in Glioblastoma - Poster #841

Presenter: Peipei Zeng, McGill University

Other Authors: Shyam Menon, Isabelle Carrier, Tala-Maria Mouannes, Stephen Robbins, Donna Senger

ABSTRACT_ID#3120

ABSTRACT

Gliomas affect approximately 2800 Canadians yearly, the majority being aggressive, high-grade glioblastomas (GBM) with dismal prognosis and limited treatment options. One major contributor to GBM progression is its ability to reshape the tumor microenvironment (TME), particularly through interactions with immune cells. Previously, we found that interleukin 33 (IL33) promotes tumor growth and increases macrophage infiltration when expressed by glioma cells, creating a pro-tumorigenic microenvironment. Currently, the mechanism of IL33 secretion into the extracellular milieu where it is believed to interact with cells within the environment remains unclear due to the absence of a conventional signal peptide. Several studies highlight intracellular communication within the GBM TME via extracellular vesicles (EVs), and recently squamous cell carcinoma has been shown to release EVs carrying IL33 that promote immunosuppressive differentiation of macrophage precursors. Herein we investigate the presence and potential function of IL33 in EVs isolated from patient-derived GBM stem-like cells. EVs were collected using ultracentrifugation and size exclusion chromatography, and characterized by nanoparticle tracking analysis, Western blot analysis, and transmission electron microscopy where the presence of IL33 was confirmed in the EV fraction. Ongoing experiments will delineate the role of secreted IL33 and its functional impact on the innate immune repertoire within GBM. Our findings aim to provide insight into the underlying mechanism of IL33 secretion and its role in immune modulation and treatment resistance in GBM.

LAY SUMMARY

Glioblastoma is an aggressive brain cancer with very poor outcomes, in part because it hijack nearby immune cells called macrophage and microglia to support tumor growth rather than fight it. A protein called Interleukin 33 (IL33) has been linked to this immune system reprogramming by engaging these cells to support their growth. How IL33 is released from the tumor cell where it influences the surrounding environment is unclear. Our research shows that tumor cells can package IL33 into extracellular vesicles, which are tiny particles that carry messages between cells. We are now investigating how these IL33-loaded particles affect the immune cells within the tumor and what messages they carry that can shift the actions of the immune cells to fuel tumor growth. Understanding this will help develop future diagnostic tools or treatment strategies that short circuit these drivers of tumor growth.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

T Cell Clonal Dynamics in Breast Cancer - Poster #842

Presenter: Rachel Catterall, McGill University

Other Authors: Élise Di Lena, Luke McCaffrey, Heather Melichar

ABSTRACT_ID#2419

ABSTRACT

In Canada, breast cancer is the most diagnosed cancer in women, and despite treatment advancements, remains a leading cause of cancer related death. Immunotherapies, which help patients' immune systems recognize and fight cancer cells, are generally underexplored in early breast cancers since the role for the immune system in controlling early disease has been unclear. Recently, our lab identified regions of breast cancer tissue enriched with an important immune cell type called T cells. The T cells are replicating and express markers that suggest the immune hotspots may help actively fight tumour growth. Interestingly, these immune hotspots are often associated with an early, preinvasive stage of breast cancer. In this project, we are studying patient samples and mouse models to understand the diversity of these immune "hotspots", how they form, and their role during disease progression. Through analysis of patient data in The Cancer Genome Atlas, we found immune hotspots across all breast cancers, including 86% of hormone receptor positive breast cancers, previously thought to have little immune involvement. Additionally, we identified hotspots in 84% of patients with preinvasive lesions. To explore hotspot formation and impact on disease progression, we developed a mouse model that uses a breast cancer cell line to form hormone receptor positive preinvasive lesions and T cells that specifically target an antigen presented by these cancer cells, allowing us to manipulate the T cell response strength and timing. Identifying new immune landscapes may enable more informed decision-making for immunotherapies and potentially new treatment options for patients.

LAY SUMMARY

- We demonstrate that preinvasive, hormone receptor positive breast cancers may be more immune active than previously appreciated.
- We identified immune hotspots, primarily made up of antitumour T cells, which may have a role in shaping the development of invasive breast cancer.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Deciphering the role of ILC2-mediated immune defence in breast cancer - Poster #843

Presenter: Qiutong Huang, University of Calgary

Other Authors: Hobin Seo, Megan Kinzel, Amrita Kaur, Alisha Elford, Pamela Ohashi, Nicolas Jacquelot

ABSTRACT_ID#2748

ABSTRACT

Innate lymphoid cells (ILCs) are immune cells that respond rapidly to microenvironmental signals to initiate immune responses. We and others found that type 2 ILCs (ILC2s) drive type 2 immune response and influence anti-tumour immunity and cancer prognosis. While ILC2s have demonstrated anti-tumour roles in various cancers, in part through the recruitment of eosinophils to mediate anti-tumour immunity, their role in breast cancer remains unclear. To investigate ILC2 infiltration within the breast tumour microenvironment, we used PyMT mice, which spontaneously develop mammary tumours and observed increased infiltration of ILC2s and eosinophils in breast tumours of PyMT mice compared to healthy mammary fat pad. Interestingly, as breast tumours progressed, the frequency of ILC2 and eosinophil infiltration within the breast tumour decreased. Additionally, we found reduced tumour infiltration of KLRG1+ inflammatory ILC2, increased PD-1 expression, and decreased IL-5 and IL-13 production. To examine the changes in breast tumour development in the absence of ILC2s compared to ILC2-sufficient controls, we crossed ILC2 deficient mice with PyMT mice and found that mice that lacked ILC2 developed palpable breast tumours earlier than ILC2-sufficient mice. These findings suggest that ILC2 dysregulation during breast tumour development may alter eosinophil recruitment within the tumour microenvironment. The role of ILC2s in modulating this environment appears essential for tumour protection, as ILC2 deficiency is associated with earlier tumour onset. Building on these findings, we plan to elucidate the ILC2-mediated protection mechanisms using a multi-omic approach to identify key signalling pathways, cytokine production, and receptors involved in ILC2-driven immune defence.

