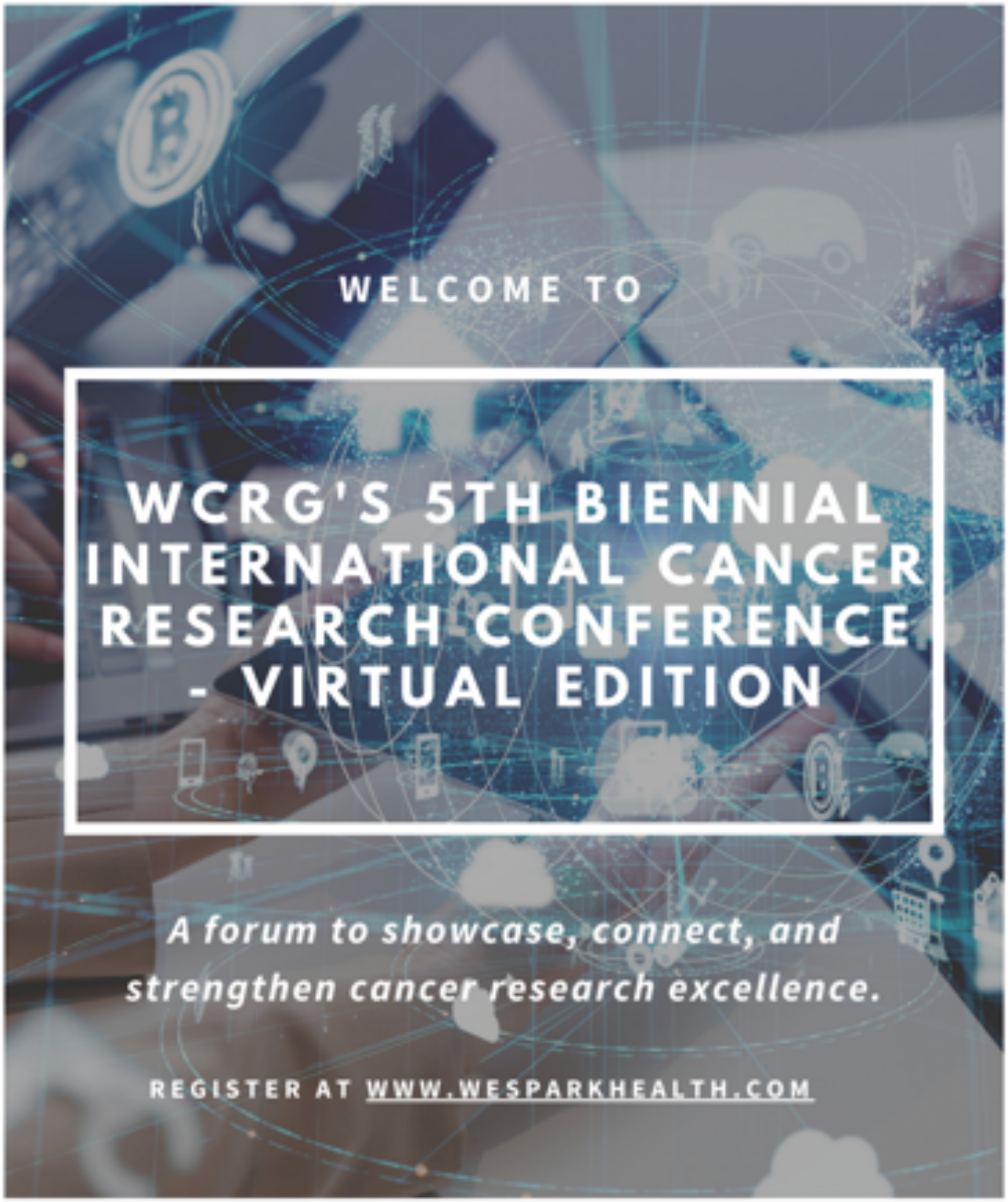

November 19 - 21, 2020

An affiliated program of WE-SPARK

Windsor Cancer Research Group

we spark HEALTH INSTITUTE

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Welcome!

Thank you for joining us for the **Windsor Cancer Research Group's 5th Biennial International Cancer Research Conference – Virtual Edition**. With over 200 registrants joining us from across Canada, the United States, the Philippines, Iran, Switzerland and South Africa, this year's conference is our largest yet. Presenters include a diverse group of researchers, clinicians, students and community partners representing a wide range of cancer research areas. We have an exciting three days ahead and we are thrilled that you are here with us!

Zoom Meeting Links

Thursday: https://us02web.zoom.us/j/89666958297
Friday: https://us02web.zoom.us/j/84811991780
Saturday: https://us02web.zoom.us/j/81149899450

Zoom Requirements

Upgrade to at least 5.3.0 or the latest version of Zoom (link to Zoom article here)

1. Sign into Zoom on desktop
2. Click your profile icon (top right hand corner) and then click **Check for Updates**
3. If there is a newer version, Zoom will download it an install it

Note: It is important your Zoom account is upgraded to the latest version to ensure you can enter and move freely between breakout rooms during the poster sessions and social networking event. The links above will give you access to all events taking place each day.

When tweeting about the conference, please use the hashtag:

#WCRG2020

ABOUT Windsor Cancer Research Group

Mission: To establish Windsor-Essex as an international hub of cancer research excellence.

Vision: A united community bridging cutting edge research with world-class cancer care.

The **Windsor Cancer Research Group (WCRG)** is an assembly of local researchers, healthcare professionals and community partners working together to build teams to strengthen our local cancer research programs and bridge collaboration with our neighbouring cancer centers. As the flagship cancer program for new WE-SPARK Health Institute that brings together health research strengths, expertise, and infrastructure from across the Windsor-Essex region of Ontario, WCRG is committed to:

- Building local cancer research programs and clinical trials to advance cancer treatments and care.
- Growing collaborative cancer research networks with national and international partners.
- Supporting state-of-the-art facilities equipped with advanced technologies for cancer imaging and diagnostics.
- Providing training opportunities for the next generation of local researchers and clinicians.
- Building knowledge translation programs focused on cancer research and education
- Engaging with our local community partners to improve health care outcomes for patients

We look forward to our continued work with our community and to an even brighter future ahead!

LAND ACKNOWLEDGEMENT

We would like to acknowledge the traditional, ancestral, unceded territory of the Three Fires Confederacy of First Nations, comprised of the Ojibway, the Odawa, and the Potawatomie. It is the territory on which the University of Windsor sits, and where we are fortunate to be delivering this virtual conference from.

It is important to honour the Indigenous community and their contributions, as without them, we would not be able to do the important and impactful work we do. It is also important to welcome our indigenous visitors to this area including the Haudenosaunee and other nations.

Schedule Overview

Thursday, November 19, 2020 https://us02web.zoom.us/j/89666958297

1:00 pm	Welcome
1:05 pm	Windsor Cancer Research Spotlight – ***"WCRG: Transformation through Collaboration"***
1:35 pm	Patient Perspective: Maria Bommarito
1:45 pm	Rapid-Fire Session
2:30 pm	Joint Session
3:45 pm	Poster Session – Group A
4:45-5:30 pm	EDI Conversations

Friday, November 20, 2020 https://us02web.zoom.us/j/84811991780

1:00 pm	Welcome
1:05 pm	Ambassador of Hope Perspective: Dr. Hakam Abu-Zahra
1:15 pm	Joint Session
2:30 pm	Rapid-Fire Session
3:15 pm	Keynote Address: Dr. Sheila Singh, MD PhD FRSC(C) - ***"Targeting clonal heterogeneity in treatment-refractory Glioblastoma with novel and empiric immunotherapies".***
4:00 pm	Poster Session – Group B
5:00-6:00 pm	Virtual Social Event – Networking & Chats and Cheers

Saturday, November 21, 2020 https://us02web.zoom.us/j/81149899450

9:00 am	Welcome
9:05 am	Student-Patient Perspective: Dereck Lau
9:15 am	Keynote Address: Dr. Lucy Godfrey, MD PhD - ***"Germline predisposition to hematopoietic malignancies"***
10:00 am	Rapid-Fire Session
10:45 am	Joint Session
12:00 pm	Closing Remarks & Award Presentation
12:15-1:00 pm	Workshop: Canadian Cancer Society – Research Information and Outreach Team (RIOT) – Windsor

Keynote Speakers

Dr. Sheila Singh, MD PhD FRSC(C)

Chief Pediatric Neurosurgeon, McMaster Children's Hospital
Division Head of Neurosurgery, Hamilton Health Sciences
Professor of Surgery and Biochemistry, McMaster University

Keynote Presentation: ***"Targeting clonal heterogeneity in treatment-refractory Glioblastoma with novel and empiric immunotherapies"***

Friday, November 20th at 3:15pm

Dr. Sheila Singh is a professor of surgery and biochemistry, chief pediatric neurosurgeon at McMaster Children's Hospital, Division Head of Neurosurgery at Hamilton Health Sciences, and scientist appointed to the Stem Cell and Cancer Research Institute at McMaster University. She holds a Tier 1/ Senior Canada Research Chair in Human Brain Cancer Stem Cell Biology, and is Director of the McMaster Surgeon Scientist Program.

Her PhD thesis described the novel identification of a population of cancer stem cells that exclusively drive the formation of brain tumours. Since 2007, Dr. Singh's lab applies a developmental neurobiology framework to the study of brain tumorigenesis. Building upon previous cell culture techniques developed for the isolation of normal neural stem cells (NSC) and applying them to brain tumours, and through development of a xenograft model to efficiently study brain tumour initiating cell (BTIC) activity, Dr. Singh's lab aims to understand the molecular mechanisms that govern BTIC self- renewal. Dr. Singh is currently studying the regulation of BTIC signaling pathways in glioblastoma, brain metastases and childhood medulloblastoma, with an ultimate goal of selectively targeting the BTIC with appropriately tailored drug and molecular therapies. Her laboratory is funded by CCSRI, CIHR, TFRI, CRS, the Stem Cell Network, McMaster Surgical Associates, Brain Canada and the Boris Family Fund.

She is scientific founder and interim CEO of a start-up company, Empirica Therapeutics, a brain cancer therapeutics company that is seeking new, data-driven and polytherapeutic treatment options for patients with Glioblastoma and brain metastases. Empirica aims to translate research discoveries co-developed at McMaster University and University of Toronto through clinical trials and into the clinic.

Dr. Lucy Godley, MD PhD

Hematology/Oncology, University of Chicago
Hospira Foundation Professor of Medicine & Human Genetics

Keynote Presentation: ***"Germline predisposition to hematopoietic malignancies"***

Saturday, November 21st at 9:15am

Dr. Godley developed her deep respect for science through her work in the Marchesi laboratories (Yale), Dr. Don Wiley (Harvard), and Dr. Harold Varmus (University of California, San Francisco and the National Institutes of Health). She completed her medical training at Northwestern University followed by Internal Medicine/Hematology-Oncology residency/fellowship at The University of Chicago. After postdoctoral research with Dr. Michelle Le Beau, Dr. Godley joined the faculty at The University of Chicago in 2003.

As a physician-scientist with both research and clinical responsibilities, Dr. Godley seeks to understand disease on a molecular basis and is able to bring that perspective to the care of her patients. Her laboratory studies the molecular drivers of inherited hematopoietic malignancies and how the distribution of covalently modified cytosines in DNA influences cellular differentiation during hematopoiesis and tumorigenesis. Dr. Godley has established a robust clinical and translational pipeline for testing for germline predisposition mutations, and individuals who test negative for known risk alleles have contributed to research leading to the identification of two new predisposition syndromes.

Dr. Godley serves as the Co-Chair of the American Society of Hematology Friday Scientific Workshop on Inherited Hematopoietic Malignancies as well as the ClinGen Myeloid Malignancy Variant Curation Expert Panel.

The Impact of Research: Perspectives

Maria Bommarito

Patient Perspective

Thursday, November 19th at 1:05pm

Maria Bommarito was born in Windsor Ontario. She is a registered dental hygienist and clinical instructor for the Dental Hygiene and Dental Assisting program at St Clair College. She is currently working towards completing her degree in Adult Education. Maria has been fighting Hodgkins lymphoma for the last 12 years and has participated in a number of clinical trials, local and abroad. She was a Woman's Ambassador for this year's Windsor Regional Cancer Centre's 2020 Lockout Cancer campaign and is a passionate advocate for cancer awareness, research and treatment.

Dr. Hakam Abu-Zahra, MD

Ambassador of Hope Perspective

Friday, November 20th at 1:05pm

Dr. Abu-Zahra graduated from Cairo University, Egypt and did postgraduate training in Hematology/Internal Medicine at Dundee and Edinburgh Universities in Scotland. In 1969, he arrived in Canada where he accepted a position as Research Fellow at Queen's University in Kingston and then Research Associate at Princess Margaret Hospital, University of Toronto. In Windsor, he was Head of the Systemic Therapy Program, then Chief Executive Officer, Windsor Regional Cancer Care and Vice-President, Cancer Care Ontario. He was Chief of the Oncology Department, Windsor Regional Hospital and Founding Member of the Windsor Cancer Centre Foundation. In 2020, Dr. Abu-Zahra was the recipient of the WCRG Ambassador of Hope award in recognition of his significant contribution to building cancer research in Windsor-Essex.

Dereck Lau

Student-Patient Perspective

Saturday, November 21st at 9:05am

Dereck is currently enrolled in the Grade 11 International Bacculeraute Program at Riverside High School. This September he celebrated being 5-year cancer free from Medulloblastoma, a form of Brain Cancer. He enjoys video games, role playing games, card games and spending time with his family. In 2017, Derek was selected as our inaugural WCRG's Researcher for a Day student.

DETAILED SCHEDULE

Thursday, November 19, 2020 https://us02web.zoom.us/j/89666958297	
1:00 pm	Welcome
1:05 pm	Windsor Cancer Research Spotlight – ***"WCRG: Transformation through Collaboratio***n" *Presented by:* ***Dr. Lisa Porter, PhD*** *– Executive Director, WE-SPARK Health Institute* ***Dr. Caroline Hamm, MD*** *– Clinical Research Director, WCRG* ***Dr. Dora Cavallo-Medved, PhD*** *– Translational Research Director, WCRG*
1:35 pm	Patient Perspective: **Maria Bommarito**
1:45 pm	 Rapid-Fire Session *Sponsored by Play for a Cure and the Cancer Research Collaboration Fund* **Moderator**: *Karen Metcalfe, Assistant Director WE-SPARK Health Institute* Abstract P54 THE LOSS OF CIRCADIAN CLOCK GENE BMAL1 INCREASES TUMOUR INITIATION IN APCMIN MICE **Kyle Stokes** Presenter Affiliation: University of Windsor Abstract P55 ANALYSIS OF THE GENOMIC LANDSCAPES OF BARBADIAN AND NIGERIAN WOMEN WITH HIGH PREVALENCE OF TRIPLE NEGATIVE BREAST CANCER (TNBC) **Shawn Hercules** Presenter Affiliation: McMaster University Abstract P56 DIKETOPYRROLOPYRROLE-BASED CONJUGATED POLYMER NANOPARTICLES TOWARD NEW THERANOSTICS FOR GLIOBLASTOMA MULTIFORME **Gage Mason** Presenter Affiliation: University of Windsor Abstract P57 PROMOTION OF THROMBIN ACTIVATABLE FIBRINOLYSIS INHIBITOR (TAFI) ACTIVATION IN THE TUMOUR MICROENVIRONMENT ATTENUATES BREAST CANCER METASTASIS IN VIVO. **Tasnim Reza** Presenter Affiliation: University of Western Ontario

	Abstract P58 EVALUATION OF NATURAL EXTRACTS IN COMBINATION WITH CHEMOTHERAPIES ON NEURO- AND GLIOBLASTOMA **Darcy Wear** Presenter Affiliation: University of Windsor Abstract P59 EPIDERMAL GROWTH FACTOR RECEPTOR SIGNALING REQUIRES SPECIALIZED CLATHRIN-LABELED STRUCTURES AND NECESSARY ACCESSORY PROTEINS **Rebecca Cabral-Dias** Presenter Affiliation: Ryerson University Abstract P60 MAGNETIC RESONANCE IMAGING ROUND METALLIC IMPLANTS USING SPRITE **Layale Bazzi** Presenter Affiliation: University of Windsor
2:30 pm	## Joint Session **Moderators**: *Krista Naccarato, Windsor Regional Hospital & Lisa Porter, University of Windsor and WE-SPARK Health Institute* Abstract O61 THERAPEUTIC STRATEGIES TO TARGET AUTOPHAGY IN METASTATIC EPITHELIAL OVARIAN CANCER **Trevor Shepherd** Presenter Affiliation: University of Western Ontario Abstract O62 TRANSCRIPTOMIC CHARACTERIZATION OF LUNG CANCER HETEROGENEITY USING SCRNA-SEQUENCING **Paola Marignani** Presenter Affiliation: Dalhousie University Abstract O63 PRECLINICAL IMAGING OF SPONTANEOUSLY METASTASIZING BREAST CANCER CELLS IN MICE **Nivin Nyström** Presenter Affiliation: Robarts Research Institute
3:45 pm	## Poster Session – Group A *Sponsored by the University of Windsor's Faculty of Science and Alumni Association* Abstract P01 THE ROLE OF SPY1 IN MAMMARY INVOLUTION AND ONCOGENESIS **Isabelle Hinch**