LAY SUMMARY

We found white blood cells with anti-tumour functions in breast tumours. Tumour cells hijack the function of white blood cells, making them less able to fight cancer. Without white blood cells tumours develop faster than normal, suggesting that white blood cells are required to protect against tumour growth.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Real-Time Imaging and Humanized Models Illuminate gamma delta T Cell Dynamics, Sphingolipid Metabolism, and AML Immunogenicity - Poster #844

Presenter: Raheleh Niavarani, University Health Network

Other Authors: Xi Lei, Sadaf Mazhab Jafari, Lyndsey De Guzman, Shirley Wang, Ralph DaCosta

ABSTRACT_ID#3355

ABSTRACT

Acute myeloid leukemia (AML) remains a clinically challenging malignancy, in part due to the emergence of chemoresistance. To better understand the immunological dynamics underlying AML progression and treatment resistance, we employ intravital microscopy in conjunction with humanized mouse models, allowing real-time visualization of human immune cell behavior within the leukemic bone marrow microenvironment. Our study focuses on the role of gamma delta (gd) T cells, a unique subset of lymphocytes with emerging relevance in cancer immunotherapy. We reveal that $\gamma\delta$ T cells engage in dynamic interactions with AML, exhibiting migratory patterns and cytotoxic activity. Notably, we examine the role of gd T cell function in relation to chemoresistance in AML. Additionally, we show that modulation of sphingolipid ceramide levels in different biological conditions reshapes the immunogenic landscape of AML cells. These findings underscore the importance of gd T cells in the AML microenvironment and suggest new avenues for targeting immune-metabolic pathways to overcome resistance and enhance immunotherapy efficacy.

LAY SUMMARY

gd T cell cytotoxicity and the resulting AML tumor cell death have been visualized for the first time in a humanized mouse model of AML. The bone marrow niche regulates the metabolism of gd T cells and tumor cells, impacting the effector function of gd T cells.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

A senescence-like state is beneficial for ovarian cancer treatment - Poster #845

Presenter: Francis Rodier, Université de Montréal

ABSTRACT_ID#3304

ABSTRACT

High-grade serous ovarian carcinoma (HGS) commonly responds to initial therapy, but this response is rarely durable. Understanding cell fate decisions taken by HGS cells and their supporting microenvironment during chemotherapy in patient could guide new therapeutic opportunities. Here we find that tissue-derived primary HGS epithelial cultures and patient-derived xenografts reflecting the original disease undergo therapy-induced senescence in response to DNA damage and first-line carboplatin/paclitaxel chemotherapy. Unlike observations obtained using cell lines, primary HGSOC cell displays a stable senescence proliferation arrest and increased p16INK4A expression, which is accompanied by persistent DNA damage, an inflammatory secretome, and senolytic sensitivity, overall suggesting new senotherapeutic avenues for the selective pharmacological manipulation of these cells. In general, whether cell senescence induced by cancer therapy is beneficial or detrimental to clinical outcomes remains unknown. Spatial single cell comparison of pre- and post-chemotherapy patient HGS tissue samples revealed changes in physio-pathological senescence biomarkers supporting a post-treatment senescence-like state. Importantly, patients with stronger senescence signatures post-chemotherapy displayed better 5-year clinical outcomes suggesting that senescence accounts, at least in part, for early beneficial cellular responses to treatment. Given that ovarian cancer HGS cells retain the capacity to undergo senescence and that senescence appears beneficial in this context, HGS senescence-centric senotherapeutic that manipulate the senescent microenvironment could be further explored.

LAY SUMMARY

Cell senescence, or "cell aging", is a feature that protect against cancer in normal cells that suffer oncogenic insults. Surprisingly, we demonstrate that very aggressive ovarian cancer cells retain the ability to activate senescence How cancer cells respond to treatment in tissues in real time in solid cancer remain mostly unexplored. Because of this whether cancer therapy-induced cell fate decisions like senescence or apoptosis are good or bad for treatment outcome is unknown. We find that ovarian cancer cells almost universally retain senescence competence including in patient tissues and that senescence in treated tissues correlate with good clinical outcomes. Our study reveals that senescence is a relevant drug target in ovarian cancer.

Inhibition of CCL2 Promotes Anti-tumor Immunity and Enhances Radiotherapy Response in Head and Neck Squamous Cell Carcinoma - Poster #847

Presenter: Cleopatra Rutihinda, Université de Sherbrooke

Other Authors: Aliya Shams, Ryma Haroun, Léa Bouchard, Ayman Oweida

ABSTRACT_ID#2508

ABSTRACT

Radioresistance remains a major challenge to effective treatment in head and neck squamous cell carcinoma (HNSCC) with 30% of patients developing locoregional recurrence. While radiotherapy (RT) elicits tumor cell death and immune activation, it also induces immunosuppressive pathways in the tumor microenvironment (TME), limiting anti-tumor responses. In this study, we investigated the role of chemokine CCL2 in mediating RT-induced recruitment of immunosuppressive M2-like tumor-associated macrophages (TAMs) in HNSCC. We hypothesized that RT induces CCL2 secretion leading to M2-like TAM polarization and infiltration, blunting RT efficacy. Human and murine HNSCC cell lines were irradiated at 10Gy. CCL2 expression was assessed via qPCR, ELISA and Western blot. Transwell migration assays evaluated macrophage migration. In vivo, orthotopic MOC2 tumors were treated with RT (2x10Gy) with or without CCL2 neutralization. Flow cytometry and immunofluorescence characterized tumor-infiltrating immune cells. RT significantly increased CCL2 mRNA and protein levels in both human and mouse HNSCC cell lines. Increased CCL2 promoted M2-like macrophage migration, an effect reversed by CCL2 neutralization in transwell migration assays. In vivo, RT promoted M2-like TAM infiltration and the combination of RT with CCL2 blockade significantly decreased tumor growth compared to either treatment alone. These findings identify RT-induced CCL2 as a key driver of M2-like TAM recruitment in HNSCC. Targeting the CCL2-CCR2 axis offers a promising strategy to enhance anti-tumor immunity and overcome radioresistance in HNSCC patients. Future work will investigate CCL2 blockade in combination with immune checkpoint inhibitors and assess its clinical relevance in patient samples.

LAY SUMMARY

Radiation therapy in head and neck cancer induces the production of a molecule called CCL2. This molecule/chemokine attracts immune-suppressing cells (M2-like TAMs) into the tumor. These M2-like TAMs can protect the tumor from the immune system and contribute to radiation resistance. Blocking CCL2 in combination with radiation prevents radiation-induced M2-like TAM recruitment into tumors and significantly reduces tumor growth in mice, offering a promising new approach to improve treatment outcomes.