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Danish Mahmood

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Raghd Alolabi

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Subidsa Srikantha, Jake Frank & Kaila Wilson

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Nitin Rai

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Nourhan Shalaby

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UNDERSTANDING THE PATHWAYS ASSOCIATED WITH PROLACTIN SIGNALING IN BONE METASTATIC BREAST CANCER CELLS
Isbel Lopetegui-Gonzalez

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PSYCHOSOCIAL AND FINANCIAL BURDEN OF CANCER DIAGNOSES IN MULTIPLE MEMBERS OF THE SAME FAMILY IN WINDSOR, CANADA
Amanda Stojcevski & Katie McLaughlin

	Abstract P27 HOW FAR AWAY IS THE FIRST BORON NEUTRON CAPTURE THERAPY CENTRE IN CANADA **Ming Pan** Abstract P28 PATTERN OF TREATMENT INITIATION AND OUTCOMES FOR PATIENTS WITH METASTATIC NON-SMALL CELL LUNG CANCER IN ONTARIO **Jerry Ding** Abstract P29 DIFFERENTIAL RESPONSES TO A CARBOPLATIN CONTAINING REGIMEN IN THE HUMAN EPIDERMAL GROWTH FACTOR RECEPTOR 2-POSITIVE, HORMONE RECEPTOR-NEGATIVE BREAST CANCER POPULATION **Vanessa Montemurri** Abstract P30 EARLY STAGE LUNG CANCER STEREOTACTIC BODY RADIATION THERAPY OUTCOMES IN WINDSOR ONTARIO: A SINGLE INSTITUTION EXPERIENCE **Nathan Doupnik**
4:45-5:30 pm	EDI Conversations **Moderator**: Dr. Juliet Daniels PhD, McMaster University **Panelists**: Jaimie Kechego, Indigenous Curriculum & Pedagogy Project Coordinator, University of Windsor & Stefanie Pest, Co-Chair of Ontario NDP LGBTQ Committee
Friday November 20, 2020 https://us02web.zoom.us/j/84811991780	
1:00 pm	Welcome
1:05 pm	Ambassador of Hope Perspective: **Dr. Hakam Abu-Zahra**
1:15 pm	Joint Session **Moderators**: *Ingrid Qemo, University of Windsor & Trevor Shepherd, University of Western Ontario* Abstract O64 FROM MOLECULAR MECHANISM TO DRUG CANDIDATE, HOW WE STOP THE VICIOUS CYCLE OF BREAST CANCER BONE METASTASIS **Carrie Shemanko** Presenter Affiliation: University of Calgary Abstract O65 TARGETED CRISPR/CAS9 ENGINEERING FOR MULTI-MODALITY REPORTER GENE-BASED CELL TRACKING.

	John Kelly Presenter Affiliation: Robarts Research Institute Abstract O66 PROPHYLATIC USE OF MEPITEL FILM FOR BREAST CANCER PATIENTS UNDERGOING CHESTWALL IRRADIATION: A SINGLE INSTITUTION EXPERIENCE **Laura D'Alimonte** Presenter Affiliation: Windsor Regional Hospital
2:30 pm	## Rapid-Fire Session *Sponsored by Play for a Cure and the Cancer Research Collaboration Fund* **Moderator**: *Tina Suntres, University of Windsor* Abstract P17 HIGHLY METASTATIC CLAUDIN-LOW MAMMARY CANCERS CAN ORIGINATE FROM LUMINAL EPITHELIAL CELLS **Patrick Rädler** Presenter Affiliation: Eppley Institute for Research in Cancer and Allied Diseases Abstract P18 MAGNETIC RESONANCE IMAGING DATA ACQUISITION ACCELERATION AND FEATURE DETECTION WITH DICTIONARY LEARNING **Mark Armstrong** Presenter Affiliation: University of Windsor Abstract P19 KNOCKOUT OF THE METABOLIC STRESS MEDIATOR LKB1 OR ITS SUBSTRATE NUAK1 ENHANCES NF-KB SIGNALLING IN A SPHEROID MODEL OF OVARIAN CANCER METASTASIS **Adrian Buensuceso** Presenter Affiliation: University of Western Ontario Abstract P20 PREDICTING BIOLOGICALLY IMPORTANT RESIDUES USING IN SILICO MUTAGENESIS IN THE TUBEROUS SCLEROSIS COMPLEX **Adam Pillon** Presenter Affiliation: University of Windsor Abstract P21 A DUAL PET/MRI REPORTER GENE SYSTEM FOR CELL TRACKING **Nourhan Shalaby** Presenter Affiliation: University of Western Ontario

	Abstract P22 THE ROLE OF PROLACTIN IN TUMOR PROGRESSION AND TUMOR MICROENVIRONMENT IN RESPONSE TO DNA DAMAGING AGENTS **Odul Karayazi Atici** Presenter Affiliation: University of Calgary Abstract P23 UNDERSTANDING THE PATHWAYS ASSOCIATED WITH PROLACTIN SIGNALING IN BONE METASTATIC BREAST CANCER CELLS **Isbel Lopetegui-Gonzalez** Presenter Affiliation: University of Calgary
3:15 pm	## Keynote Address **Dr. Sheila Singh, MD PhD FRSC(C)** *"Targeting clonal heterogeneity in treatment-refractory Glioblastoma with novel and empiric immunotherapies".* **Moderator***: Dr. Lisa Porter, PhD* *Sponsored by the Office of the Vice-President of Research and Innovation*
4:00 pm	## Poster Session – Group B *Sponsored by the University of Windsor's Faculty of Science and Alumni Association* Abstract P07 CRYSTAL STRUCTURE AND ACTIVITY-BASED LABELING REVEAL THE MECHANISMS FOR LINKAGE-SPECIFIC SUBSTRATE RECOGNITION BY DEUBIQUITINASE USP9X **Cody Caba** Abstract P10 SIMULATED CANCER MUTATIONAL SIGNATURES TO CLASSIFY CANCER TYPES USING AN ALIGNMENT-FREE MACHINE LEARNING-BASED APPROACH **David Chen** Abstract P31 THE NKR-P1B:CLR-B AXIS' INHIBITORY EFFECTS ON NK CELL-MEDIATED IMMUNE RESPONSES AGAINST MAMMARY TUMORS **Abdel Hendy** Abstract P32 EVALUATION OF HIGH-SENSITIVITY BREAST SPECIFIC POSITRON EMISSION MAMMOGRAPHY (PEM) SYSTEM **Justin Stiles**

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Lindyann Lessey

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Josée Jarry + Hannah Lauzon

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Priyanka Philip

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Dorota Lubanska

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	OPTIONS IN CANCER. **Abedalrhman Alkhateeb** Abstract P54 THE LOSS OF CIRCADIAN CLOCK GENE BMAL1 INCREASES TUMOUR INITIATION IN APCMIN MICE **Kyle Stokes** Abstract P56 DIKETOPYRROLOPYRROLE-BASED CONJUGATED POLYMER NANOPARTICLES TOWARD NEW THERANOSTICS FOR GLIOBLASTOMA MULTIFORME **Gage Mason** Abstract P57 PROMOTION OF THROMBIN ACTIVATABLE FIBRINOLYSIS INHIBITOR (TAFI) ACTIVATION IN THE TUMOUR MICROENVIRONMENT ATTENUATES BREAST CANCER METASTASIS IN VIVO. **Tasnim Reza** Abstract P58 EVALUATION OF NATURAL EXTRACTS IN COMBINATION WITH CHEMOTHERAPIES ON NEURO- AND GLIOBLASTOMA **Darcy Wear** Abstract P59 EPIDERMAL GROWTH FACTOR RECEPTOR SIGNALING REQUIRES SPECIALIZED CLATHRIN-LABELED STRUCTURES AND NECESSARY ACCESSORY PROTEINS **Rebecca Cabral-Dias**
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10:00 am	 Rapid-Fire Session *Sponsored by Play for a Cure and the Cancer Research Collaboration Fund* **Moderator**: *Dr. Simon Rondeau-Gagne, PhD* Abstract P24 A RETROSPECTIVE SINGLE CENTER STUDY INVESTIGATING THE CLINICAL SIGNIFICANCE OF GRADE IN TRIPLE NEGATIVE BREAST CANCER **Sarang Upneja** Presenter Affiliation: Schulich School of Medicine & Dentistry Abstract P25 MEASLES REACTIVITY IN WINDSOR PATIENTS POST AUTOLOGOUS VERSUS ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION: A RETROSPECTIVE REVIEW **Kayla Negus** Presenter Affiliation: Schulich School of Medicine & Dentistry Abstract P26 PSYCHOSOCIAL AND FINANCIAL BURDEN OF CANCER DIAGNOSES IN MULTIPLE MEMBERS OF THE SAME FAMILY IN WINDSOR, CANADA **Amanda Stojcevski & Katie McLaughlin** Presenter Affiliation: Schulich School of Medicine & Dentistry Abstract P27 REAL WORLD EXPERIENCE – STOPPING STUDY IN CHRONIC MYELOID LEUKEMIA **Greg Yousif** Presenter Affiliation: Schulich School of Medicine & Dentistry Abstract P28 PATTERN OF TREATMENT INITIATION AND OUTCOMES FOR PATIENTS WITH METASTATIC NON-SMALL CELL LUNG CANCER IN ONTARIO **Jerry Ding** Presenter Affiliation: Schulich School of Medicine & Dentistry Abstract P29 DIFFERENTIAL RESPONSES TO A CARBOPLATIN CONTAINING REGIMEN IN THE HUMAN EPIDERMAL GROWTH FACTOR RECEPTOR 2-POSITIVE, HORMONE RECEPTOR-NEGATIVE BREAST CANCER POPULATION **Vanessa Montemurri** Presenter Affiliation: Schulich School of Medicine & Dentistry

	Abstract P30 EARLY STAGE LUNG CANCER STEREOTACTIC BODY RADIATION THERAPY OUTCOMES IN WINDSOR ONTARIO: A SINGLE INSTITUTION EXPERIENCE **Nathan Doupnik** Presenter Affiliation: Schulich School of Medicine & Dentistry
10:45 am	## Joint Session **Moderator**: *Dr. Dora Cavallo-Medved, PhD & Dr. Laurie Freeman, PhD* PROJECT FLAMINGO **Dr. Liana Roodt** Presenter Affiliation: Groote Shuur Hospital Breast and Endocrine Unity and University of Cape Town, South Africa Abstract O67 CANADIAN CLINICAL TRIALS NAVIGATOR: IMPROVING ACCESS TO CLINICAL TRIALS **Caroline Hamm** Presenter Affiliation: University of Windsor Abstract O68 RETHINKING PROSTATE CANCER IMAGING **Lisa Porter** Presenter Affiliation: University of Windsor
12:00 pm	## Closing Remarks & Award Presentation *Dr. Dora Cavallo-Medved & Dr. Caroline Hamm* *Student poster awards sponsored by the University of Windsor's Alumni Association and the Faculty of Science* *Rapid Fire Awards sponsored by Play for a Cure and the Cancer Research Collaboration Fund*
12:15-1:15 pm	## Workshop for Students and Trainees **Canadian Cancer Society – Research Information and Outreach Team (RIOT) – Windsor** *Organized by Dr. Bre-Anne Fifield, University of Windsor*

ABSTRACTS

ABSTRACT P01

THE ROLE OF SPY1 IN MAMMARY INVOLUTION AND ONCOGENESIS

Isabella Hinch[1*], Bre-Anne Fifield[1], Lisa Porter[1]

[1]University of Windsor

One in eight women will be diagnosed with breast cancer. From puberty to menopause, factors attributed with breast cancer fluctuate with the natural mammary development. A period of increased breast cancer risk, with increased metastasis and mortality, occurs following childbirth. This may be linked to the process of involution: mammary gland remodeling to non-lactating tissue balancing high rates of apoptosis and cell regeneration. These processes are controlled by the cell cycle and its regulators. The cyclin-like protein Spy1 can enable cell proliferation and override apoptosis. Spy1 levels have been found to be elevated in all types of human breast cancers. Interestingly, levels of Spy1 are also elevated during involution. We hypothesized that Spy1 protects the cell population necessary for normal mammary gland reconstitution post involution. To address this, an in vitro mock involution model was deployed with the murine epithelial cell line (HC11) over a time course of delivery and withdrawal of hormonal cues. This was paired with in vivo tissue collection of the mouse model overexpressing Spy1 in the mammary gland (MMTV-Spy1) over an involution time course for analysis. In vitro results suggest the ability of Spy1 of maintaining high proliferative abilities and stemness post-differentiation; whereas in vivo data indicates decreased proliferation and apoptosis in involution maintaining higher epithelial content. This research begins to articulate the role of Spy1 during normal mammary involution in maintaining the survival of epithelial cell populations, and how overexpression could potentially play a role in the predisposition of the breast to oncogenesis.

ABSTRACT P02

PARADISE TREE EXTRACT (SIMAROUBA GLAUCA) SELECTIVELY INDUCES CELL DEATH, ENHANCES EFFICACY OF COMMON CHEMOTHERAPEUTICS AND REDUCES THEIR TOXICITY IN IN-VITRO AND IN-VIVO

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Although chemotherapeutics have proven to be effective in treating metastatic breast cancer, their limited target selectivity has resulted in adverse side effects, rendering them unsuitable for long-term usage. Alternatively, certain natural extracts provide a promising strategy to selectively target cancer while being safe to consume. Specifically, paradise tree (Simarouba glauca) has shown potential anti-cancer activity; however, its efficacy against cancer, mechanism of action, and interaction with standard chemotherapies have not been investigated. We have demonstrated the anti-cancer activity of ethanolic paradise tree extract (PTE) in triple-negative and ER-positive breast cancer cell lines and its interaction with chemotherapeutics when used in combination. Our results have shown that PTE selectively triggers apoptosis in breast cancer cells while having limited effects on noncancerous cells. Importantly, we have found that PTE enhances the anti-cancer efficacy of chemotherapeutics, cisplatin and taxol, when given in combination, while reducing their toxicity in noncancerous cells. Furthermore, PTE inhibits growth of human tumor xenografts in immunocompromised mice and reduces the adverse effects of chemotherapy. Our findings suggest that PTE could be a safe and effective treatment for breast cancer. Most importantly, as a supplement to chemotherapeutic regimens, it could enhance anti-cancer effects and reduce chemo-related toxicity.

ABSTRACT P03

DEVELOPING A NOVEL CELL-BASED CANCER THERAPY EXPRESSING POTENT TRAIL FUSION PROTEINS

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Cell-based therapies represent a relatively new approach to treating cancer. Healthy cells can be genetically engineered *ex vivo* to produce powerful anticancer proteins prior to reinfusion into a patient.

The tumour necrosis factor apoptosis-inducing ligand (TRAIL) is proapoptotic protein that has been shown to specifically kill cancer cells. However, systemically administered soluble TRAIL (sTRAIL) failed to show significant clinical benefit because the sTRAIL complexes degraded into inactive

subunits. Previous studies show that cell-based therapies expressing sTRAIL require assistance of common chemotherapeutic drugs (e.g. doxycycline, mitoxantrone, or 5-fluorouracil), increasing risk for off-target effects.
Recently, more stable and potent single chain TRAIL (scTRAIL) and fragment crystallizable scTRAIL (fc-scTRAIL) fusion proteins have been developed. Here we explored the ability to genetically engineer HEK293T cells with these novel TRAIL fusion proteins and the natural, membrane bound TRAIL (memTRAIL). We used bioluminescence imaging (BLI) to monitor the cancer killing activity of these therapeutic cells when co-cultured with firefly luciferase expressing breast (MDA-MD-231), prostate (PC3MLN4), and ovarian (OVCAR-8) cancer cells.
Our BLI shows that HEK293T expressing fc-scTRAIL were superior at killing PC3MLN4. However, the HEK293T expressing memTRAIL were superior at killing OVCAR-8. HEK293T expressing scTRAIL or fc-scTRAIL were highly resisted by OVCAR-8.
Future work will involve studying the mechanism of resistance in OVCAR-8, the cancer cell-specific trend of sensitivity to different TRAILs, and the effects of tumour homing immune cells expressing these TRAILs on preclinical models of cancer metastasis. Continued work may yield a new and more potent cell-based therapy for the treatment of metastatic disease.