Genotype-Dependent Variability in Radiation-Induced Immunogenic Cell Death in NSCLC Cell Lines - Poster #848

Presenter: Ryma Haroun, Université de Sherbrooke

Other Authors: Cleopatra Rutihinda, Léa Bouchard, Ayman Oweida

ABSTRACT_ID#2713

ABSTRACT

Background: Radiotherapy remains a cornerstone in the treatment of non-small cell lung cancer (NSCLC), yet its efficacy is often limited by intrinsic or acquired radioresistance. Recent evidence suggests that the capacity of tumor cells to undergo immunogenic cell death (ICD) following radiation may influence treatment outcomes by shaping the antitumor immune response. Here, we investigate how distinct oncogenic mutations affect radiosensitivity and the induction of ICD hallmarks in NSCLC. **Methods:** We used murine NSCLC cell lines with defined oncogenic alterations: LLC46 and CMT54-10 (KRAS G12C), EA1 and EA2 (EML4/ALK), and mEGFR del19.1 and del19.2 (EGFR del19). Cells were irradiated with increasing doses (0, 2, 6, 10 Gy), and clonogenic assays assessed radiosensitivity. ICD was evaluated by measuring calreticulin (CALR) surface exposure via flow cytometry at 12, 24, and 48 hours post-irradiation. Conditioned media collected at these time points will be analyzed for ATP release (luminescence assay) and HMGB1 secretion (ELISA). **Results:** Clonogenic assays revealed mutation-dependent radiosensitivity. LLC46 and CMT54-10 were more radiosensitive than EA1, EA2, and both mEGFR lines. CALR exposure was dose- and time-dependent in LLC46, peaking at 10 Gy after 48 hours. CMT54-10 showed moderate CALR induction. In contrast, EA1, EA2, and mEGFR lines showed minimal CALR exposure. Conditioned media collected post-irradiation will be analyzed to determine the extent of ATP and HMGB1 release. **Conclusion:** These data suggest that the genetic background of NSCLC influences their response to radiation and their capacity to undergo ICD. Ongoing analyses will clarify whether these differences correlate with functional immune activation, potentially guiding genotype-tailored radiosensitization strategies or immunoradiotherapy approaches.

LAY SUMMARY

Radiation therapy is commonly used to treat non-small cell lung cancer (NSCLC), but some tumors resist treatment. We are studying how genetic differences in cancer cells affect their sensitivity to radiation. Some tumor cells die in a way that activates the immune system, this is called immunogenic cell death (ICD). We found that NSCLC cells with certain mutations (like KRAS G12C) are more likely to trigger ICD after radiation compared to others (like EGFR del19 or EML4/ALK fusions). We measured key signs of ICD, such as the appearance of a protein called calreticulin on the surface of cells, which acts like a distress signal to immune cells. Cells with KRAS G12C mutations showed more of these signals after radiation, suggesting they might better stimulate the immune system. Our goal is to better understand how the genetic makeup of tumors influences both their response to treatment and their ability to activate immune defenses. This could help tailor therapies that combine radiation and immunotherapy, depending on each patient's tumor profile.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Reprogramming Tumour Associated Macrophages for Tumour Eradication - Poster #849

Presenter: Sandra Hwang, McMaster University

Other Authors: Eden Kapcan, Anjalee Wijetunge, Anthony Rullo

ABSTRACT_ID#3122

ABSTRACT

Immunotherapy leverages the host immune system to combat tumour progression. However, its efficacy is often limited by the immunosuppressive tumour microenvironment (TME). A major contributor to this suppressive environment is tumour-associated macrophages (TAMs), which promote angiogenesis, metastasis, and immune evasion. Despite the pro-tumour phenotype of TAMs, its plasticity opens the possibility for reprogramming into an anti-tumour phenotype. Recent studies have shown that activating toll-like receptor-7 (TLR-7) with small molecules like resiquimod (R848) induces this shift. Although effective, TLR agonists like R848 carry risks of systemic toxicity due to limited targeting specificity. To address this, we utilize CD206, an endocytic receptor overexpressed on TAMs, as a targeted delivery pathway. However, existing CD206 ligands exhibit low affinity, limiting their ability to drive efficient endocytosis. To overcome this, we synthesized a CD206-binding glycopeptide with avidity-enhanced affinity and conjugated it to R848, creating an avidity-dependent drug conjugate (MiniAvDC) designed for TAM-specific reprogramming. Preliminary data confirm that our glycopeptide exhibits enhanced CD206 binding and internalization, as demonstrated by biolayer interferometry, fluorescence polarization, flow cytometry and fluorescence microscopy. Additionally, real-time PCR results indicate that MiniAvDC effectively reprograms TAM-like macrophage cell lines. We hypothesize that the avidity-enhanced affinity of MiniAvDC will lead to TAM-specific reprogramming, shifting TAMs from a pro-tumour to an anti-tumour state, resulting in tumour eradication. This strategy holds promise for improving immunotherapy outcomes by reshaping the TME into a more immune-permissive environment.

LAY SUMMARY

Some cancer cells can trick the patient's immune system into helping them grow instead of killing them. One type of immune cell, called macrophages, can be found near the tumours. These are known as tumour-associated macrophages (TAMs). Instead of fighting the cancer, TAMs can protect tumour cells and stop other immune cells from attacking, allowing the tumour to grow and spread. Although problematic, TAMs are flexible and can change their behaviour depending on signals they receive. Recently, scientists have found that a drug called R848 can send the right signals to turn TAMs back into cancer-fighting cells. To make sure R848 reaches the TAMs safely and effectively, we created a drug delivery system called avidity drug conjugate (MiniAvDC). This system connects R848 to a molecule that specifically targets TAMs and only releases the drug once inside the cell. This helps retrain the TAMs to attack cancer cells rather than protect them.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

An innovative in vitro model of cancer cell co-culture in a 3D vascularized microenvironment - Poster #850

Presenter: Seyedeh Minoo Alavi, UdeM

Other Authors: Anaïs Darracq, Arman Jafari, Houman Savoji, Noël Raynal

ABSTRACT_ID#3280

ABSTRACT

Conventional 2D cancer cell cultures inadequately mimic tumor-host interactions, limiting their predictive value. This limitation contributes to the failure of 90% of clinical drug candidates during clinical trials. 3D in vitro models offer improved realism but face challenges such as high costs and technical complexity. This study aims to develop an innovative scaffold for creating vascularized three-dimensional (3D) cell culture system. This system is designed to better replicate the tumor microenvironment, enabling more accurate studies of drug responses. The mold is a 3D-printed structure with two interlocking parts and 25 pillars simulating a capillary network. Cancer cells (5 million or more) are mixed in a 10% GelMA solution with culture medium and LAP, then polymerized under UV light for 2–3 minutes. The formed model is removed and transferred to culture plate. Initial mechanical and structural validation tests, including compression analysis, SEM imaging, swelling, and diffusion assays with media and fluorescent dyes, confirm the structural integrity of the model. Cell viability and functionality (tested for breast cancer cells, for lung cancer cells, and neuroblastoma cells) were assessed through drug treatments and brightfield microscopy demonstrated cell viability and proliferation within the model over one month. Experimental results, particularly the comparative drug response of cells within the model to clinically approved and failed drugs, demonstrate that our model provides a more pharmacological relevant model. This model has potential for predicting drug responses more accurately, bridging the gap between preclinical and clinical studies.

LAY SUMMARY

Traditional flat (2D) cancer cell cultures do not accurately reflect how tumors behave inside the body, which is one reason many drugs fail in clinical trials. Our research uses a 3D model that better mimics the real tumor environment. We created a new 3D-printed tool, which allows us to grow cancer cells inside a gel that mimics tissue. This model also includes tiny channels like blood vessels to help study how drugs move and act in more realistic conditions. We tested this model with several types of cancer cells and found that it supports long-term growth and gives more reliable results when testing both successful and failed cancer drugs. This system could help researchers better predict which drugs will work in patients.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Identifying Regulators of Human Leukocyte Antigen-I Silencing in SCLC to Enhance the Efficacy of Immunotherapies - Poster #851

Presenter: Pinky Sharma, University of Manitoba

Other Authors: Yale Michaels, Joel Pearson

ABSTRACT_ID#2458

ABSTRACT

Background: Small cell lung cancer (SCLC) is an aggressive neuroendocrine tumor with a 5-year survival rate of under 7%. Standard therapies include chemotherapy and recently immune checkpoint inhibitors (ICIs) are approved. However, ICIs offer limited benefit. This is largely due to low human leukocyte antigen-I (HLA-I) expression and impaired antigen presentation, driven mainly by epigenetic and transcriptional silencing rather than mutations. **Objectives:** Our work aims to deduce the epigenetic and transcriptional regulators of HLA-I silencing in SCLC, and to identify strategies to increase HLA-I expression to improve immunotherapy efficacy in SCLC patients. **Methods:** We will perform a pooled CRISPR-Cas9 knockout screen targeting epigenetic and transcription factors in SCLC cell lines. We will isolate cells with elevated HLA-I expression using cell sorting and identify enriched guide RNAs through deep sequencing to reveal genes whose knockout upregulates HLA-I. We will validate candidate genes using individual knockouts and pharmacological inhibitors. We will further test whether these treatments sensitize SCLC cells to immune cell recognition and tumor cell destruction. **Results:** We validated the knockout efficacy of the lentiCRISPR v2 vector system in SCLC cell lines. We also demonstrated that HLA-I could be upregulated in these cells, to identify a key positive control for the screen—establishing an appropriate model for the screen. **Conclusion:** This study will provide critical insights into the molecular mechanisms of HLA-I silencing in SCLC. Identifying key transcriptional and epigenetic regulators could inform the development of novel therapeutic strategies aimed at reversing immune evasion and improving immunotherapy outcomes in SCLC patients.

LAY SUMMARY

Why current immunotherapy doesn't work well in SCLC: Small cell lung cancer (SCLC) is very aggressive and doesn't respond well to immune-based treatments because the cancer hides from the immune system by turning off a key molecule (HLA-I) needed for immune recognition. **What the study is trying to do:** We aim to find out which genes are responsible for turning off HLA-I in SCLC. This could help uncover ways to turn HLA-I back on and make cancer cells visible to the immune system again. This will eventually set a foundation for discovering new targets to improve immunotherapy—a type of cancer treatment that stimulates a patient's immune system to attack tumours. **Early progress made:** We established a robust experimental model system consisting of SCLC in vitro cell lines confirming that HLA-I expression can be upregulated in these cells. Thereby, setting the stage for identification of genes that turn off HLA-I in SCLC.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Chemotherapy Reduces Protein Synthesis in T-cell Acute Lymphoblastic Leukemia - Poster #852

Presenter: Mathew Tempel, University of Manitoba

ABSTRACT_ID#2868

ABSTRACT

Background: Treatment for T-cell acute lymphoblastic leukemia (T-ALL) is limited to an aggressive chemotherapy regimen of vincristine, dexamethasone and L-asparaginase (VXL) that causes long-term side effects. Improvements to T-ALL treatment are needed but challenging due to the limited knowledge on how T-ALL cells respond and adapt to chemotherapy. Protein synthesis has emerged as a promising targetable pathway given its importance for cell function in homeostatic and stress conditions. **Aim:** To define the effects of chemotherapy on T-ALL cell function, informing novel therapeutic strategies. **Methods:** I determined the lethal concentration (LC50) of VXL by assessing viability of T-ALL cell lines by flow cytometry. I also assessed cell cycle distribution and changes in phosphorylation of the ribosomal protein S6 (pS6), which is an established marker of protein synthesis. I have validated the effects on protein synthesis using OP-Puromycin incorporation into nascent peptides in the same T-ALL cell lines. **Results:** Treatment of the T-ALL cell lines with the LC50 of VXL caused a significant reduction in pS6 after only 1 hour of exposure, prior to any measurable changes in cell cycle distribution. This decrease in pS6 is followed by a decrease in protein synthesis around 24 hours of VXL treatment. **Conclusion:** My results suggest that protein synthesis is impaired in response to chemotherapy-induced stress rather than the decreased proliferation which occurs later in T-ALL cells. Given these effects, targeting key components of this stress response might limit the capacity of T-ALL cells to adapt, thereby preventing resistance to chemotherapy and improving outcomes.