ABSTRACT P04

EXPLORING THE ROLE OF NKR-P1B:CLR-B INTERACTION IN MOUSE MAMMARY TUMOUR IMMUNOSURVEILLANCE

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Natural killer (NK) cells are large, granular, and cytotoxic innate lymphocytes which do not require prior antigenic exposure to target cancerous and virally infected cells. NK cells possess activating and inhibitory receptors which may activate or inhibit NK cell activity, respectively. In mice, the inhibitory NKR-P1B receptor on NK cells recognizes the C-type lectin-related protein-b (Clr-b) ligand expressed on most autologous cells but downregulated on many tumour cell lines. In B cell lymphoma models, the disruption of NKR-P1B:Clr-b interaction results in delayed tumour development and progression, suggesting that blockage of inhibitory signals from NKR-P1B augments NK cell activity against lymphoma cells. NKR-P1B is the closest homolog of human NKR-P1A receptor on NK cells, therefore, the mouse NKR-P1B:Clr-b system is a relevant model for the human NKR-P1A:LLT1 system in cancer immunosurveillance. The goal of our research is to evaluate the role of NKR-P1B:Clr-b interaction in a solid tumour model, namely, breast cancer. To study this inhibitory axis in vitro, we are performing cytotoxicity assays by evaluating the killing activity of NKR-P1B-deficient and wild-type (WT) NK cells against Clr-b-deficient and WT target cells (E0771 breast cancer cell line). In vivo, we are using a E0771 cell-induced mammary tumour model and a spontaneous tumour model to evaluate mammary tumour growth, and the activity of tumour-infiltrating NK cells in NKR-P1B-deficient and WT mice. These studies will reveal the role of NKR-P1B:Clr-b interactions in NK cell-mediated immunosurveillance of mammary tumours.

ABSTRACT P05

SKP2-CYCLIN A INTERACTION IS NECESSARY FOR MITOTIC ENTRY AND MAINTENANCE OF DIPLOID

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Skp2, the substrate recognition component of the SCF ubiquitin ligase complex, has been implicated in the targeted destruction of a number of key cell cycle regulators and the promotion of S-phase. One of its critical targets is the Cyclin dependent kinase (Cdk) inhibitor p27 (Dacapo or Dap in flies), and indeed the overexpression of Skp2 in a number of cancers is directly correlated with the premature degradation of p27. Skp2 was first identified as a protein that interacts with Cyclin A in transformed cells, but its role in this complex has remained unclear. Here we demonstrate that Skp2 interacts directly with Cyclin A in Drosophila and we provide evidence that this interaction is required in vivo to maintain Cyclin A levels and permit mitotic entry. Failure of mitotic entry in Skp2 mutant cells results in polyploidy. This mitotic function for Skp2 is distinct from its role in targeting p27 for destruction to promote S-phase

ABSTRACT P06

EXPERIENTIAL LEARNING IS THE KEY TO DEVELOPING TRANSFERABLE SKILLS IN CANCER RESEARCH AND EDUCATION

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Although cancer is the leading cause of death in Canada, many Canadians are unaware that cancer is a diverse set of diseases that require different diagnostic tools and treatment strategies. Thus, education focusing on cancer research is critical to advance current public conversations around the disease. It is hypothesized that university students immersed in cancer research and education through experiential learning become effective community educators. The Windsor Cancer Research Group (WCRG) offers many experiential learning opportunities for undergraduate and graduate students by connecting them to local researchers, health care professionals, patients and caretakers. In order to gain more insight into

an empirical measure of the training program's success and needs for improvement, a survey was developed to assess both the transferable skills acquired by student participants of WCRG's experiential learning opportunities. The results from this study show that these experiences enrich the training of students, reinforces their interest in biomedical careers, encourages them to be community educators, and builds self-efficacy. Student involvement in creating accessible platforms embedded with educational tools for the public, and its impact on patient care, also addresses a clear social need for proper communication about cancer research and the promotion of healthy and safe communities.

ABSTRACT P07

CRYSTAL STRUCTURE AND ACTIVITY-BASED LABELING REVEAL THE MECHANISMS FOR LINKAGE-SPECIFIC SUBSTRATE RECOGNITION BY DEUBIQUITINASE USP9X

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Ubiquitination is an important post-translational modification that regulates almost every aspect of cellular functions. Chains of different topology each have a distinct role in dictating the function and fate of the modified proteins. Deubiquitinases (DUBs) reverse ubiquitination and how they achieve chain and substrate specificity is a topic of immense interest owing to their therapeutic potentials. We obtained the atomic details of the USP9X catalytic core, a DUB involved in cancers and developmental disorders. The structure revealed its unusual mechanisms of action using a set of activity-based ubiquitin probes. These novel probes will propel future investigation of how DUBs recognize and process ubiquitin chains and identify potential new sites on DUBs for drug discovery.

ABSTRACT P08

ANAL CANAL CANCER TREATMENT OUTCOME IN WINDSOR, ONTARIO: A SINGLE INSTITUTION EXPERIENCE

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Purpose: Anal canal cancer (AC) is a rare malignancy that often carries poor prognosis. Standard treatment of locally advanced AC has been concurrent chemotherapy and radiation therapy for several decades. In recent years, volumetric-modulated arc therapy (VMAT) has emerged as a new technology designed to reduce toxicities. This study reviews our single institution experience in 18 years to see if this innovative treatment has translated into a better outcome for AC patients.

Methods: We collected consecutive data on 182 AC patients who visited Windsor Regional Hospital (WRH) cancer program between 2002 and 2019. Kaplan-Meier survival curves were generated to compare patients that received VMAT versus conventional radiation therapy (CRT).

Results: Median age of our cohort was 61 years (33-98), 36% were male, 4% were HIV positive, 62% were squamous cell carcinoma, 86% received radiotherapy, including 45% VMAT and 40% CRT, 74% received 45 Gy or higher dose, 49% had surgery, and 72% had chemotherapy. The 3/5 year overall survival (OS) are 60%/47%, and the 3/5 year local control (LC) are 73%/ 67%, respectively. VMAT had a higher 3/5 year OS of 68%/49%, versus CRT at 54%/45%, p=0.0494 and 0.3445, respectively. The 3/5 year LC was not significantly different in the two groups, 69%/60% versus 74%/71% (p=05893. and 0.1504). There was no treatment related death and 20% had Grade 3-4 acute toxicity.

Conclusions: AC treatment outcome in our institution is comparable to published historical data. Our findings support the use of VMAT for localized AC with better OS and similar LC.

Keywords: Anal canal cancer, Volumetric-modulated arc therapy, Radiation therapy

ABSTRACT P09

DECIPHERING TARGETABLE PATHWAYS THROUGH NOVEL ARTIFICIAL INTELLIGENCE ALGORITHMS FOR THE TREATMENT OF RARE TUMOURS

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Current precision oncology approaches primarily use gene panels in which genes are deemed pathogenic based on association with cancer-causing pathways. However, this approach has limitations because genetic variations need to be correlated with downstream RNA expression patterns to accurately identify genes actively involved in cancer growth. An emerging field in molecular oncology involves the application of artificial intelligence (AI) to mine genomic and transcriptomic datasets pertaining to a particular tumour and identify the most likely aberrant pathways to target.

We will conduct a prospective phase II observational study to determine whether multiomics-derived cancer therapies improve patient outcomes relative to current standards of care. We will recruit 15 patients with relapsed, refractory solid tumours for whom effective therapeutic options do not exist. Biopsy specimens at the time of diagnosis and/or relapse will be processed to isolate genomic DNA and RNA. Whole-exome sequencing and RNA sequencing will be performed and subjected to AI-derived multiomics algorithms to identify targetable molecular pathways and provide therapeutic options specific to each patient's tumour type. We will also perform parallel literature reviews to evaluate the AI-generated justification of targetable pathways playing a causal role in promoting cancer growth. Impacts of multiomics-derived therapy will be measured using both primary (e.g. time to tumour progression) and secondary (e.g. number of clinic visits) endpoints.

This study will help determine whether current AI-derived personalized cancer therapy options are a superior alternative to existing standards of care, and will help guide future research into the incorporation of AI into day-to-day healthcare delivery.

ABSTRACT P10

SIMULATED CANCER MUTATIONAL SIGNATURES TO CLASSIFY CANCER TYPES USING AN ALIGNMENT-FREE MACHINE LEARNING-BASED APPROACH

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Somatic mutagenesis in cancer is a phenomenon that perturbs genome sequence organization given the mutational signatures that characterize global non-random biases in sequence composition arising from mutagen exposures, mutational mechanisms, and faulty DNA repair. Genomic signatures can be used for classification purposes with demonstrated sensitivity to the level of individual species. Early classification of cancer types using mutational signatures in cancer genomes is a novel intraspecific application of the ML-DSP, an ultra-fast, alignment-free, approach that uses Machine Learning with Digital Signal Processing for sequence dissimilarity analysis. Here, we present SomaticSimu, a novel tool to reconstruct somatic mutation signatures in silico within human chromosome 22 (50 Mb). One hundred sequences (n=1300) of thirteen different cancer subtypes of bone, breast, central nervous system (CNS), and myeloid cancer with different mutational signatures were simulated based on the mutation catalogues from the 2020 Pan-Cancer Analysis of Whole Genomes. SomaticSimu simulates single and double base-pair substitutions, and single base pair insertions and deletions with biologically representative signature combinations, mutation types, and total mutation burden in a directed evolution approach. SomaticSimu generated sequences with imposed mutational signatures were used as a training dataset to calibrate ML-DSP classification of the cancer subtypes. This achieved up to 100% classification of cancer vs. noncancer genomic signatures. ML-DSP achieves high accuracy in classification within subtypes of Bone (95.5%), Breast (82%), Central Nervous System (98%), and Myeloid (64.2%) cancer sequences. SomaticSimu facilitates the detection of cancer using machine learning classification and identification of biomarkers within the landscape of cancer mutational signatures.

ABSTRACT P11

THE TUMOUR SUPPRESSOR TUBERIN ACTS AS A GATEKEEPER OF CELL PROLIFERATION AND SURVIVAL.

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**The students contributed equally to the preparation of the pathways and the poster.*

The tumour suppressor Tuberin together with its partner Hamartin forms the Tuberous Sclerosis Complex (TSC). This complex is responsible for the control of cell growth and cell proliferation. Disruption of the actions of the TSC complex, by mutations in one of the genes (TSC1/TSC2), carries severe consequences for the cell metabolism causing proliferative diseases, such as Tuberous Sclerosis and cancers.

Tuberin is the regulatory protein of the TSC complex. It is a large protein (200KDa), containing many Kinases' phosphorylation sites that are phosphorylated according to nutrient and growth factor availability. Once phosphorylated, Tuberin can interact with and modify other proteins,

such as Rheb and Cyclin B1, and regulate mTORC1 (cell growth) and the cell cycle (cell proliferation) respectively. During times of ample nutrition, Tuberin is inhibited by growth factor signaling, including direct phosphorylation by Akt, allowing for activation of mTOR and subsequent protein synthesis. In the presence of high serum, and/or Akt signaling, direct binding between Tuberin and the G2/M cyclin, Cyclin B1, is stabilized and the rate of mitotic entry is decreased.
Our lab has identified the Tuberin/CycB1 complex as an important cell cycle checkpoint linking mitotic onset with the nutritional status of the cell to control cell growth. Tuberin's primary structure is widely conserved between species, from Drosophila to mammals, highlighting the importance of this protein for the survival of multiple different species.
Tuberin is not only responsible for the control of cell growth, but it is also part of multiple cellular pathways, including DNA damage-inducing to apoptosis, autophagy, cell adhesion, and cell migration.
Our poster summarizes the cellular pathways regulated by Tuberin, hoping to clarify the importance of this tumour suppressor protein for proper cell survival and the prevention of proliferative diseases.
The pathways depicted here were created by the Tuberin group undergraduate and graduate students during the COVID-19 pandemic in 2020.

ABSTRACT P12

APPLYING WOMEN'S KNOWLEDGE TO BREAST CANCER PREVENTION: INCLUDING WOMEN AS PARTNERS IN COMMUNITY HEALTH TO CLOSE GAPS IN BREAST CANCER RISK KNOWLEDGE

Jane McArthur[1*]
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Increased understanding between medical science and social science could improve breast cancer prevention strategies, particularly where risks are related to involuntary, environmental exposures. The way women understand breast cancer risk is dynamic, contextualized, multisectoral, and relational and offers insights into spaces of understanding. Common themes emerged in studying women's understandings of breast cancer risks: information discrepancies, distrust, and experience as knowledge. These concepts, as described in women's narratives, reflect the sociocultural contexts in which the women live, including their workplaces, the health care systems and broader institutional and governing structures. Analysis of the women's stories exposes the interrelationship between the knowledge used to construct their breast cancer risk understandings and their living and working environments. Breast cancer risk, as seen from women's standpoint, is not solely a biomedical phenomenon residing in the body, determined by genetics or lifestyle choices, but is experienced by women in a nested set of social, cultural and political relationships. Dominant biomedical narratives obscure the experiences of the women, including the spaces they identify breast cancer risks. The women in the study transcend the implication that they are personally responsible for controlling their risks for breast cancer at work and elsewhere, pointing to external, environmental and structural factors seemingly outside their control. Women construct strategies for addressing uncertainty created by the myriad risks for breast cancer beyond those that are modifiable. The research suggests we should see women as knowledge producers and interpreters and include them as partners in community health and breast cancer prevention strategies.

ABSTRACT P13

CHARACTERIZING THE ANTI-CANCER EFFICACY OF PARADISE TREE EXTRACT ON HUMAN MELANOMA CELLS

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Skin cancers such as melanoma are the most common form of cancer in the world. When treating patients, surgical resection and chemotherapy are two options traditionally used. However, surgery has shown success only in early-stage melanoma and current chemotherapeutics have limited efficacy and high non-specific toxicity. Thus, there is a need to discover novel complementary therapies for use alongside standard chemotherapeutics. Natural extracts have been scientifically validated for anti-cancer efficacy in the past. Paradise Tree Extract (PTE) from Simarouba glauca or "Lakshmi Taru" is one such extract that has been shown to possess anti-cancer activity. In previous studies, it has been evaluated to show anti-cancer potential on leukaemic cancer cell lines. However, it has not been investigated for use to treat human melanoma. Thus, for the first time, we characterized PTE efficacy in human melanoma cell lines A375 and G361 and its selective induction of apoptosis. In addition, the cellular mechanisms behind its anti-cancer activity were elucidated. Furthermore, the interaction of PTE with standard chemotherapeutics was evaluated, specifically involving paclitaxel and dacarbazine, the latter being the only approved single-agent chemotherapeutic for melanoma. The results found indicate there is no negative interaction with either drug and instead, a slight enhancement

of anti-cancer activity. Overall, the results of this study indicate that PTE has the potential to be used as a selective and efficacious melanoma treatment either alone or in combination with current standards of care.

ABSTRACT P14

DESIGN A PEPTIDE DRUG FOR RHEUMATOID ARTHRITIS IN CANCER PATIENTS

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Individuals with Autoimmune Diseases such as Rheumatoid Arthritis (RA) experience a slightly increased risk for developing certain types of cancers, including hematological disorders and some solid tumors. RA is an autoimmune disease, caused by improper recognition of self-peptides, particularly human cartilage glycoprotein and type II collagen, by specific human leukocyte antigen (HLA) receptors. Normally T-cell specific for these peptides are destroyed in the thymus before they are released, preventing autoimmunity. However, certain post-translational modifications, especially citrullination, can lead to "self-peptide" recognition by non-self T cells: in the case of RA, one HLA protein, out of about 1700 possible ones, is responsible for 65% of RA cases. If this protein could be blocked, drugs could be developed that interrupt the disease at its root cause without affecting the rest of the immune system; this is the focus of research in the Trant Lab. This presentation will briefly overview the approach, including the drug design, and will focus on the molecular biology work accomplished to date.

ABSTRACT P15

ANTI-CANCER EFFECTS OF LONG PEPPER AND ROSEMARY IN COMBINATION TREATMENTS ON COLORECTAL CANCER CELLS

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The second most commonly diagnosed cancer in Canada is colorectal cancer (CRC), and the most common chemotherapeutic for this cancer is FOLFOX. Although great advances have been made for early diagnosis and surgical tumor removal, advanced stages of cancer are extremely difficult to treat. The only option that remains is chemotherapy, which causes severe side-effects due to its non-selective nature of affecting non-cancerous cells. Natural health products (NHPs) have been studied over the centuries and offer a promising supplement to the cancer treatments used today. NHPs contain various bioactive compounds that not only exhibit anti-cancer properties but also induce health benefits, including reduced inflammation and improved digestion. The focus of this project is to investigate the ability of Long Pepper Extract (LPE) and Rosemary Extract (RE) to induce cell death in two colon cancer cell lines, HCT116 and HT29, without harming a healthy colon cell line, NCM460. Initial results have indicated that these extracts induce apoptosis in colon cancer cells at low doses. We will conduct separate experiments to determine the dose- and time-dependent action of LPE and RE on all three cell lines, followed by combination treatments to assess their potential additive anti-cancer effects. Our next aim would be to investigate the interaction of LPE and RE with FOLFOX for colon cancer treatment. The mechanism of induction of apoptosis by LPE and RE in cancer cells will be investigated and discussed. If successful, these findings can lead to the development of supplemental treatment for colorectal cancer patients.

ABSTRACT P16

MODELING AND CHARACTERIZING SYNCHRONOUS BILATERAL BREAST CANCER METASTASIS IN MICE USING DUAL-BIOLUMINESCENCE IMAGING

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Synchronous bilateral breast cancer (SBBC) is the development of malignant lesions in both breasts at the time of diagnosis or within 12 months. It occurs in approximately 2% of breast cancer patients, and compared to women with unilateral breast cancer, SSBC patients have higher rates of metastasis and lower overall survival. However, little is known about the metastatic properties of cells from each tumour, which can directly contribute to patient outcome. Here we developed the first mouse model of SBBC by orthotopically implanting human breast cancer cells into bilateral mammary fat pads, and we used non-invasive imaging to study the biodistribution of metastases from each tumour. To track cells from each tumor, we engineered them to express two recently developed and highly sensitive BLI reporters called Akaluc or Antares2 and performed

dual-bioluminescence imaging (dual-BLI) for 6 weeks after implantation (n=10 mice). Cells were also co-engineered with either the fluorescence reporter tdTomato or zsGreen for endpoint fluorescence microscopy. Dual-BLI showed lung metastasis from both primary tumors in nine of 10 mice as early as day 21, and BLI signal from metastases continued to increase over time. Unexpectedly, microscopy revealed that a large percentage of metastases were composed of a mixture of both tdTomato and zsGreen cells from both primary tumors. Our SBBC model suggests metastatic cross-seeding is a common occurrence, which may contribute to tumour heterogeneity and treatment resistance. These unexpected findings may explain the worse outcome of SBBC patients and should provide future insight on patient treatment and management.

ABSTRACT P17

HIGHLY METASTATIC CLAUDIN-LOW MAMMARY CANCERS CAN ORIGINATE FROM LUMINAL EPITHELIAL CELLS

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Claudin-low mammary cancer is a highly aggressive subtype of triple-negative breast cancer (TNBC). The prominent stem cell characteristics and a high metastatic potential of claudin-low mammary cancers confers additional challenges to the already difficult-to-treat TNBCs. However, despite the poor outcome for patients with this subtype, relatively little is known about its cell-of-origin or oncogenic drivers. One major reason for this stagnation in progress was the lack of suitable models that could recapitulate disease initiation and progression. Thus, our laboratory developed a novel transgenic mouse model that allowed us to show that persistent oncogenic RAS signaling leads to the development highly metastatic triple-negative mammary tumors that fall within the claudin-low subtype. Since these tumors arise spontaneously from the mouse mammary gland epithelium, we were able to trace the origin of claudin-low mammary tumors to the luminal epithelial compartment and show that continuous oncogenic RAS signaling is capable of driving transdifferentiation from a luminal into a mesenchymal state. Ablation of the driver oncogene in claudin-low mammary cancer cells revealed that RAS cooperates with other molecular mechanisms to maintain their mesenchymal-like differentiation state. Not only did we identify that claudin-low mammary cancers can arise from luminal cells through oncogenic RAS signaling, our mouse model will also serve as an important model for future studies to identify metastatic drivers, cancer-dormancy factors, as well as immunological components that are important to identify in the fight against triple-negative, claudin-low mammary cancers.

ABSTRACT P18

MAGNETIC RESONANCE IMAGING DATA ACQUISITION ACCELERATION AND FEATURE DETECTION WITH DICTIONARY LEARNING

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Magnetic Resonance Imaging (MRI) is a powerful imaging modality with excellent soft tissue contrast. Contrast agent can be used to “tag” individual cells. Time-lapse MRI can be used to track the motion of tagged cells, providing insights in the studies of inflammatory diseases and metastasis of cancer. However, current methods are not capable of detecting cells traveling faster than 1µm/s due to temporal blurring. In addition, the manual cell counting is a cumbersome task. In this work, a dictionary learning based technique has been developed to accelerate the MRI data acquisition and aid in the task of locating cells. Dictionary learning is a machine learning technique, in which features of an image can be ‘learned’ as atoms. Images can be represented using a sparse combination of atoms. This provides a constraint in non-linear image reconstruction with data sampled below the Nyquist criteria. The locations of features can be obtained from the atom coefficients. This technique will significantly increase the MRI temporal resolution and improve the cell detection process.

ABSTRACT P19

KNOCKOUT OF THE METABOLIC STRESS MEDIATOR LKB1 OR ITS SUBSTRATE NUAK1 ENHANCES NF-KB SIGNALLING IN A SPHEROID MODEL OF OVARIAN CANCER METASTASIS

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Most epithelial ovarian cancer (EOC) patients are diagnosed at an advanced stage, and despite surgery and chemotherapy, relapse with chemo-resistant disease. Thus, improving outcomes requires new ways of limiting metastasis. Liver Kinase B1 (LKB1), encoded by STK11, regulates intracellular metabolism in response to stress. We established that LKB1 is required for EOC spheroid viability in vitro and efficient metastasis in orthotopic mouse models. We also showed that the pro-metastatic functions of LKB1 are likely AMPK-independent, and the AMPK-related kinase NUAK1 is a key LKB1 target in EOC with pro-metastatic function. Here, we sought to identify pathways altered in EOC cells by loss of LKB1 or NUAK1. Knockout of STK11 was performed in OVCAR8 and iOvCa147 cells using CRISPR/Cas9 genome editing; knockout of NUAK1 was performed in OVCAR8 cells.

Microarray analysis revealed an increase in NF-κB- and inflammation-associated transcriptional signatures in EOC spheroids, which was enhanced by loss of LKB1 or NUAK1; qRT-PCR confirmed elevated expression of representative NF-kB target genes. Immunoblot analysis revealed that p-RelA (Ser536) and nuclear abundance of the related transcription factors RelB and p52 were increased in EOC cells lacking LKB1 or NUAK1. Notably, EOC cells lacking NUAK1 exhibited increased spheroid viability, which was abrogated by pharmacological inhibition of NF-κB.

Here, we present evidence that NF-κB signaling is induced in EOC spheroids, and that loss of LKB1 or NUAK1 further elevates NF-κB activity. We postulate that inflammatory signaling in EOC cells and spheroids is a stress response that modulates cell viability during EOC metastasis.

ABSTRACT P20

PREDICTING BIOLOGICALLY IMPORTANT RESIDUES USING IN SILICO MUTAGENESIS IN THE TUBEROUS SCLEROSIS COMPLEX

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In many genetic diseases, such as cancer, clinical mutations are identified early in the study of the disease and the resultant phenotype is characterized by patient symptoms and survivorship. However, the underlying molecular mechanism of these mutations' effects are rarely studied and understood. In this study, we use a bi-directional approach to address this concern. We created clinically identified mutations in the TSC2 gene, encoding the protein Tuberin, which is mutated in Tuberous Sclerosis; a disease known to cause the aberrant cellular growth, forming tumours named hamartomas. Tuberin binds to the G2/M cyclin, Cyclin B1. This complex regulates cell proliferation and cell size. We examined the ability of these clinical mutants to form a complex with Cyclin B1 and control cell division and cell proliferation. Using in silico mutagenesis, we determined if there is any structural modification in the Tuberin/Cyclin B1 complex. Conversely, computer-derived mutations were identified that would show a reduction in Cyclin B1-Tuberin binding, one of those mutations was successfully cloned and tested using HEK293 cells to correlate the phenotype obtained with the efficacy of the computer modelling system. Our research provides a new method of testing molecular outputs of clinically identified mutants using an in silico approach, which would allow for faster identification of molecular mechanisms behind clinical phenotypes and the design of drugs to use in the treatment of diseases involving cell growth and proliferation, as Tuberous Sclerosis and cancers.

ABSTRACT P21

A DUAL PET/MRI REPORTER GENE SYSTEM FOR CELL TRACKING

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Various molecular imaging tools and labelling techniques have been developed for preclinical and clinical cell-tracking. However, there is still a need for safer, more sensitive and reliable methods to track emergent cellular therapies. Multi-modal imaging is a common technique that

harnesses the benefits of more than one imaging tool to answer questions on cell viability, functionality, biodistribution and persistence of transplanted therapeutic cells. For this work, we have developed a dual PET and MRI reporter gene system that uses non-immunogenic human-derived genes which can uptake clinically used probes to allow detection with two clinically relevant modalities. PET enables extremely sensitive, highly quantitative information while MRI provides fine spatial resolution with soft tissue differentiation and the ability for longitudinal monitoring due to its lack of ionizing radiation. We have engineered and evaluated functionality of cells expressing a human-derived (1) Organic Anion Transporter Polypeptide 1B3 (OATP1B3) which can uptake the MRI contrast agent Gd-EOB-DTPA, commonly known as Primovist and (2) a sodium Iodide Symporter (NIS) which can uptake the PET tracer, 18F-tetrafluoraborate (18F-TFB). In-vitro incubation with Primovist showed 45% higher Primovist uptake than in non-OATP1B3 expressing cells. Significant (p<0.001) 18F-TFB uptake (45-fold) in NIS-expressing cells compared to non-NIS expressing cells was observed. Similarly, in-vivo results showed positive contrast enhancement in T1-weighted MRI images in OATP1B3-expressing cells and higher SUV values in NIS-expressing cells in comparison to non-engineered cells. This cell system has the ability to track cells and retrieve invaluable information in a clinically relevant, non-invasive, safe and sensitive manner.

ABSTRACT P22

The Role of PROLACTIN in Tumor Progression and Tumor Microenvironment in Response to DNA Damaging Agents

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Prolactin (PRL) acts as a survival factor for breast cancer cells, however the in vitro and in vivo mechanisms are unknown. In our in vitro studies we demonstrated that PRL increased the viability of breast cancer cells to DNA-damaging chemotherapeutics and ATM contributes to the PRL-JAK2-STAT5-HSP90 pathway in mediating cellular resistance to DNA-damaging agents. In order to investigate the role of autocrine PRL in tumor progression in response to DNA damage, we used a novel model of breast cancer recurrence. Breast cancer cells were treated with DNA damaging agent doxorubicin in the cell culture, in the presence or absence of PRL, and the cells were injected into mammary fat pad of immune deficient SCID mice. Interestingly, in orthotopic xenograft studies, autocrine prolactin from human breast cancer cells increased the tumor latency of doxorubicin induced DNA damaged cells in SCID mice compared to untreated or prolactin or doxorubicin alone. We hypothesize that this is in part due to the cross-talk of the prolactin and DNA damage response pathways that may be affecting the tumor microenvironment. In order to test our hypothesis, we injected mice with Anti-asialo GM1 antibody and observed that the depletion of Anti-asialo GM1 target immune cells caused a trend of early tumor formation of autocrine prolactin secreting human breast cancer cells treated or not treated with doxorubicin. Overall, our study provides a unique experimental approach to understand the role of PRL in tumor progression and tumor microenvironment in a cross-talk with DNA damage repair pathway.

ABSTRACT P23

UNDERSTANDING THE PATHWAYS ASSOCIATED WITH PROLACTIN SIGNALING IN BONE METASTATIC BREAST CANCER CELLS

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Background: Breast cancer is one of the most diagnosed among women. It is associated with osteolytic bone metastasis. Our group demonstrated that high prolactin (PRL) receptor levels in the primary tumour shorten the time to the development of bone metastases in breast cancer patients. To understand the mechanism underlying this behaviour, PRL-induced molecules were identified in the conditioned media from the breast cancer cell lines SKBR3 and MCF7, using an array of human cytokines. Our objectives are to use bioinformatics tools to identify the main pathways in which these molecules are involved, and to standardize an in vivo model to evaluate their role in bone metastases associated to breast tumors that secrete PRL. Methods: The PRL-induced cytokines were analyzed using the Gene2Function tool in the FUMA GWAS platform. Additionally, our group derived luciferase-2 and green fluorescence protein-expressing MCF7 cells that were selected by their ability to grow in the bone of immunodeficient mice and engineered them to secrete human PRL (MCF7-LGB-hPRL) or to carry the empty vector (MCF7-LGB-EV). We analyzed in vitro the luciferase activity of these cells, the differences in proliferation, and their ability to secrete PRL. The cells were injected intratibial in nude mice; the tumor growth was evaluated by bioluminescence and the lesions in the bones were detected using microcomputed tomography

(micro CT). Results and Discussion: The analysis of the gene set in the FUMA GWAS platform confirmed that several of the gene products are detectable in the blood. Some of them are associated with angiogenesis, STAT5 signaling, epithelial to mesenchymal transition and cell motility. MCF7-LGB-hPRL and MCF7-LGBEV cells have luciferase activity, which increases proportionally to the number of cells. Only MCF7-LGB-hPRL cells release PRL. Using an Alamar Blue assay, we observed that MCF7-LGB-hPRL cells proliferate more than MCF7-LGB-EV; this suggests that autocrine/paracrine PRL signaling stimulates cell proliferation in this cell line. Six days after the injection of MCF7-LGB-hPRL or MCF7-LGB-EV cells, all the mice had detectable tumors. At day 120, the mice were sacrificed, and the tibias were analyzed by micro CT. In those that received MCF7-LGB-hPRL cells, the bone loss was higher than in those that received MCF7-LGB-EV cells. Conclusions: The PRL-induced molecules are involved in pathways that could explain the role of PRL in bone metastasis. Our in vivo model is suitable to distinguish the effect of PRL in breast cancer associated bone loss; it can be manipulated in order to evaluate the PRL induced molecules contribution to bone loss. These studies can be the basis for the identification of molecules that could be used to predict the risk to develop breast cancer-associated bone metastases, or as therapeutic targets to avoid or treat them.

ABSTRACT P24

A RETROSPECTIVE SINGLE CENTER STUDY INVESTIGATING THE CLINICAL SIGNIFICANCE OF GRADE IN TRIPLE NEGATIVE BREAST CANCER.

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Purpose: To investigate the predictive value of histological grade in triple negative breast cancer (TNBC).
Methods: We retrospectively analyzed 305 TNBC patient charts from 2004-2017 at Windsor Regional Cancer Center with triple negative defined as estrogen (ER), progesterone (PR), and HER-2 negative. The significance of grade with respect to demographic and treatment variables as well as patient outcomes was determined.
Results: Eighty-two percent of the patients with TNBC presented with grade 3 tumors. There were found to be 10, 45, and 250 patients with tumor grades 1,2,3, respectively. On univariate analysis, grade 1 patients were less likely to receive chemotherapy (P=0.008). The overall survival rates were 90.12%, 64.4%, and 77.2%, for patients with grade 1, 2 and 3 tumors respectively.
Grade 3 tumors had significantly better relapse-free survival than patients with grade 2 tumors.
Overall relapse rates were 70%, 55.6%, and 75.6%, respectively for patients with tumor grades 1,2, and 3 (P=0.04). The overall difference in survival among the three groups was statistically significant (P= 0.019). Comparing between grade 2 and grade 3, we determined that patients with grade 2 tumors had a 5.5-fold increased risk of death (HR = 5.513; 95% CI 1.2-25.6) and shorter time to relapse (HR=1.9; 95% CI 1.1-3.2) after five years from time of diagnosis.
Conclusion: Grade was shown to have positive predictive value in determining relapse. Grade 2 patients showed poorest disease-free survival and faster time to relapse after the 5-year mark hence we recommend stratifying TNBC patients by grade in future clinical trials.

ABSTRACT P25 **not participating in poster session*

MEASLES REACTIVITY IN WINDSOR PATIENTS POST AUTOLOGOUS VERSUS ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION: A RETROSPECTIVE REVIEW

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As the antivaccine movement has increased, vaccine preventable illnesses have also increased. The resurgence of measles globally has been noted, specifically the United States received 1200 new cases of measles in 2019, which is the highest number of cases since 1994. Standard of care for patients post hematopoietic cell transplant (HCT) includes repeating all childhood vaccinations. However, compliance with recommendations is unknown. A retrospective chart review investigated HCT patients between 2000-2019 from the Windsor Regional Hospital (WRH) cancer centre. Patients were excluded from data collection if they were deceased before an MMR reactivity test could be done or if they were lost to follow up. A total of 83% of autologous HCT (N=57) and 66% of allogeneic HCT (N=47) patients had serology tested to measure MMR reactivity post-transplant prior to live vaccinations. Overall, allogeneic HCT patients had more measles, mumps, and rubella reactivity than autologous HCT patients (Table 1). All patients not reactive to measles need to be re-vaccinated 24 months after treatment according to 2019 American Society for Transplantation and Cellular Therapy guidelines. The majority (66%) of allogeneic HCT patients had a myeloid malignancy, while 70% of autologous patients has a

diagnosis of multiple myeloma. Demonstrated by the poor MMR response, multiple myeloma patients may account for this evidence of less robust immune system. This does emphasize the need for MMR vaccinations post HCT for multiple myeloma patients and raises the question regarding immunization for at risk non-transplant eligible patients.

ABSTRACT P26

PSYCHOSOCIAL AND FINANCIAL BURDEN OF CANCER DIAGNOSES IN MULTIPLE MEMBERS OF THE SAME FAMILY IN WINDSOR, CANADA

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Background: A cancer diagnosis causes significant financial and psychosocial burden. However, little research has been done to elucidate the additional burden in families with multiple members diagnosed with cancer. The objective of the current study is to assess the psychosocial and financial burden of multiple cancer diagnoses within a family.