LAY SUMMARY

Chemotherapy induces a stress response that can be detected in cells after only 1 hour of treatment due to reduced ribosomal protein S6 phosphorylation/ activation before any changes in cell cycle. This stress response induces a detectable reduction in protein synthesis within 24 hours as determined by OP-Puromycin incorporation in nascent peptides. My data supports the hypothesis that protein synthesis is a logical target for novel chemotherapeutics targeting T-cell acute lymphoblastic leukaemia due to its central role in homeostasis and stress induced adaptation of cancer cells.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Epitype-immune interactions in high-grade serous ovarian cancer - Poster #853

Presenter: Sarah Nersesian, Ottawa Hospital Research Institute

Other Authors: John Abou-Hamad, Emma Durocher, Barbara Vanderhyden, David Cook

ABSTRACT_ID#3358

ABSTRACT

Treatment options for high-grade serous ovarian cancer (HGSC) are limited, and immunotherapy is only effective in ~10% of patients. There is a critical need to better understand the dynamic mechanisms of immune evasion in HGSC, which may be linked to local epithelial cells within tumours. We identified three epithelial phenotypes (“epitypes”: SecA, SecB, Cil) consistently across HGSC tumour samples within an integrated single-cell atlas. Understanding how each epitype interacts with immune cells can inform immunotherapy strategies applicable across HGSC tumours. To explore these interactions, we analyzed gene expression and cell communication using a single-cell RNA sequencing atlas (n=471 patients, ~2 million cells). Predicted co-localization and intercellular signaling were validated using targeted spatial transcriptomics on whole tissues (n=9 tumours) and a tissue microarray (n=101 tumours, n=15 normal). The three transcriptionally distinct epitypes show clinical relevance: SecB cells are present in nearly all samples but enriched in metastatic and chemo-treated tumours. SecB’s gene expression and communication patterns support an immune-modulating role, including association with IFN signaling and class I HLA pathways. Spatial mapping shows SecB often surrounds luminal regions and co-localizes with diverse immune cell types. To functionally validate these findings, co-culture assays are underway using HGSC cells and immune populations isolated from patient ascites, analyzed by flow cytometry. These preliminary findings establish a framework for designing targeted immunotherapies based on epithelial state and suggest that the SecB epitype plays a key role in immune evasion. Targeting SecB-specific mechanisms may improve immunotherapy response in HGSC.

LAY SUMMARY

High-grade serous ovarian cancer (HGSC) often avoids detection by the immune system, and current immunotherapy only works for about 10% of patients. Our research focuses on how different types of epithelial cancer cells may help tumours escape immune attack. We identified three main types of epithelial cancer cells—called epitypes (SecA, SecB, Cil)—across hundreds of HGSC tumours. These epitypes behave differently and interact with immune cells in unique ways. One type, SecB, is especially common in tumours that have spread or been treated with chemotherapy. SecB appears to play a key role in helping tumours evade the immune system. It shows high activity in immune-related pathways and is often found near immune cells in tumour tissue. These findings suggest that targeting the SecB epitype could lead to better immunotherapy options for more patients with HGSC. Co-culture experiments using real patient cells are underway to test this approach further.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Mapping the tumor microenvironment upon Laser Interstitial Thermal Therapy (LITT) in a Mouse Glioma Model - Poster #854

Presenter: Thomas Klonisch, University of Manitoba

Other Authors: Jeremy Spence, Santhosh Anandhan, Nimrat Kaur, Farhana Begum, Dana Henderson, Victor Spicer, Rene Zahedi, Sabine Hombach-Klonisch

ABSTRACT_ID#3108

ABSTRACT

Introduction: Glioblastoma accounts for 48%-60% of adult high-grade primary brain tumors and has a 5-year survival of only 4.8%. LITT is a minimally-invasive hyperthermic ablation method indicated for inoperable brain tumors. We have established a preclinical murine model of LITT in glioma to study cellular changes and explore future treatment targets. Methods: Syngeneic CT2A glioma cells were orthotopically implanted into C57BL/6 mice using a stereotactic frame. Tumor size was monitored using T2-weighted MRI. We performed LITT using a custom 1064nm fiber optic laser or surgical sham with conventional tissue damage. Brains, cervical lymph nodes, and plasma were collected at relevant timepoints, providing both local and systemic views of the inflammatory/immune processes in response to LITT. Results: LITT conditions were optimized to ensure effective ablation of tumor tissue. Multiplexed immunofluorescence highlighted spatiotemporal changes to resident and immune cell populations including activation of resident microglia and phagocyte recruitment. Astrocytes at the LITT site differed in marker expression suggesting distinct subpopulations. Single-cell spatial transcriptomics identified well-defined gene clusters indicative of distinct cell populations in mouse tumor brains at different time points post-LITT. Analysis of plasma proteomes was performed to reveal systemic protein markers in LITT brains which may be relevant in underpinning immunomodulatory mechanisms. Conclusion: We have established a LITT murine glioma model for investigating LITT effects on the tumor, tumor-microenvironment, and the systemic plasma proteome. This unique preclinical model empowers the discovery of combined LITT-drug treatments for improved therapeutic efficacy with the aim of improving quality of life for glioma patients.

LAY SUMMARY

We have established a mouse model for LITT which is a minimally-invasive method that uses laser heat to target and ablate brain tumors. We use several imaging techniques for multicolor characterization of innate and adaptive immune cells at different time points post-LITT in mouse brain tissues. We have determined gene expression profiles at LITT tissues sites using single cell spatial transcriptomics.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

A deep-learning approach for tissue mapping and cell phenotyping in imaging mass cytometry data analysis - Poster #856

Presenter: Toby Astill, Standard BioTools

Other Authors: James Mansfield

ABSTRACT_ID#2892

ABSTRACT

Imaging Mass Cytometry™ (IMC™) technology is a multiplexed imaging technique that generates high-dimensional spatial data at subcellular resolution without the complications of autofluorescence and cyclic imaging. It is ideal for cancer research and applicable for all types, both in a research and clinical study setting. IMC technology has two distinct whole slide imaging (WSI) modes: Preview Mode (PM) and Tissue Mode (TM). PM rapidly scans a stained tissue to provide a comprehensive overview within minutes, while TM provides fast acquisition of the entire tissue at 5-micron resolution, mapping out the distribution of over 40 markers and revealing tissue heterogeneity. Both of these WSI modes enable researchers to make informed decisions about selecting tissue areas that warrant closer examination at single-cell resolution. Following PM, regions of interest (ROIs) are selected on the same slide for high-resolution imaging using Cell Mode (CM). This facilitates single-cell analysis of the ROIs identified during PM. These imaging modes together with an automated slide loader function support non-stop acquisition of tissue samples all week.