Methods: Participants were recruited to the study by oncologists at Windsor Regional Hospital (WRH) if they had a first degree family member with a cancer diagnosis or treatment within five years of their own. Telephone interviews were conducted using a structured questionnaire addressing perceived psychosocial and financial burden of concurrent cancer diagnosis and/or treatment. Quantitative analysis utilized Likert scales to assess financial and psychosocial burden. Qualitative analysis was completed by identifying common themes from patient interviews.

Results: The analysis included 9 participants. Financial burden was endorsed by 44% of participants, 75% of which did not use financial aid services. There was a significant increase in participant stress levels related to their family member's diagnosis compared to their own. Themes extracted from interviews included patients acting as caregivers to each other, unique stressors related to cancer diagnosis and treatment in multiple family members, positive perspectives and attitudes, and improving and continuing valuable support services.

Conclusions: Cancer patients with a first degree family member also diagnosed with cancer experience unique stressors, but under utilize support services. It is important for healthcare providers caring for this patient population to deliver compassionate care, and to emphasize and facilitate access to all resources available.

ABSTRACT P27

HOW FAR AWAY IS THE FIRST BORON NEUTRON CAPTURE THERAPY CENTRE IN CANADA

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Introduction: Boron Neutron Capture Therapy (BNCT) offers an approach to overcoming radiation resistance in cancers and potential cure after maximal conventional therapies. Low cost Accelerator-based BNCT (AB-BNCT) are needed to expand the research. We conducted a literature review to assess current status of BNCT technique around the world with emphasis on feasibility of BNCT using compact accelerator-based neutron source (CANS).

Prerequisites for building BNCT centre: We have established cell lines and animal models to test Boron agents and then radiobiology of BNCT in preclinical trials. We will evaluate in vitro and in vivo tumor response to BNCT, and neutron dosimetry before starting clinical trials for suitable cancer patients. Positron emission tomography (PET) will help develop routine Boron concentration measurement protocol, using 18F-BPA.

Facility design and treatment planning system (TPS): At present, there are 14 AB-BNCT facilities in 8 countries. There are at least four companies marketing complete systems of AB-BNCT, i.e. Neutron Therapeutics (NT), Sumitomo Heavy Industries (SHI), TAE Life Sciences, and Neuboron Medtech Ltd. SHI's NeuCure is the first one to obtain approval to start routine BNCT in March 2020. NT has constructed nuBeam in Finland and Japan. Both are supported by TPS from RayStation.

Funding: A New Frontiers in Research Fund Transformation Grant up to $24 million is being applied to make CANS available within the next 2 to 4 years (NFRFT-2020-00021).

Conclusions: There is great support from multiple institutions and universities across Canada. It is feasible to build the first BNCT Centre in Canada using CANS.

ABSTRACT P28

PATTERN OF TREATMENT INITIATION AND OUTCOMES FOR PATIENTS WITH METASTATIC NON-SMALL CELL LUNG CANCER IN ONTARIO

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Introduction: The impact of diagnosis and treatment delay on outcomes in advanced non-small cell carcinoma (NSCLC) is not well understood. In this study, we examined the effect of the length of time to the first chemotherapy treatment initiation and the other factors affecting the survival of the patients.

Methods: This retrospective study used data from the Institute of Clinical Evaluative Sciences (ICES) and identified 4520 patients in Ontario who were diagnosed with stage IV NSCLC between 2007 and 2017, treated using chemotherapy. Using Cox's PH regression model, we adjusted the analysis for location (rural vs urban); gender; distance from nearest cancer center; first chemotherapy treatment used; income brackets; and age.

Results: The regression analysis indicated that type of the chemotherapy, length of time to the first treatment, and distance from the nearest center had a statistically significant impact on survival. Paclitaxel was associated with less risk of death when compared to Vinorelbine, Gemcitabine, and Docetaxel. Every additional 10 km distance from the nearest cancer center was associated with 0.5% increased risk of death. A longer time to the first treatment was associated with better survival. In fact, every 10 days increase in wait time was associated with 0.5% decrease in the risk of death.

Conclusion: We conclude from this study that newer chemotherapy agent use and living closer to the cancer center is associated with better survival. Longer time between diagnosis and treatment leading to better survival could perhaps be explained by asymptomatic or less symptomatic patients receiving treatment later.

ABSTRACT P29

DIFFERENTIAL RESPONSES TO A CARBOPLATIN CONTAINING REGIMEN IN THE HUMAN EPIDERMAL GROWTH FACTOR RECEPTOR 2-POSITIVE, HORMONE RECEPTOR-NEGATIVE BREAST CANCER POPULATION

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Background: The HER2-positive, HR-negative breast cancer population is widely unexplored. Studies have shown decreased toxicity benefits when using carboplatin based chemotherapy in HER2+ patients. We set out to investigate the benefits of using carboplatin in the HER2+/HR- population specifically.

Methods: We explored our database of 1734 patients from 2004-2010 finding a total of 96 HER2+/HR- patients. Twenty patients were removed because they did not have invasive cancer or did not receive chemotherapy. A retrospective chart review was then conducted to compare progression free survival and overall survival in 76 HER2+/HR- patients, of which 62 received non-carboplatin containing therapy and 14 received TCH (carboplatin containing).

Results: 5.54% of patients in our database fit the phenotype of HER2+/HR-. Over a three year follow up, a progression free survival rate of 85.71% in the carboplatin group was significantly different than 79.03% in the non-carboplatin group. An overall survival rate of 92.86% in the carboplatin group was significantly different than 74.19% in the non-carboplatin group. The limitation here is the small sample size of the carboplatin group and shorter follow up time.

Conclusions: In conclusion, the HER2+/HR- phenotype is a small subset of patients, but we have shown that they have improved outcomes with carboplatin containing chemotherapy. The restrictions of a small retrospective study define this as hypothesis-generating only. A prospective study investigating this population of patients would help to define the best treatment for this unique group.

ABSTRACT P30

EARLY STAGE LUNG CANCER STEREOTACTIC BODY RADIATION THERAPY OUTCOMES IN WINDSOR ONTARIO: A SINGLE INSTITUTION EXPERIENCE

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This retrospective review focuses on the outcomes of stage-I non-small cell lung cancer (NSCLC) patients treated with different radiation therapy modalities at Windsor Regional Hospital between 2015 and 2020. We collected a total of 140 consecutive patients' data who received stereotactic body radiotherapy (SBRT), hypofractionation, conventional fractionation, and palliative radiation. The primary and secondary endpoints were the differences in local control (LC) and overall survival (OS). The numbers of patients in each treatment subgroups were: SBRT (45), hypofractionation (78), conventional fractionation (8), and palliative (9). Patient median age was 74 (range 52-91). Cox's proportional hazards regression analysis was performed. Differences in age and gender between subgroups were not statistically significant. Kaplan-Meier curves were plotted for LC and OS. Post-treatment 2-year LC was found to be significantly better with SBRT (100%), versus hypofractionation (85%), conventional fractionation (80%), and palliative (73%). OS at 2 years were: SBRT (90.48%), hypofractionation (77.43%), conventional fractionation (62.50%), and palliative (44.44%). Proportion of patients who experienced post-treatment radiation pneumonitis or dermatitis were: SBRT (7%, 2%), hypofractionation (8%, 3%), conventional fractionation (13%, 25%), and palliative (0%, 0%), respectively. Hazzard ratio analysis found: every 20 years of patient age were associated with 3.8 folds increased death (95% CI: 1.592—9.772). Compared to SBRT, risk of death was higher for other treatments, 2.6 times for hypofractionation and 5.8 times for palliative. In conclusion, our experience confirms SBRT can provide durable local control with comparable rate of post-treatment complications versus other radiation modalities for early stage NSCLC.

ABSTRACT P31

THE NKR-P1B:CLR-B AXIS' INHIBITORY EFFECTS ON NK CELL-MEDIATED IMMUNE RESPONSES AGAINST MAMMARY TUMORS

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Natural Killer (NK) cells kill their target cells, such as cancer cells, by inducing apoptosis in the target cells. NK cell functions are regulated by signals from an array of activating and inhibitory receptors. Activating NK cell receptors recognize ligands associated with cellular stress and oncogenic transformations. NK cells also recognize antibody-bound target cells through their Fc receptors, which bind to the Fc portion of the antibodies and mediate antibody-dependent cell-mediated cytotoxicity (ADCC). NK cell mediate ADCC against HER2+ breast cancer cells coated with anti-HER2 antibody (Trastuzumab). Cancer cells also express ligands, which can bind to the inhibitory NK cell receptors, leading to NK cell inhibition and immune evasion. An example of the inhibitory receptor in mice is NKR-P1B, which recognizes C-type lectin-related glycoprotein b (Clr-b). In this project, we study the impact of NKR-P1B:Clr-b recognition in HER2-targeted ADCC against a mouse mammary adenocarcinoma cell line expressing human HER2 (E0771-HER2 cells). Using the mouse NK cells and E0771-HER2 mammary tumour cells in in vitro cytotoxicity and HER2-targeted ADCC assays with or without disruption of NKR-P1B:Clr-b interactions, we will determine whether NKR-P1B negatively regulates NK cell's ADCC activity, and whether the blockade of the NKR-P1B:Clr-b axis will enhance HER2-targeted ADCC activity of NK cells against mammary tumours. The mouse NKR-P1B:Clr-b axis is analogous to the human NKR-P1A:LLT-1 axis, and the findings from this work will serve as a proof-of-concept for the blockade of the human NKR-P1A:LLT-1 axis to enhanced NK cell activity and anti-HER2 therapy in HER2+ breast cancer patients

ABSTRACT P32

EVALUATION OF HIGH-SENSITIVITY BREAST SPECIFIC POSITRON EMISSION MAMMOGRAPHY (PEM) SYSTEM

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Molecular Breast Imaging with Positron Emission Mammography (PEM) scans has a potential to move the field of breast cancer diagnosis forward by addressing existing limitations related to poor diagnostic imaging in dense breasts and poor prognostic information for pre-invasive cancers. Combined with novel imaging biomarkers, the technology could identify specific cohorts of patients who would benefit from emerging personalized treatments. However, wide-spread clinical use of PEM requires a significant improvement in technology for a balance between Spatial Resolution and Sensitivity. In comparison to whole-body PET scanners, the Sensitivity must be significantly improved to allow the

administered dose of a biomarker to be reduced by at least 5 times, thus reducing the radiation burden on the undiagnosed patient population. In addition, fast image acquisition is required to reduce exam time and to improve patient throughput.
Here we report the results of the National Electrical Manufacturers Association (NEMA) NU-4 performance measurements, phantom images, and clinical images for the high-resolution high-sensitivity PEM system developed for an optimal balance between a Resolution, Sensitivity and Count Rate capability. An in-plane Spatial Resolution of 2.3 mm, Sensitivity of 3.5%, and peak Noise Equivalent Count Rate of 17.8 kcps are determined. Micro-hotspot Phantom lesions are resolved down to the 1.35 mm hot-rods, and the NU-4 Image Quality Phantom shows the 2 mm hot-rod resolved. Clinical Images show clear visualization of malignant tissues in a variety of scanned patients. The demonstrated best-in-class Sensitivity should translate into a marked improvement in the low dose clinical performance of PEM scanners.

ABSTRACT P33

RUXOLITINIB HELPS CLEAR POLYCYTHEMIA VERA AND SQUAMOUS CELL CARCINOMA

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Polycythemia Rubra Vera (PV) is a clonal stem-cell disorder characterized by unregulated production of red cells, white cells, and platelets and complicated by extramedullary hematopoiesis, myelofibrosis, and acute leukemia. The 78-year-old-gentleman was diagnosed with PV in August 2004 and had squamous cell carcinoma (SCC) of the skin. Ruxolitinib is a JAK/STAT inhibitor. He was started on 20 mg twice daily for 16 months starting on August 7, 2012 until reduced to 15 mg due to anemia on January 22, 2014. Since Ruxolitinib initiation, his skin lesions significantly improved. His facial lesions resolved almost completely. He gained a healthy amount of weight and improved both physically and mentally. He went up to 91.8 kg from 88.8 on January 23, his blood pressure is 133/70- his systolic blood pressure is still considered high, however his diastolic is normal. His pulse is healthy at 65, respiratory rate was 20. Followed by dermatology, his skin continued to improve with no change in treatment. This case shows that there is some similarity between SCC and PV as Ruxolitinib managed to clear both. It is common to see Ruxolitinib clear PV, and there are also cases where it has caused aggressive SCC in patients. However, this patient was cleared of both diseases. We believe this happened due to a rare chance of SCC tumour growth and proliferation happening through JAK/STAT pathway induction. Hence, both cancers were targeted by the potent and selective oral inhibitor of both JAK2 and JAK1 protein kinases.

ABSTRACT P34

PORTABLE MAGNETIC RESONANCE SCANNER DIRECT CURRENT COIL DEVELOPMENT FOR ACCESSIBLE CANCER SCREENING

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Developments for cancer diagnosis can be immensely advanced through the wider access to screening technology. Magnetic resonance imaging (MRI) is favourable within tomography because it provides high resolution 3D images with rich soft tissue contrast. No harmful radiation is required in this non-invasive technique. However, there is insufficient access because of the high instrument cost and high maintenance cost. We are developing a low cost portable MRI that can enable point-of-care diagnosis.
MRI requires a homogenous static magnetic field. The cost effectiveness and portability of the novel MR scanner were achieved at the expense of poor magnetic field homogeneity. Shimming coils can be employed to improve the homogeneity and image quality. MRI also requires three-dimensional magnetic field gradients to encode the spatial information and form an image, which are generated by gradient coils. These direct current coils must be specifically designed based on the magnet properties, including the magnetic field distribution, space constraint, and heat dissipation. In this work, our recent development of shimming and gradient coils will be presented.

ABSTRACT P35

DISSECTING THE TUBERIN-CYCLIN B1 INTERACTION BEFORE CELL DIVISION

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Tuberous Sclerosis (TS) is a genetic disorder that causes benign tumours to form in the kidneys, brain, skin, and other organs. The disease is caused by inactivating mutations in either the TSC1 or TSC2 gene encoding for Hamartin and Tuberin. Mechanistically, Hamartin and Tuberin form a tumour suppressor complex by negatively regulating the mTORC1 pathway and inhibiting protein synthesis. Our lab has characterized how Tuberin regulates the G2/M transition through binding to and controlling the localization of Cyclin B1 (CycB1). We have determined that phosphorylation of five key residues in CycB1 relaxes this binding interaction and permits accumulation of CycB1 in the nucleus and mitotic onset. We hypothesize that CycB1 may also affect the biology of Tuberin through key residues on CycB1.
We assessed this question through 3 aims: 1) Construct CycB1 phospho-mutants using site-directed mutagenesis to determine the sites that mediate its binding to Tuberin. 2) Quantify the mitotic index for each CycB1 mutant and changes in Tuberin cellular localization. 3) Develop fluorescent tools to study the localization of Tuberin by using CRISPR-Cas9. Our results show that decreases in Tuberin-CycB1 binding lead to increased Tuberin nuclear localization and increased mitotic onset. Understanding how Tuberin can regulate proper cell division will further our understanding of how tumour disorders such as TS and cancers develop.

ABSTRACT P36

CHARACTERIZATION OF KAISO OVEREXPRESSING MICE SUBJECTED TO CARCINOGENIC TREATMENT

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Colitis-associated cancer (CAC) is a colorectal cancer subtype that is characterized by chronic intestinal inflammation. Long duration and early onset of inflammatory bowel disease (IBD) can progress to CAC via an accumulation of mutations in intestinal epithelial cells that result in abnormal growth. IBD sufferers have ~2x greater likelihood of developing CAC than the general population. However, the molecular mechanisms underlying this transition from IBD to CAC remain unknown. Initial characterization of the transcription factor Kaiso revealed roles in the regulation of vertebrate development, inflammation, and tumorigenesis in colorectal and other types of cancer. Studies examining the effects of intestinal-specific Kaiso overexpression in mice revealed phenotypic characteristics common to IBD sufferers such as crypt expansion and hyperplasia. Thus, we hypothesized that Kaiso-induced inflammation primes the intestinal environment for polyp formation when subjected to a second "carcinogenic" assault. Kaiso overexpressing mice were treated with the carcinogen azoxymethane (AOM) and the detergent dextran sodium sulphate (DSS) to investigate if they would work synergistically to accelerate intestinal inflammation to polyp formation. AOM and DSS treated intestinal tissues exhibited widespread areas of focal hyperplasia and dysplasia, ulceration and severe inflammation that worsened in Kaiso overexpressing tissues. There were also signs of increased neutrophil infiltration into the lamina propria and indications of altered cellular proliferation in intestinal tissues. Kaiso-overexpressing mice also exhibited impeded weight gain that was exacerbated by AOM and DSS treatment. These findings together support a role for Kaiso in promoting intestinal inflammation, the regulation of intestinal repair and tumorigenesis.