LAY SUMMARY

IMC technology provides high-resolution, multiplexed imaging at the subcellular level without issues like autofluorescence or the need for cyclic imaging. Two whole slide imaging modes—Preview Mode and Tissue Mode—enable rapid scanning and detailed tissue mapping of over 40 markers to identify areas for deeper analysis. Cell Mode allows single-cell resolution imaging of selected regions, supported by an automated slide loader for continuous, high-throughput tissue sample analysis.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

A novel in vitro 3D cancer model based on a modular tissue engineering approach - Poster #857

Presenter: Nima Daneshvar Baghbadorani, University of Saskatchewan

Other Authors: Michael Dean Chamberlain, Mira Bosso, Rowen Green, Taylor Dzikowski

ABSTRACT_ID#3019

ABSTRACT

Breast cancer is one of the leading causes of death worldwide. Cancer stem cells (CSCs) as the regenerative subpopulation of a tumor, underlying resistance, metastasis and relapse can be counted as major contributors to this high mortality rate. Our study aims to decipher tumor cell behavior by adapting modular tissue engineering to make a novel 3D cancer model, called “microtissues”. The initial version of microtissues is a free-floating collagen hydrogel embedded only with cancer cells. Starting with triple negative breast cancer (TNBC) cell lines, our findings revealed that microtissues mimicked clinical levels of chemoresistance, and naturally developed a hypoxic core. Given the established role of hypoxia in promoting stemness, along with the observed chemoresistance, we started to assess microtissues in terms of stemness. Flow cytometric analysis of four different TNBC microtissues using a multi-color panel of established and emerging CSC markers showed distinct expression compared to 2D-cultured cells. Notably, the Leucine-rich repeat-containing G-protein coupled receptor 5 (LGR5) was significantly upregulated in microtissues. Contrarily, its expression was below 1% in other tested 3D models, including spheroids and Geltrex™ domes. Although breast cancer patient cohorts have identified LGR5 as a prognostic factor and possible target for cancer therapy, this marker has been poorly characterized among in vitro studies. Overall, our findings highlight the potential of microtissues in recapitulating fundamental features of a tumor, including treatment resistance, hypoxia and stemness. Future work will focus on elucidating the role of LGR5, and its downstream signaling pathways in determining tumor cell behavior.

LAY SUMMARY

Our model could advance personalized cancer treatment. Our study can be counted as a step forward towards recruitment of these microtissues in the personalized medicine field, to operate drug sensitivity tests in the lab for patients on the microtissues developed by their own tumor cells, guiding treatment decisions without exposing patients to trial-and-error therapies.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Therapeutic targeting of extracellular vesicles in breast cancer metastasis - Poster #858

Presenter: Urvi Patel, Western University

Other Authors: Braeden Medeiros, Austyn Roseborough, David Goodale, Alison Allan

ABSTRACT_ID#2503

ABSTRACT

Breast cancer is a prominent cause of cancer diagnosis and death in women worldwide. The triple negative breast cancer (TNBC) subtype has the worst prognosis and an increased propensity for lung metastasis. Previously, our lab demonstrated that extracellular vesicles (EVs) from TNBC primary tumours can prime the lung to develop a pre-metastatic niche. Therefore, we hypothesized that TNBC-derived EVs induce molecular and functional changes in the lung microenvironment, and therapeutic inhibition of EV production/release reduces TNBC metastasis. We treated lung-metastasizing TNBC cells with tipifarnib, which a previous high throughput screen identified as an inhibitor of EV production and release (Datta et al., 2018). We observed that tipifarnib treatment of TNBC cells inhibited EV release and reduced migration in vitro as well as lung metastasis in vivo relative to vehicle control. To explore the molecular mechanisms underlying these functional effects, we assessed the expression of proteins involved in EV production including ALIX (ESCRT-dependent), nSMase2 (ESCRT-independent) and Rab27a (secretion). Expression of nSMase2 and Rab27a was significantly decreased in response to tipifarnib treatment in SUM159 and MDA-MB-231-LM2 cells while no change was observed in ALIX expression. These results indicate that tipifarnib influences ESCRT-independent production and secretion of EVs. Ongoing work is aimed at elucidating the EV cargo through RNAseq analysis. These studies will help us understand the role EVs play in lung metastasis and whether inhibiting EVs is a potential therapeutic strategy against TNBC metastasis.

LAY SUMMARY

Triple negative breast cancer has poor outcomes and often spreads to distant organs in a process called metastasis. The lung is a common location for metastasis. Breast cancer cells can release small particles called extracellular vesicles (EVs) that travel to the lungs, helping create a favourable environment for metastasis. We tested a drug, tipifarnib, to interrupt these EVs in triple negative breast cancer cells. We observed tipifarnib reduced the release of EVs, lowered the levels of proteins involved in EV production and decreased the mobility of the triple negative cancer cells. With this study, our goal is to better understand how triple negative breast cancer spreads to the lungs and find new ways to reduce metastasis.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Dual functions of IL-33 orchestrate tumour-associated macrophages in glioblastoma - Poster #859

Presenter: Tala-Maria Mouannes, McGill University

Other Authors: Shyam Menon, Xueqing Lun, Peipei Zeng, Jianbo Zhang, Isabelle Carrier, Eduardo Diez, Alisha Poole, Donna Senger, Stephen Robbins