ABSTRACT P37

INTEGRATIVE ANALYSIS OF MIRNA AND MRNA: AN APPLICATION TO SPARSE CANONICAL CORRELATION ANALYSIS

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Motivation: miRNA are non-coding RNA, they regulate mRNA expression. There have been numerous statistical methods available for finding relationships between them. Sparse Canonical Correlation Analysis is a multivariate statistical method introduced to find cross-correlation between two data types. Results: NCI 60 cancer Agilent miRNA and mRNA expression were used and have obtained a sparse set of miRNA and mRNA expression are highly correlated. Furthermore, gene set enrichment analysis is used to obtain the biological functionality of the expressions.
Key Words: CCA; SCCA; Multivariate Statistical Methods; High-Dimensional Data; miRNA expression; mRNA expression.

ABSTRACT P38

CRANIOSPINAL IRRADIATION IN THE TREATMENT OF CHEMOTHERAPY REFRACTORY LEPTOMENINGEAL METASTASIS FROM BREAST CANCER: A CASE REPORT

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Leptomeningeal carcinomatosis is a relatively uncommon complication of solid tumours that is associated with significant morbidity and mortality. Prognosis is typically weeks to months and the neurologic complications of this disease can significantly affect quality of life. The role of craniospinal irradiation is unclear as evidence exploring this treatment option is limited. Despite lack of evidence its use has decreased due to its associated acute toxicities and newer intrathecal alternatives. Here we report the case of a 50-year-old patient who received craniospinal irradiation for chemotherapy-refractory leptomeningeal disease, with survival well beyond the median and good quality of life for the majority of that time. This patient's remarkable survival and performance after treatment suggests that craniospinal irradiation could be considered more frequently in the treatment of leptomeningeal metastases. To our knowledge this is the first case with significant survival following craniospinal irradiation for chemotherapy refractory disease presented. Further study on the use of craniospinal irradiation to treat leptomeningeal metastasis is recommended.

ABSTRACT P39

ADMINISTRATIVE TIMING OF PACLITAXEL IN COMBINATION WITH CARBOPLATIN INFLUENCES TREATMENT EFFICACY OF TRIPLE-NEGATIVE BREAST CANCER

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Breast cancer is the second highest cause of death from cancer in Canada. Triple-negative breast cancer (TNBC) accounts for 10-15% of all cases and has a poorer prognosis than other breast cancer subtypes. TNBC lacks expression of the estrogen receptor, progesterone receptor, and the human epidermal growth factor receptor 2 (HER2), which are common therapeutic targets in breast cancer. The standard of care for treatment of TNBC instead consists of adriamycin (A), paclitaxel (T), cyclophosphamide (C), and sometimes carboplatin (Ca) to target various aspects of the cell cycle in order to induce cell cycle arrest. Timing of administration may affect cell cycle arrest, and alterations in cell cycle mediators may also influence the efficacy of treatment. The purpose of this study was to determine how the addition and timing of treatments influence the cell cycle and how this knowledge can be used to help determine more effective timing of treatment administrations. MDA-MB-231 TNBC cells were treated with AC, T, T+Ca, or Ca at various time points. Flow cytometry and Trypan Blue exclusion assay were used to determine cell cycle progression and proliferation rate. It was found that different combinations of drugs resulted in the arrest of cells at various phases of the cell cycle which may affect responsiveness to subsequent treatments. This information can be used to help determine the most effective timing of treatment and may help improve the 5-year survival rate of patients with TNBC.

ABSTRACT P40

INVESTIGATING ALDH AS A BCSC MARKER AND THE EFFECT OF SPY1, A NON-CANONICAL CYCLIN-LIKE PROTEIN, ON THE BCSC POPULATION

Nick J. Philbin[1*], Ellen M. Laurie[1], Bre-Anne Fifield[1], Lisa A. Porter[1]

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Breast cancer is the most commonly diagnosed cancer in women, and the tremendous heterogeneity of the disease complicates treatment. Triple Negative Breast Cancer (TNBC) occurs in 10-15% of the breast cancer diagnoses and typically has poorer outcomes than other subtypes of breast cancer. This is largely due to lack of targeted therapies and the existence of a population of cells known as breast cancer stem cells (BCSCs). BCSCs are more resistant to therapy and capable of driving patient relapse. Cell cycle mediators may play a key role in driving expansion of this population of dangerous cells. Spy1, a cyclin-like protein, promotes cell cycle progression through the G1/S, and the G2/M phase of the cell cycle and has been shown to be elevated in TNBC patients. Using an in vitro model of TNBC (MDA-MB-231 cell line), the relative abundancy of the BCSC population can be assessed to determine if increased levels of Spy1 can expand the BCSC population resulting in more aggressive, invasive and fatal cancers. BCSCs can be identified using markers such as the ALDH isoforms via measurement of ALDH activity or ALDH expression reporter constructs. This work seeks to determine the most effective method of monitoring ALDH activity and if Spy1 is capable of regulating the BCSC population elucidating potential therapeutic promise.

ABSTRACT P41

EXPLORING MICROENVIRONMENTAL INFLUENCES ON GLIOMA PROGRESSION AND AGGRESSIVENESS

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The tumour microenvironment, composed of proliferating tumour cells and various non-tumour cell types including immune cells, blood vessels and fibroblasts, plays a crucial role in driving cancer progression and aggressiveness. The interactions between tumour cells and their microenvironment underly this increased proliferation. We postulate that in gliomas, this is due in part to microenvironmental remodelling wherein we observe changes in concentrations of vinculin and hyaluronic acid. In gliomas, the tumour microenvironment is unique due to the presence of distinct neural cell types including astrocytes, neurons and microglia. To explore the role of these brain tissue specific cells within the tumour microenvironment, we employed a host of in vitro and in vivo techniques to observe microenvironmental remodelling and explore influences on tumour characteristics. We also explored whether or not direct contact between gliomas and the microenvironment is necessary to initiate remodelling or alter aggressiveness, or if factors secreted by the tumour into the microenvironment are sufficient. Interestingly, we observed increases in markers associated with disease progression and tumour aggressiveness when tumour cells were able to interact, both directly or indirectly, with specific components of the brain tumour microenvironment. This study contributes to the growing body of literature on the effects of the microenvironment in cancer progression.

ABSTRACT P42

RETROSPECTIVE CHART REVIEW OF STAGE 3 NON-SMALL CELL LUNG CANCER IN THE WINDSOR REGIONAL CANCER CENTER FROM 2008 TO 2015

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Lung cancer is a commonly diagnosed malignancy that remains a leading cause of cancer death in Canada. Poor survival may be influenced by most cases being diagnosed at a late stage. In Canada from 2012-2016, non-small cell lung cancer (NSCLC) accounted for approximately 88% of lung cancer diagnoses. 20% of lung cancers were diagnosed at stage 3, with a 3-year predicted net survival of 22%. To evaluate lung cancer outcomes at the Windsor Regional Cancer Center we conducted a retrospective chart review of patients clinically diagnosed with stage 3 NSCLC from 2008 to 2015. For the 253 patients identified (38.3% female, 61.7% male) we collected information on patient characteristics, tumor factors and treatment received. Treatment was categorized into standard treatment including chemotherapy and radical radiation, no treatment and non-standard treatment encompassing all other treatments. The patient population age range was 39 to 88 years, with a median age of 68 years. The primary tumor was found either in the right or left upper lobe in 64.7% of patients. Overall 3-year survival considering all-cause death was 20.75%. Comparing treatment groups, the 3-year survival for patients who received no treatment, non-standard treatment and standard treatment was 9.68% (95% CI 2.47, 22.91), 9.51% (95% CI 5.22, 15.34) and 41.09% (95% CI 30.78, 51.10) respectively. In conclusion, we found 3-year survival of stage 3 NSCLC patients treated at Windsor Regional Cancer Center to be comparable to the national rates. However, there were differences between treatment groups with standard treatment having a higher 3-year survival.

ABSTRACT P43

RECURRENCE-FREE SURVIVAL AND OVERALL SURVIVAL OF STAGE 2 AND 3 COLON CANCER PATIENTS MANAGED WITH OR WITHOUT CHEMOTHERAPY

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The benefits of adjuvant chemotherapy are contested in stage 2 and 3 colon cancer. This retrospective review examined outcomes of colon cancer patients treated with or without adjuvant chemotherapy, stratified by stage 2 or 3. Chart data reviewed was of patients diagnosed with stage 2 or 3 colon cancer from 2013 to 2018 at a single center. The primary endpoint was recurrence-free survival (RFS) and the secondary endpoint was overall survival (OS), which are reported with their 95% confidence intervals at 3 years and 5 years respectively. Kaplan-Meier curves compared via stage-stratified log-rank tests over the entire follow-up period did not detect differences between the chemotherapy groups and the surveillance groups in RFS (log-rank=0.13, p=.72) nor in OS (log-rank=0.015, p=.90). In stage 2 patients (n=151), 3-year RFS was estimated to be

76% (54-88%) for the chemotherapy group and 86% (79-91%) for the surveillance group, while 5-year OS was estimated to be 62% (37-80%) for the chemotherapy group and 62% (52-71%) for the surveillance group. In stage 3 patients (n=163), 3-year RFS was estimated to be 69% (60-76%) for the chemotherapy group and 61% (43-74%) for the surveillance group, while 5-year OS was estimated to be 59% (50-68%) for the chemotherapy group and 55% (37-70%) for the surveillance group. This data indicates that chemotherapy does not predict survival outcomes based on disease stage alone, and that other prognostic factors must be considered before management decisions regarding chemotherapy.

ABSTRACT P44

INVESTIGATING THE ANTICANCER ACTIVITY OF A NOVEL ROSEMARY EXTRACT IN COLON CANCER

Benjamin Scaria[1*], Christopher Raad[1], Jana Khanafer[1], Anthony Haddad[1]

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Side effects of non-selective treatments remains a major issue in cancer therapy. Natural health products (NHP's) are known to have anticancer properties, such as anti-proliferative and anti-inflammatory effects. Rosemary extract (RE) has been shown to have various anticancer effects in multiple cancer models in-vitro and in-vivo via different pathways. A novel rosemary extract, with 5% rosmarinic acid has been synthesized by our industry partners, Synthite industries. We are currently investigating the in-vitro efficacy of the extract in inhibiting the viability of cancerous and non-cancerous colon cells, namely using HCT-116 and HT-29 colon cancer cell lines, and NCM460 as the normal cell line. The objectives are to characterize the dose-dependent effect of RE on cell viability in cancerous cells lines and determine the effect on the normal colon cell line; elucidate the nature of drug-drug interactions when combined with FOLFOX (commonly used chemotherapy drug in colon cancer); elucidate the mechanism of action involved in the effect on cell viability; and conduct an in-vivo study to determine the efficacy of RE in a mouse model. Preliminary results from trypan blue and AVPI cell viability assays indicate the extract induces apoptosis in both HCT-116 and HT-29 cells at various time points. Furthermore, there is an additive effect when treated in combination with FOLFOX as opposed to the extract alone, displaying positive drug-drug interactions. However, the mechanism of apoptotic induction and the investigation of in-vivo efficacy of the extract remains to be characterized.

ABSTRACT P45

SENSORS FOR MEDICAL APPLICATIONS

Calvin Love[1*]

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Medical applications for electronic nose devices have been explored extensively over the past several decades. The gas sensing elements in these devices analyze breath exhaled from the subject, targeting specific volatile organic compounds (VOCs) that act as biomarkers for respiratory diseases such as asthma, COPD and lung cancer to name a few. Typically, these gas sensing elements are designed to react to a single analyte of interest and for this reason many sensors must be employed to test for multiple respiratory diseases at once, increasing e-nose production cost, size and power consumption. In the proposed design, multiple VOC concentrations are measured using a single gas sensor element. An embedded microheater varies the temperature of the sensor, causing the functionalized polymer to react to different VOCs at different temperature setpoints. A miniaturized impedance analysis and frequency spectrometry circuit is utilized to measure the shift in electrical characteristics of the polymer under exposure to test gases during sensor temperature cycling. This highly undegradable design limits the number of micromachined sensing elements necessary to test for multiple VOCs in single e-nose system; reducing the production cost, power consumption and sensor footprint making respiratory disease testing more widely available.

ABSTRACT P46

A NOVEL LOW FREQUENCY PIEZOELECTRIC MICROMACHINED ULTRASONIC TRANSDUCER (PMUT) FOR MEDICAL IMAGING APPLICATIONS

Jay Nagarajan[1*]

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There has been decline in the breast cancer causalities around 1.3% per year from 2013 to 2017 due to early detection using advanced imaging technologies. This signifies the early detection of breast cancer. In exchange, this increases the patient's survival rate and treatment choices.

Microelectromechanical (MEMS) systems are one of the platforms that enable early detection. In this research, a MEMS-based transducer is proposed which can overcome disadvantages from its counterparts like Capacitive Micromachined Ultrasonic Transducers (CMUT). The designed transducer can operate at lower frequencies to support image applications with higher penetration when running on lower voltage. The size of detection devices can be anywhere between 100 to 500 micrometers. So, it can be manufactured and packaged inside a single chip which can be handheld. The designed chip consists of 50 individual devices with varying shapes and structures. These chips can potentially be planted within a handheld imaging device that can easily identify tumor cells against the fatty tissues in the breast. The resonant frequency and acoustic output pressure of the proposed designs were measured and compared using COMSOL Multiphysics based on their respective piezo layer areas. The frequency of resonance for the emitted PMUT is between 0.5 MHz and 2 MHz. The fabricated individual circular PMUT achieves a high acoustic output pressure of 39 kPa at 1.3 MHz and the rectangular PMUT provides 4.7 kPa of acoustic pressure at 1.4 MHz. These results indicate that the proposed PMUT design delivers high acoustic pressure at a lower frequency range which can be implemented for high resolution imaging.

ABSTRACT P47

REAL WORLD EXPERIENCE – STOPPING STUDY IN CHRONIC MYELOID LEUKEMIA

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This retrospective chart review had the purpose of showing that chronic myeloid leukemia (CML) patients can stop their tyrosine kinase inhibitor (TKI) treatment safely while remaining in treatment-free remission (TFR), without some of the strict laboratory criteria traditionally used in clinical trials in the field. The outcome of cessation of therapy for 9 chronic-phase CML patients who stopped their TKI treatment for various reasons is presented. The total duration of TKI of treatment before the stopping trial had a median of 102 months (range 64-193). The duration that each patient lasted in a MR4.0 response level (qt-PCR results of less than 0.01% for BCR-ABL1 transcripts on the International Standard) before stopping their TKI treatment was a median of 46 months (range 12-179). Among all patients, 7 (78%) were considered successful and 2 (22%) were considered not successful in remaining in TFR. Patients were tested either every 6 or 12 weeks after stopping treatment to monitor for a loss of major molecular response (MMR) as an endpoint for determining reoccurrence. With this patient experience data, we hope to pave the way for a prospective trial that can give evidence for a new clinical paradigm that would be helpful in the real world with every 6-week testing and using MR4.0 as the necessary depth of remission. These laboratory criteria and requirements accessible in Ontario facilities are essential for these new guidelines, in order to stop otherwise lifelong TKI treatment for many CML patients of the region and others experiencing similar barriers.

ABSTRACT P48

RESULTS OF SAFETY MEASURES IN RADIATION ONCOLOGY DEPARTMENT DURING THE FIRST WAVE OF COVID-19 PANDEMIC

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Introduction: The incidence and mortality rates of COVID-19 are higher with cancer diagnosis. This study is to share the experience of our radiation oncology department to reduce impact on vulnerable cancer patients.

Methods: We have taken precautious measures including restricted visitor policy; active screening; social distancing; Telemedicine for most consultation and follow up visits; universal COVID-19 swabbing test for all cancer patients before starting treatment planning or radiotherapy; full personal protective equipment (PPE) for staff doing CT simulation or delivering treatment; mandatory face mask for everyone in the building.

Results: We saw 267 new consults in the 10 weeks between March 16 and May 24, 2020, vs 274 in the same period last year. There is no significant difference in average consults per Radiation Oncologist, 44.5 (30-60) vs 45.7 (24-67), p=0.799 (Student's t-test); or wait time within provincial target of 2 weeks, 93.5% vs 97%, p=0.074. We performed 193 swabbing tests for 183 patients, with 10 patients being swabbed twice. Only 0.52% tested positive (1 asymptomatic case), lower than many other cancer institutions reported in the literature and no cases among staff. During the same 10 weeks, confirmed cases in our community and the province increased from 0 to 912 (6.05% positive tests) and from 142 to 25,904 (4.18% positive tests), with 63 and 2,102 deaths, respectively.

Conclusion: It is possible for frontline health care team to minimize the risk of cancer patients getting COVID-19 and avoid treatment interruptions by planning safety measures early during the first wave of pandemic.

ABSTRACT P49

ASHTANGA YOGA FOR BREAST CANCER SURVIVORS: REGAINING BODY OWNERSHIP, COMMUNITY BUILDING, AND SPIRITUAL DEVELOPMENT

Hannah Lauzon[1*], Ashley Howard[1], Josée Jarry[1]

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Upon completing treatment, some breast cancer survivors face a post-cancer journey in which they endure the negative effects of cancer and its treatment on various aspects of their life, including psychological functioning and physical mobility. For example, 38% of breast cancer survivors report severe anxiety and 22% report severe depression. Further, many experience physical limitations due to scar tissue and general weakness. As such, it is imperative to identify post-treatment interventions that may address these sequelae. The goal of the present study was to assess the effectiveness of an Ashtanga yoga intervention to relieve some of these psychological and physical negative consequences of breast cancer and its treatment. Participants were 12 women who had completed treatment for stages one to three breast cancer. Participants attended 16 yoga classes over eight weeks, comprised of training in asana, controlled breathing, and Ashtanga's philosophical foundations. Participants also attended bi-weekly focus groups, where they discussed their experiences of the intervention. Reflexive thematic analysis of the transcripts revealed stable themes that were consistently discussed through all focus groups: a sense of community and support within the intervention, and a sense of loss associated with breast cancer and its treatment. Analyses also revealed an evolution from a focus on physical limitations and the physical challenges of the practice, to a focus on the spiritual and emotional gains from the practice. This research highlights the benefits of implementing Ashtanga yoga to support the psychological well-being of breast cancer survivors.

ABSTRACT P50

DEXAMETHASONE INCREASES TOXICITY OF CYCLOPHOSPHAMIDE IN ZEBRAFISH

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Dexamethasone (Dex) is frequently given to cancer patients to counteract the negative side effects of chemotherapeutics. Although, Dex has proven to be an effective anti-emetic for those receiving chemotherapy, it is unclear whether Dex contributes to the more severe side effects of these drugs that plague cancer patients, such as heart and liver failure that can lead to patient death. Being able to study the toxicity of Dex and chemotherapeutics in vivo is critical to determine if Dex is the optimal anti-emetic for cancer patients. Zebrafish have become a prominent model to study drug toxicity and in this study, we use zebrafish embryos to determine the toxicity of the combination of Dex and common chemotherapeutics, cyclophosphamide and paclitaxel. Administering Dex and cyclophosphamide together, caused extreme heart and liver toxicity in zebrafish embryos. However, Dex and paclitaxel, together, did not show the same severe toxicity. Gene expression analysis revealed that Dex alone increased inflammatory gene response and Dex and cyclophosphamide, together, increased mmp-9 expression, which has previously been indicated in cardiac toxicity. These results together suggest that Dex interacts with chemotherapeutics to cause toxicity to major organs. It is imperative that Dex be reconsidered as a universal antiemetic for all cancer patients and alternatives need to be explored.

ABSTRACT P51

RESOURCES, EDUCATION, NUTRITION, EXERCISE AND WELLNESS (RENEW) – A CANCER WELLNESS PROGRAM

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Background: The Erie St Clair Regional Cancer Program (ESCRCP) realized a gap in organized support, information and education for patients nearing end of treatment/transitioning back to community, often resulting in increased anxiety. RENEW development and implementation was undertaken with the objective to meet the identified patient needs for a true person-centred approach.

RENEW was developed based on extensive mixed methods research including interviews, focus groups, and surveys with patients, caregivers/providers. Results identified areas of need and informed program content and design leading to RENEW comprising an education series and an exercise program. An interdisciplinary team facilitates interactive education sessions. The exercise program, delivered by specially trained instructors, provides structured sessions based on Cancer Care Ontario guidelines.

Results: Overall participant feedback:

83% of attendees indicated that they would not have received this information elsewhere

96% of attendees agree/strongly agree that the sessions helped ease their anxiety

96% of attendees agree/strongly agree they learned about the community resources available to them
98% of attendees agree/strongly agree that they were satisfied with the sessions.
Participation in the 2019 exercise program more than tripled since 2018.
Discussion/Conclusions:Through extensive collaborative partnerships across the region, RENEW has proven to be an innovative and successful program. Program expansion plans include partnering with additional fitness facilities, diversifying the educational topics presented and increasing frequency of sessions. Future plans also include a collaborative research study focused on the exercise program with the University of Windsor and University of Toronto.

ABSTRACT P52

VALIDATION OF A NOVEL NANOPARTICLE SYSTEM FOR SELECT TARGETING OF THERAPY-RESISTANT CELL POPULATIONS IN GLIOBLASTOMA

Dorota Lubanska[1], Fatima Nadeem[1*], Gage Mason[2*], Mitchell DiPasquale[2], Aleena Malik[2], Drew Marquardt[2], Lisa A. Porter[1], Simon Rondeau-Gagné[2]

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Glioblastoma (GBM) remains one of the most aggressive forms of brain cancer with dismal treatment options. Successful therapy of GBM is hampered by poor blood-brain barrier drug penetration and the existence of immature tumour initiating cells (TICs), positive for CD44 receptor. CD44 has been demonstrated previously to drive therapy resistance and relapse in patients with GBM. Hence, therapies offering effective bioavailability and selective TIC targeting are of high demand. Our team has successfully synthetized conjugated polymer nanoparticles (CPNs) of blood-brain barrier- penetrating diameter and electrotherapeutic potential. The particle design includes Hyaluronic Acid (HA), a ligand for CD44 receptor, allowing for selective targeting of CD44 positive TICs. Initial characterization in our lab demonstrated concentration- dependent selective CPN uptake by CD44+ GBM cells and antiproliferative activity of the CPNs. Our next steps are to evaluate the impact of CPNs on the biology of glioblastoma using patient samples and a pre-clinical animal model. This project brings together a multidisciplinary research team to generate and evaluate innovative tools with a potential to improve diagnostics and treatment strategies for patients with GBM.

ABSTRACT P53

NOVEL BIOINFORMATIC APPROACH TO ANALYZE LARGE-SCALE, NEXT-GENERATION SEQUENCING-BASED TARGETED GENE PROFILING DATA FOR PERSONALIZING TREATMENT OPTIONS IN CANCER.

Abedalrhman Alkhateeb[1*], Govindaraja Atikukke[1], Ashraf Abou Tabl[1], Julianna Facca[1], Luis Rueda[1]

[1]University of Windsor

Advances in genomics technologies have opened a world of molecular diagnostic possibilities that are now available to cancer patients and their oncologists. In a growing number of cases, the ability to detect specific driving oncogenes in cancer results in the use of precision medicines that directly target the oncogenic driver, with dramatic results for patients. At ITOS, we use state-of-the-art, targeted genomic sequencing technology using several different gene panels including TSO500, a gene panel covering over 500 genes that are associated with cancer. These genomic variations identified include simple mutations, copy number alterations, insertion/deletions (INDELs), gene fusions, microsatellite instability (MSI) as well as detection of tumor mutational burden (TMB).

Here, we introduce a bioinformatics pipeline for systematic analysis of the data generated from TSO500 to generate a clinically actionable personalized report for an individual cancer patient. First, our pipeline incorporates the genomic tools for aligning DNA and RNA reads to the reference human genome to create variants call file (VCF). The VCF is further analyzed using our integrative approach that includes several curated precision oncology knowledge bases such as OncoKB, the Catalogue Of Somatic Mutations In Cancer (COSMIC), dbSNP, as well as literature review and manual curation to ultimately generate a comprehensive and informative personalized report for an individual cancer patient.

ABSTRACT P54

THE LOSS OF CIRCADIAN CLOCK GENE BMAL1 INCREASES TUMOUR INITIATION IN APCMIN MICE

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Circadian rhythms are autonomously running 24h cycles in physiological processes driven by a molecular time-keeper known as the circadian clock: a transcription-translation feedback cycle composed of the transcription factors Bmal1 and Clock as well as their repressors Per and Cry. The circadian clock regulates over 40% of the genome rhythmically. Chronic circadian disruption, such as shift work, has many negative health outcomes. Colorectal cancer is most frequently initiated through a mutation in the Wnt pathway regulator, Apc. Attempts to provide a mechanistic link between tumourigenesis and circadian disruption have been unclear due to the use of mice on mixed genetic backgrounds and poor circadian models. To address the clock's role in cancer, we created an isogenic strain by crossing Apcmin mice, a common intestinal tumour model, with Bmal1 mutant mice (Bmal1-/-), which lack a functioning circadian clock. RNA sequencing of intestinal organoids, a 3D cell culture method, revealed 41 circadian targets including Tead4 a regulator of intestinal stem cells. Bmal1-/- mice harbour twofold more tumours than Bmal1+/+ mice and upregulate hippo pathway members including Tead4 while downregulating Wnt pathway members. Apcmin; Bmal1-/- organoids display enhanced self-renewal which is reduced to Apcmin; Bmal1+/+ levels in the presence of a Tead4 inhibitor. The loss of circadian clock function in the intestinal epithelium leads to increased tumourigenesis caused in part by the dysregulation of hippo pathway mediator Tead4. This research has important implications for the understanding of intestinal stem cell biology in health and disease.

ABSTRACT P55 **not participating in poster session*

ANALYSIS OF THE GENOMIC LANDSCAPES OF BARBADIAN AND NIGERIAN WOMEN WITH HIGH PREVALENCE OF TRIPLE NEGATIVE BREAST CANCER (TNBC)

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Breast cancer (BCa) is currently a major cause of death in women worldwide and women of African Ancestry (WAA) have a disproportionately higher mortality rate compared to women of European ancestry. This is partially explained by the higher prevalence of aggressive triple negative breast cancer (TNBC) in WAA. The high prevalence and poor outcome of TNBC in WAA emphasizes the importance of unravelling the genetic, molecular and epidemiological factors that contribute TNBC onset in WAA. Clinical data and surgical specimens were collected from Barbadian and Nigerian women and the Surveillance, Epidemiology, and End Results (SEER) database was used to collect clinical data for non-Hispanic White (NHW) and non-Hispanic Black (NHB) women with BCa in the US. Epidemiological analyses revealed that BCa incidence was higher in younger Barbadian women (15-59 years old) than in NHW and NHB, and TNBC prevalence among Barbadian women is ~2.5x higher than the prevalence in NHW. This suggests a possible genetic predisposition that could partially explain the high TNBC prevalence and aggressive clinical course in WAA globally. To investigate this, whole exome sequencing (WES) was conducted on DNA extracted from tumour and adjacent non-tumour sections from 31 Barbadian and Nigerian TNBC specimens. Preliminary analysis revealed mutations and copy number variations in well-known oncogenes

such as TP53 and other novel genes. Ongoing studies are focused on functional validation of a shortlist of novel genetic alterations in our WAA cohort to identify unique biomarkers driving aggressive TNBC in WAA.

ABSTRACT P56

DIKETOPYRROLOPYRROLE-BASED CONJUGATED POLYMER NANOPARTICLES TOWARD NEW THERANOSTICS FOR GLIOBLASTOMA MULTIFORME

Gage Mason[1*], F. Nadeem[1], D. Lubanska[1], M. DiPasquale[1], A. Malik[1], D. Marquardt[1], L. A. Porter[1], S. Rondeau-Gagne[1]
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Advent of nanotechnology introduced novel promising solutions in treatment of therapy- resistant conditions such as cancer. Glioblastoma multiforme (GBM) is one of the most aggressive types of cancer with median survival of only 15 months. Successful therapy of GBM is hampered by existence of treatment resistant populations of stem -like tumour initiating cells (TICs) and poor blood-brain barrier (BBB) drug penetration. Hence, therapies offering effective bioavailability and selective TIC targeting are of high demand. Here, we demonstrate the synthesis of spherical, diketopyrrolopyrrole (DPP)-based conjugated polymer nanoparticles (CPNs) with an average diameter of 109nm. The addition of fluorescein conjugated Hyaluronic Acid (HA) to the CPN design allows for selective targeting of CD44 positive TICs. We characterize the HA conjugated CPNs using transmission electron microscopy (TEM), dynamic light scattering (DLS) and small angle neutron scattering (SANS). This study evaluates the CPN uptake, selectivity and effects on glioma cells in vitro. We show that generated CPNs selectively target CD44 positive glioma cells, demonstrate concentration and cell cycle phase- dependent uptake and can affect glioma proliferation and metabolic activity, when administered at higher concentrations. Our data demonstrates that HA-conjugated CPNs, developed in this study, constitute a potential novel, selective therapeutic approach in designing treatment strategies against GBM.

ABSTRACT P57

PROMOTION OF THROMBIN ACTIVATABLE FIBRINOLYSIS INHIBITOR (TAFI) ACTIVATION IN THE TUMOUR MICROENVIRONMENT ATTENUATES BREAST CANCER METASTASIS IN VIVO.

Tasnim Reza[1*], Nivin Nystrom[1], John Ronald[1], Michael Boffa[1]
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Enhanced extracellular proteolysis is a key factor driving metastasis of breast and other cancers. An emerging regulator of extracellular proteolysis is thrombin-activatable fibrinolysis inhibitor (TAFI), a plasma zymogen that is activated by thrombin in complex with thrombomodulin (TM). Activated TAFI (TAFIa) downregulates pericellular plasminogen activation to plasmin. Plasmin can cleave many extracellular matrix components and activate proteases, thereby promoting metastasis of cancer cells. Indeed, we have found that the promotion of TAFI activation with TAFI-specific TM attenuates metastatic behaviours of breast cancer cells in vitro. We undertook the current study the evaluate the anti-metastatic potential of TAFI-specific TM in vivo, in a mouse xenotransplantation model. MDA-MB-231 cells were transduced with lentivirus encoding wild-type (wt) TM or TAFI-specific (F376A/M388A) TM along with tdTomato and firefly luciferase from a tricistronic expression system, or a control virus encoding only tdTomato and luciferase. Only expression of TAFI-specific TM reduced cell invasion in vitro. The transduced cells were injected into the mammary fat pad of 6-8-week-old NOD SCID gamma (NSG) mice, and the mice were subjected to bioluminescence imaging over 44 days. Although primary tumours grew at a similar rate in all cases, metastasis was observed in the axillary lymph nodes of mice implanted with cells expressing no TM or the wt TM, but not at all in mice implanted with cells expressing TAFI-specific TM. These experiments reveal an exceptional anti-metastatic activity of TAFI-specific TM, suggesting a novel approach to control metastatic breast cancer.

ABSTRACT P58

EVALUATION OF NATURAL EXTRACTS IN COMBINATION WITH CHEMOTHERAPIES ON NEURO- AND GLIOBLASTOMA

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It is estimated that approximately 211,400 Americans are living with malignant brain tumours (often neuro- or glioblastoma) with an average 5-year survival rate of only 36%. Furthermore, brain tumours are the leading cause of cancer-related death for children ages 0-19. In particular, glioblastoma is one of the most deadly, non-treatable forms of cancer. This highlights the need for the generation of more effective treatments.

Unfortunately, there are few chemotherapeutic options which have limited success. Worst of all, these treatments are very toxic with adverse side effects rendering them unsuitable for long-term use. As an alternative, various natural extracts already shown to be safe for consumption provide a potential therapeutic strategy to selectively target cancer cells. Specifically, Piper longum, also known as long pepper, has demonstrated anti-cancer activity while showing little to no effect on normal healthy cells. Indeed, we have evaluated the efficacy of ethanolic Long Pepper Extract (LPE) in neuroblastoma and glioblastoma cell lines and observed that it is extremely effective at triggering apoptosis in both. Most importantly, human glioblastoma cells that were extremely resistant to two of the most common chemotherapeutics (taxol and cisplatin) were very efficiently killed by LPE. As a result, LPE should be further investigated on neuroblastoma and glioblastoma cell lines to determine its mechanism of action and its interactions with commonly used chemotherapies and other natural extracts. In conclusion, our early results indicate that LPE could be an effective and non-toxic treatment for brain cancer.

ABSTRACT P59

EPIDERMAL GROWTH FACTOR RECEPTOR SIGNALING REQUIRES SPECIALIZED CLATHRIN-LABELED STRUCTURES AND NECESSARY ACCESSORY PROTEINS

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The Epidermal Growth Factor (EGF) Receptor (EGFR) controls many aspects of cell physiology, including growth, survival, migration and metabolism. Importantly, upregulation of EGFR contributes to proliferation and survival of many cancers. Upon binding its ligand, EGFR activates several signaling intermediates, and simultaneously is recruited to clathrin-labeled structures (CLSs). We previously uncovered that perturbation of clathrin, but not of receptor endocytosis, impairs EGF-stimulated activation of Akt signaling. Also, some EGFR signaling intermediates such as phosphorylated Gab1 are enriched with a subset of CLSs. We have also uncovered that the clathrin-binding protein target of myb-like 1 (TOM1L1) and the TOM1L1-binding Src-family kinase Fyn are recruited and enriched in a subset of CLSs. We proposed that CLSs have a direct role controlling EGFR signaling at the plasma membrane prior to receptor internalization, and the clathrin-accessory proteins Fyn and TOM1L1 are necessary in CLSs signaling. How CLSs and clathrin control EGFR signaling is not well understood, which we have examined here. We uncovered that the perturbations of TOM1L1 or Fyn phenocopy perturbations of clathrin heavy chain with regards to EGFR signaling. We generated stable cell lines for inducible controlled expression of various mutants of TOM1L1 and Fyn at near-endogenous levels to perform knockdown-rescue experiments to dissect the molecular mechanism by which TOM1L1 and Fyn contribute to EGFR signaling. Using total internal reflection fluorescence microscopy of cells expressing fluorescent clathrin, TOM1L1 and/or Fyn at near-endogenous levels, together with automated image analysis, we find that Fyn and TOM1L1 are selectively recruited to a subset of CLSs. CLSs that recruit TOM1L1 and Fyn exhibit unique properties, such as distinct lifetimes and Epsin recruitment. Silencing of TOM1L1 or Fyn impact dynamics of CLS harboring EGFR. These results suggest that TOM1L1 and Fyn are recruited to a distinct subpopulation of signaling-specialized CLSs that mediate certain aspects of receptor signaling directly at the plasma membrane.