ABSTRACT_ID#3135

ABSTRACT

Glioblastoma (GBM) is an aggressive brain tumour characterized by a profoundly immunosuppressive microenvironment dominated by tumour-supportive myeloid cells. Our group has identified interleukin-33 (IL-33), a dual-function cytokine with nuclear and extracellular roles, as a key modulator of this innate immune landscape. While full-length IL-33 promotes glioma progression by recruiting immunosuppressive glioma-associated macrophages (GAMs), a nuclear-deficient variant lacking the nuclear localization sequence (IL-33 Δ NLS) halts tumour growth and prolongs survival. This shift is characterized by the emergence of glioma-inhibiting macrophages (GIMs), a pro-inflammatory population with anti-tumour activity. Preliminary cytokine profiling reveals that IL-33 Δ NLS-expressing glioma cells secrete a distinct repertoire of immune-stimulatory factors, including IL-12p40 and TNF α . These cytokines are known to enhance innate immune activation, suggesting that the loss of IL-33's nuclear localization reshapes tumour-intrinsic signaling to drive myeloid reprogramming. Together, these findings position IL-33 as a critical regulator of macrophage polarization in GBM, where the balance between its nuclear and extracellular functions may be key in determining the shift between tumour-promoting and tumour-suppressive immune states. Ongoing studies aim to define the transcriptional programs that distinguish glioma-inhibiting macrophages from their tumour-promoting counterparts using bulk and single-cell RNA sequencing. By integrating molecular profiling with functional co-culture assays, this work seeks to uncover the mechanisms by which IL-33 shapes the tumour immune landscape and to identify targetable pathways for reprogramming tumour-associated macrophages (TAMs) toward a tumour-suppressive phenotype. This approach offers a promising avenue for leveraging myeloid plasticity to improve therapeutic outcomes in glioblastoma.

LAY SUMMARY

Glioblastoma, the most aggressive form of brain cancer, creates a tumour environment that attracts and reprograms immune cells to support tumour growth instead of fighting it. These immune cells—macrophages and microglia—are part of the body's innate immune system and are collectively known as tumour-associated macrophages (TAMs). We discovered that a protein called IL-33, produced by glioma cells, plays a key role in shaping this immune environment. IL-33 has two functions: one inside the cell and one outside. When IL-33 enters the cell nucleus, it promotes tumour growth by converting immune cells into tumour-supporting cells. However, when IL-33 is prevented from entering the nucleus, the tumour shrinks and immune cells become more active in attacking the cancer. Using gene analysis and laboratory models, we are investigating how IL-33 changes the behaviour of immune cells. This research could lead to new strategies to “reprogram” these cells to fight glioblastoma, offering new hope for therapies that target the tumour's immune environment.

Tumour Biology, Immunology and Microenvironment

Monday, November 3, 2025

Elucidating the role of phosphoproteome dynamics in regulating stemness and metastatic behavior in breast cancer cells - Poster #860

Presenter: Vasudeva Bhat, Western University

Other Authors: David Goodale, Carl Postenka, Daniel Menyhart, Laszlo Gyenis, David W Litchfield, Alison L Allan

ABSTRACT_ID#2534

ABSTRACT

Patients with triple-negative breast cancer (TNBC) are highly prone to lung metastasis, contributing to >90% mortality. The failure of current therapies in the metastatic setting highlights the urgent need for effective interventions and early detection, emphasizing the importance of identifying key drivers of metastasis. We previously demonstrated that breast cancer stem cells (BCSCs) identified by high aldehyde dehydrogenase activity (ALDH) and CD44 expression exhibit higher metastatic and lung colonization potential relative to their non-BCSC counterparts. However, the specific role of lung-derived soluble factors (LDSFs) in modulating TNBC stemness and metastasis remains poorly understood. In the current study, we hypothesized that stemness/plasticity, regulated by the lung microenvironment and post-translational modifications (PTMs) such as phosphorylation, can drive TNBC disease progression and therapy resistance. We observed that LDSFs secreted in the presence of a primary TNBC tumor significantly enhance the acquisition of a stem-like phenotype in breast cancer cells. In particular, fibroblast growth factor 2 (FGF2) enhances stemness acquisition in TNBC cells by suppressing the tumor suppressor, Dachshund homolog 1 (DACH1). Interestingly, prolonged DACH1 suppression is achieved only with sustained FGF2 signaling, and sustained FGF2 signaling is regulated by AKT phosphorylation. Phosphoproteomics analysis reveal that LDSFs secreted in the presence of a primary TNBC tumor activates the PI3K-AKT signaling pathway required to maintain stemness/plasticity. The results of this study uncover novel molecular mechanisms by which LDSFs regulate TNBC stemness and metastasis. Targeting the stemness acquisition process offers an innovative approach to understanding and potentially mitigating metastatic disease in patients in the future.

LAY SUMMARY

Triple-negative breast cancer (TNBC) is aggressive and harder to treat once it spreads to the lungs. We believe lung factors fuel TNBC cells, making them more adaptable and invasive. Our research explores how these factors trigger molecular changes through phosphorylation, a process that switches proteins on or off, altering networks that control cancer cell division, growth, and survival. We focus on how these signals promote stemness, enabling cancer cells to resist treatment and form new tumors in the lung. Using advanced techniques, we study how lung-derived factors activate TNBC signaling pathways, enhancing survival and formation of new tumors in the lung. By identifying and blocking key signals, we aim to uncover new strategies to limit lung metastasis and improve early detection. This work could improve breast cancer prediction, prevention, and treatment, saving lives.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

Investigating Temporal and Spatial Changes in Proteins Involved in Migration of Breast Cancer Cells in a Novel 3D Tumour Model - Poster #863

Presenter: Taylor Dzikowski, University of Saskatchewan

Other Authors: Dean Chamberlain

ABSTRACT_ID#2759

ABSTRACT

Among patients with the same breast cancer subtype, there can be significant differences in metastasis rates from the primary tumour to establish secondary tumours. This variation may be attributed to dynamic expression of metastasis-related proteins within primary tumours. The phenomenon can be studied using 3D tumour models. Our lab has developed a 3D tumour model of free-floating collagen cylinders containing cancer cells, termed microtissues. An advantage of this model is that cells can spontaneously migrate in collagen without influence from plastic rigidity. Our ongoing research aims to investigate the mechanisms behind the spontaneous migration of cancer cells within microtissues. Transcriptional analysis in two triple negative breast cancer (TNBC) cell lines, HCC1806 and MDA-MB-231, has revealed differences in EMT markers between the two cell lines as they migrate in microtissues. We utilized western blotting (WB) and immunofluorescent confocal microscopy (IF) to analyze the spatial and temporal changes in migration-related proteins. Interestingly, WB revealed temporal changes in migratory markers, while IF revealed that microtissues develop tumour heterogeneity despite containing a single cell line. Consistent with literature, these two TNBC cell lines exhibit differences in EMT status in microtissues, where HCC1806 is more epithelial and MDA-MB-231 is more mesenchymal. Thus far, we have observed that E- and N-cadherin, cytokeratin 14, and vimentin, are expressed in select cells in early timepoints of microtissue development, but not in every cell. In summary, our research focuses on using microtissues to characterize phenotypes of migratory breast cancer cells, paving the way for identifying potential metastasis drug targets.

LAY SUMMARY

The purpose of this study is to create a three dimensional tumour model that allows us to analyze how breast cancer cells migrate in the tumour microenvironment, and monitor how proteins that allow cancer cells to move and invade tissues change in activity over time during tumour development and metastasis. This knowledge will be fundamental in developing inhibitors of cancer cell migration and invasion. The implications of this research would allow oncologists to prevent breast cancer from spreading, so that once the breast tumour is removed, breast cancer patients would become cancer free.

CD47-mediated Cell Death is Enhanced by Bcl2/Mcl1 Inhibition and is Regulated by p21-activated Kinases - Poster #864

Presenter: Pascal Leclair, University of British Columbia

ABSTRACT_ID#3185

ABSTRACT

Acute lymphoblastic leukemia (ALL) is the most common pediatric cancer with a 5-year survival rate of >85%. However, the panoply of treatment required to accomplish this remain highly toxic, with side-effects including developmental defects and cardiotoxicity. Therefore, the use of adjuvant therapy that can enhance drug efficacy at lower concentrations are highly sought. CD47 is a transmembrane receptor protein most commonly known for its role as a “don’t eat” signal, with additional roles in cell adhesion, spreading, and migration. However, a less studied and underexploited property for CD47 is its role as an inducer of caspase-independent cell death. The anti-CD47 antibody, CC2C6, exhibits differential abilities to induce cell death in a variety of ALL cell lines and in patient-derived xenografts. We sought to uncover combination therapies able to enhance or simply sensitize tumors exhibiting low or no response to CC2C6 as a monoagent. Combined treatment of CC2C6 with inhibitors of Bcl2 or Mcl1, agents that induce caspase-dependent cell death, produced synergistic effects in ALL cells. We also found that CC2C6-induced cell death involved p21-activated kinase (PAK) activity, cofilin and modulation of the F-actin cytoskeleton. This was evidenced by pharmacological modulation of CD47-mediated cell death using inhibitors targeting cdc42/Rac1, PKD, ROCK and F-actin polymerization. We conclude that CD47-ligation induces extensive actin reorganization, a process required for efficient induction of cell death, and that tumour cells can be sensitized to CC2C6 treatment by targeting F-actin cytoskeletal dynamics.

LAY SUMMARY

- Adjuvant therapies are sought to reduce the long-term side-effects from the harsh treatment required to obtain the <85% 5-year survival rate in acute lymphoblastic leukemia.
- Anti-CD47 antibody can induce non-apoptotic, caspase-independent cell death, which can be sensitized by co-incubation with sub-optimal concentrations of cancer therapy agents which induce caspase-dependent cell death.
- Depolymerization and repolymerization of actin structures are required for efficient induction of CD47-mediated cell death.
- PAKs, cofilin and other regulators of actin dynamics regulate CD47-mediated cell death.

Tumour Biology, Immunology and Microenvironment

Sunday, November 2, 2025

The Emerging Role of Basophils in Non-Muscle-Invasive Bladder Cancer - Poster #865

Presenter: Geneviève Trépanier, Research Centre of the CHU de Québec – Université Laval

Other Authors: Typhaine Gris, Paul Toren

ABSTRACT_ID#2774

ABSTRACT

Non-muscle-invasive bladder cancer (NMIBC) is marked by a high recurrence rate (>40 %) despite transurethral resection and intravesical BCG immunotherapy, highlighting the urgent need for novel prognostic biomarkers. In our retrospective multicentre study, we identify circulating basophils as independent predictive factors for both recurrence (HR = 1.9; $p = 0.010$) and tumour progression (HR = 2.3; $p = 0.020$). Traditionally linked to inflammation, basophils also appear to contribute to immunosuppression by modulating the tumour microenvironment (TME) and promoting BCG resistance. We hypothesize that basophil expansion reflects systemic immune dysregulation facilitating recurrence. Our project combines (1) characterization of activation profiles in circulating and tumour-associated basophils, and (2) investigation of their interactions with cancer cells to clarify their impact on the TME. Flow cytometry of 60 NMIBC patients revealed significantly increased granzyme B expression ($p = 0.0008$) and a trend toward higher interleukin (IL)-4 and IL-13 in circulating basophils compared to healthy controls. These findings suggest a paradoxical mechanism where granzyme B, typically antitumoural, may instead drive immune evasion and tissue invasion. To explore tumoural recruitment of these cells, urine-based analyses targeting basophils are currently underway. In parallel, in vitro co-culture models show that tumour cell-activated basophils selectively inhibit helper T cell proliferation ($\sim 30\%$; $p = 0.004$), without affecting cytotoxic T cells. This selective suppression underscores their potential immunosuppressive role within the TME. Our results position basophils as key mediators of BCG resistance and accessible blood-based biomarkers for high-risk NMIBC patients, paving the way for personalized risk stratification and optimized therapies.

LAY SUMMARY

Some patients with non-muscle-invasive bladder cancer have higher numbers of basophils, a rare type of white blood cell, in their blood. Our research shows that these patients are more likely to have their cancer return or progress, even after receiving standard treatments like surgery and BCG immunotherapy. Measuring basophil levels in the blood could help doctors identify which patients are at higher risk of recurrence or progression. This information may allow for more personalized monitoring and treatment plans, potentially improving patient outcomes. Our ongoing and future research is focused on understanding why basophils are increased in some patients and how they interact with cancer cells and the immune system. We are studying the specific signals and behaviors of basophils in both the blood and the tumor environment. By learning more about the role of basophils, we hope to discover new ways to predict which patients are at risk and to develop targeted therapies that could prevent cancer from coming back or getting worse. Ultimately, our goal is to use this knowledge to help personalize bladder cancer care, making treatments more effective for each individual patient.

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