ABSTRACT P60 **not participating in poster session*

MAGNETIC RESONANCE IMAGING ROUND METALLIC IMPLANTS USING SPRITE

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Magnetic Resonance Imaging (MRI) is an imaging modality that applies magnetic fields to non-invasively and safely image human anatomy. It has excellent soft tissue contrast that allows distinction between various types of tissues, including those afflicted by cancer. MRI requires a homogeneous static magnetic field, which cannot be maintained around a metal. According to the Canadian Joint Replacement Registry (CJJR), a combined total of over 137,000 joint replacement surgeries were performed in 2018-2019 in Canada, representing a 20% increase over the last five years. Areas of repair include the knees and hips and involve the insertion of metallic or ceramic implants. Due to their high susceptibility,

metal implants severely distort the local magnetic field, resulting in MRI image artifacts such as voids and distortions in the surrounding tissue. This impedes assessment of the region around the implant using traditional MRI methods. Single Point Ramped Imaging with T1 Enhancement (SPRITE) MRI techniques are pure phase encoding imaging sequences that are immune to local magnetic field inhomogeneities. We propose using SPRITE MRI to acquire high quality distortion-free images. The method has been demonstrated in phantom measurements and compared to standard techniques. SPRITE MRI can be applied to other systems that are challenging for traditional MRI.

ABSTRACT O61

THERAPEUTIC STRATEGIES TO TARGET AUTOPHAGY IN METASTATIC EPITHELIAL OVARIAN CANCER

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Epithelial ovarian cancer (EOC) metastasis is unique amongst carcinomas as it spreads by direct dissemination into the peritoneal cavity to seed secondary tumours. To this end, we have developed an in vitro spheroid model of EOC metastasis and exploited this system to discover novel pathobiological processes to be targeted as unique therapeutic vulnerabilities. One such process is autophagy, a well-conserved stress-induced mechanism to facilitate cell survival during starvation-like conditions. The expression and activity of phosphorylated AMP-activated protein kinase (AMPK) and Unc-51-like kinase 1 (ULK1) are significantly increased in EOC spheroids and this is correlated closely with autophagy induction. Indeed, AMPK and ULK1 knockdown directly reduced autophagy activation in EOC spheroids. Next, we applied pharmacologic approaches by treating EOC spheroids with two different small molecule inhibitors: the CAMKKbeta inhibitor, STO-609, that reduces AMPK activity; and the ULK1/2 inhibitor, MRT-68921. Both of these inhibitors potently affected multiple autophagy markers: LC3 processing and p62/SQSTM1 expression were reduced; and the green:red fluorescence ratio of the mCherry-eGFP-LC3B reporter was increased. Multiple established EOC cell line spheroids treated with these compounds, and patient-derived spheroids with MRT-68921, significantly reduced cell viability. Working recently with the OICR Drug Discovery Program, we determined the toxicology and pharmacology of MRT-68921 in female NOD/SCID mice; we will now test its utility in treating orthotopic metastasis xenografts. We predict that an effective therapeutic regimen of MRT-68921 administration will reduce metastatic progression by blocking ULK1-mediated autophagy as a crucial cell survival mechanism in advanced ovarian cancer.

ABSTRACT O62

TRANSCRIPTOMIC CHARACTERIZATION OF LUNG CANCER HETEROGENEITY USING SCRNA-SEQUENCING

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Background: Lung cancer is the leading cause of cancer-related deaths globally. To improve survivorship, a better understanding of the molecular heterogeneity of lung cancer is required in order to design unique precision medicines to treat an individual's cancer. We applied a single-cell RNA sequencing (scRNA-seq) approach to profile differential gene expression to identify unique biomarkers and key genes associated with the heterogeneity of lung carcinogenesis.
Methods: We constructed 3'-end cDNA libraries from lung cancer cell lines using the Fluidigm C1 and sequenced the libraries using illumina Nextseq. t-SNE and heat map clustering analyses were performed on normalized gene expression values. Fold change values of DEGs were validated using a qRT-PCR. Gene expression maps and PPIN were created using cell line- or cluster-specifically expressed genes.
Results: Over 82.9 million 3'-end cDNA sequence reads from 1,441 single cells of the four cell lines were successfully aligned to 24,424 genes on human reference genome. t-SNE clustering analysis classified the single-cells into four distinct groups that were composed of single-cells more than two cell lines. Heat-map clustering analysis revealed 1,970 genes that were group-specifically expressed. Following a comparison of normalized gene expression values, we detected a total of 2,735 DEGs showing ≥|2| fold change difference among four clustered groups. Inflammatory chemokines were highly expressed in groups containing single cells from A549, H460 and Calu3 cell lines compared with a group dominantly containing H1299 single cells.

Conclusion: We successfully applied scRNA-seq to characterize lung cancer single-cell transcriptomes. The antigens we detected by clustering analyses are key genes associated with the heterogeneity of lung cancer. Our goal is to improve lung cancer survivorship and quality life for lung cancer patients through scRNA-seq guided precision medicines.

ABSTRACT O63

PRECLINICAL IMAGING OF SPONTANEOUSLY METASTASIZING BREAST CANCER CELLS IN MICE

Nivin Nyström [1*], Timothy J Scholl[1], John A. Ronald[1]

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Preclinical animal models are invaluable tools, as they offer a wholistic approach relative to in vitro studies, and more variable control than clinical trials. Yet, although metastasis accounts for the vast majority of cancer-related deaths, 75% of recent preclinical studies did not examine metastasis at all, largely due to difficulty in identifying metastases (Gengenbacher Nature Reviews Cancer 2017). We therefore sought to develop a tool that enables tracking of spontaneously metastasizing cancer cells in animal models. In this study, we establish a reporter gene called Organic anion-transporting polypeptide 1b3 (Oatp1b3) capable of taking up a paramagnetic contrast agent called Gd-EOB-DTPA. Our objective was to assess Oatp1b3 for whole-body tracking of cancer cells in a spontaneous metastasis model on magnetic resonance imaging (MRI). Breast cancer cells were engineered to express luciferase for bioluminescence imaging (BLI) and Oatp1b3 for MRI, and implanted into nod scid gamma mice (n=10). We successfully imaged the stepwise progression of the cancer as it spread from the primary tumour, through the lymphatic nodal system, and the emergence of numerous micro-metastases in the lungs. Importantly, our system operates at high sensitivity, enabling detection of small populations of cells (~1000), and at high resolutions (0.1 mm-cubed), allowing for delineation of lesion boundaries on MRI, that are not resolvable on BLI. Oatp1b3 fulfills a long-standing gap in our ability to accurately study metastatic disease over time, and offers a path towards deep-tissue tracking of any Oatp1b3-engineered cell type with combined high resolution, sensitivity, 3D spatial information, and surrounding anatomical context.

ABSTRACT O64

FROM MOLECULAR MECHANISM TO DRUG CANDIDATE, HOW WE STOP THE VICIOUS CYCLE OF BREAST CANCER BONE METASTASIS

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Breast cancer metastasizes to the bone in over 70% of advanced breast cancer cases, and results in high morbidity and death. Currently, 30-50% patients relapse on current bone targeted treatments, and better treatments are required. Breast cancer bone metastasis produces predominantly lytic bone disease, characterized by a vicious cycle initiated by the secretion of factors from the breast cancer cells at the site of bone metastasis that stimulates breakdown of bone (lysis) and the release of calcium and growth factors that stimulate breast cancer cell proliferation. My lab discovered, using quantitative immunohistochemistry, that high prolactin (PRL)-receptor (PRLR) levels in the primary breast tumor were associated with a shorter time to bone metastasis. We identified the PRLR on circulating tumor cells of advanced breast cancer patients and also in rare secondary breast cancer metastases of the bone. The molecular mechanism we discovered is that PRL stimulates breast cancer cells to secrete factors such as sonic hedgehog and novel factors that induce osteoclast differentiation and bone lysis. Micro-computed tomography demonstrates the damaging impact of PRL in the bone's tumour microenvironment. We screened over 1700 FDA-approved pharmaceuticals to inhibit PRL-induced breast cancer cell-mediated induction of osteoclasts (bone breaking down cells) finding a high-quality candidate with in vivo impact on lytic bone metastasis. The timeline for making clinical impact with a repurposed pharmaceutical is much shorter than with de novo drug development, and we hope to have an impact on breast cancer patients with metastasis to the bone.

ABSTRACT O65

TARGETED CRISPR/CAS9 ENGINEERING FOR MULTI-MODALITY REPORTER GENE-BASED CELL TRACKING.

John Kelly[1*], Moe Saee-Marand[1], Nivin Nyström[1], Melissa Evans[1], Yuanxin Chen[1], Francisco Martinez[1], Amanda Hamilton[1], John Ronald[1]
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Imaging reporter genes can provide longitudinal whole-subject information on the biodistribution, growth and survival of engineered cells in pre-clinical disease models and clinical cellular therapies. Often, cells are engineered with randomly-integrating vectors, which may alter normal cell function or lead to oncogenesis. Targeted editing of cells at a safe harbor locus may overcome some of these safety concerns. We reported the first CRISPR/Cas9 homology-directed repair (HDR) system targeted to the AAVS1 safe harbor locus for in vivo reporter tracking of edited cells, yet the efficiency of this system was less than ideal. Here, we built Homology Independent Targeted Integration (HITI) CRISPR/Cas9 minicircle donors for precise safe harbor-targeted knock-in of fluorescence (tdTomato), bioluminescence (Luc2), and MRI (Oatp1a1) reporter genes. Our results showed greater knock-in efficiency for HITI minicircles over HDR minicircles in several human cell lines. HITI-engineered clones demonstrated functional fluorescence and bioluminescence reporter activity as well as significant Oatp1a1-mediated uptake and retention of the clinically-used MRI agent Gd-EOB-DTPA. As few as 106 cells in a 50 ul subcutaneous injection was detected in vivo and cell pellets consisting of only 10% HITI-engineered cells were visible above background in vitro with the Oatp1a1 MRI reporter system. In pre-clinical cancer models, contrast-enhanced MRI improved the conspicuity of both sub-cutaneous and metastatic Oatp1a1-expressing tumours prior to them being palpable or even readily visible on pre-contrast images. Our work demonstrates the first CRISPR/Cas9 HITI system for knock-in of large reporter gene constructs at a safe harbor locus, enabling multi-modal longitudinal in vivo imaging of cells.

ABSTRACT O66

PROPHYLATIC USE OF MEPITEL FILM FOR BREAST CANCER PATIENTS UNDERGOING CHESTWALL IRRADIATION: A SINGLE INSTITUTION EXPERIENCE

Laura D'Alimonte[1*], Fatima Oshin[1]
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Purpose: Women receiving radiation therapy (RT) treatment for a diagnosis of breast cancer can experience significant skin toxicities in both the acute and late phases. Prophylactic use of mepitel film has been shown to reduce the severity of skin reactions however, there has been limited adoption into RT practice. We report a skin toxicity analysis of the prophylactic use of mepitel film for breast cancer patients undergoing chestwall and supraclavicular nodes irradiation.

Methods: A retrospective chart audit was conducted on 127 breast cancer patients treated between October 1, 2016 and December 31, 2019; 114 charts contributed data for analysis. Mepitel film application occurred at first fraction, either prior to or immediately after treatment delivery. Application of the film was applied to cover the entire treatment fields with the exception of the ipsilateral neck fold at the supraclavicular field and in the axillary fold of the tangent fields due to lack of adhesion. Skin assessment was completed on Day 0, Day 25, and the fraction at which the patient presented with radiation induced skin changes was collected. Toxicity was graded using Radiation Oncology Toxicity Grading (RTOG) acute skin criteria.

Results: All patients were treated with tangential fields to the chestwall (50Gy in 25 fractions) and parallel opposed fields to the supraclavicular region (45Gy in 25 fractions). Almost all patients (94%) were treated with 0.5 cm supraflab bolus on their chestwall field. 43% patients presented with any form of radiation dermatitis changes at the end of their week 3 of treatment. Grade 2 toxicity rate was seen in 64% of patients. Of these, 22% and 26% (chestwall and supraclavicular region, respectively) had Grade 2 toxicity seen in mepitel film covered treatment areas. Conversely, Grade 2 toxicity occurred in 31% and 49% patients with mepitel uncovered areas ipsilateral neck fold and ipsilateral axillary fold, respectively. 32% patients had grade 2 toxicity in only one of these areas and 12% patients had grade 2 toxicity in all four of these areas. Only 2.6% patients progressed to grade 3 toxicity by the end of their treatment. Although, all 3 patients had grade 3 toxicity in all four regions of the treatment area

Conclusions: Although moist desquamation was experienced at high frequency to the areas of high friction such as, neck fold and axilla in the absence of mepitel film, our findings support that chestwall areas experienced a low frequency of moist desquamation with the use of prophylactic mepitel film. In addition, there was a reduced severity in toxicity (progression into grade 3) in our high-risk population. Further audit of our experience is required to support the continued use of prophylactic use mepitel film to reduce moist desquamation incidence and intensity and consequently, improve patients' QOL while undergoing radiotherapy.

ABSTRACT O67

CANADIAN CLINICAL TRIALS NAVIGATOR: IMPROVING ACCESS TO CLINICAL TRIALS

Caroline Hamm[1*], Youshaa El-abed[2], Lee Mcgrath[3], Krista Naccarato[2], Steven Sundquist[4], Soham Ashtekar[2], Janet Dansey[4], Dora Cavallo-Medved[1]

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Clinical trials, although academically accepted as the most effective treatment available for cancer patients, are dramatically underused in North America. Poor accrual to clinical trials remains a significant problem throughout North America.
A clinical-trials navigator (CTN) program was piloted in 2019 in Windsor, Ontario, servicing all of Canada. Patients and/or their health care professionals could apply to have the CTN search for a cancer clinical trial, analyzing the patient's current status and disease, and provide a list of potential clinical trials.
One hundred and eighteen patients accessed this program between March 2019 and February 2020. As seen in previous CTN programs, the more-rare disease sites: glioblastoma multiforme, pancreatic cancer and sarcoma were over-represented.
We were able to enroll an extra 5% of patients referred, onto treatment clinical trials. Our study identified similar issues as previous trials. Eligibility criteria excluded 38% of those looking for trials. Twenty per cent of this motivated population had no trial available for them. Surprisingly, 21% of patients had a decline in their health and died before they could be referred or enrolled onto a clinical trial. The median time from referral to death was 109 days in those that passed. The reasons for these late referrals to clinical trials need to be fully understood, as this referral pattern will continue to hamper the growth of clinical trials.
The CTN program also increased referrals to clinical trials outside of the CTN program at the pilot site. The reason for this change is under investigation.

ABSTRACT O68

RETHINKING PROSTATE CANCER IMAGING

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Researchers in the prostate field have been leaders in demonstrating the value of multidisciplinary research coming together to find new innovative solutions that benefit patients. As such, the detection and treatment of prostate cancer continues to evolve and improve mortality rates for more than 23,000 Canadians each year. Despite the successes, treatment resistant forms of the disease continue to emerge requiring new approaches and ideas. Neuroendocrine Prostate Cancer (NEPC) is one example of a high-mortality, treatment-resistant form of the disease that requires more rapid and effective approaches to detect and treat.

Prostate-specific membrane antigen (PSMA) is highly overexpressed in most prostate cancers and is clinically visualized using PSMA-specific probes incorporating Glutamate-Ureido-Lysine (GUL). PSMA-PET imaging is rapidly approaching standard of care, dramatically improving the visualization of metastatic prostate lesions. Work in our lab has determined that NEPC expresses low levels of PSMA and androgen receptor and high levels of select glucose transporters and hexokinases. Using bioinformatic approaches along with *in vivo* and *in vitro* assays our data supports that NEPC tumours may be very amendable to imaging by ^{18}F-FDG, perhaps being more FDG avid than PSMA-PET. Interestingly, clinical measurements using GUL-based probes have reported the ability to detect NEPC metastatic tumours with PSMA-PET. In collaboration with chemistry collaborators we have identified that NEPC upregulates a PSMA-like aminopeptidase and a series of metabrotropic glutamate receptors that show affinity for PSMA. This work presents opportunities to develop novel targets to detect and treat NEPC.

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Thank you to the WCRG Conference Organizing Committee and our group of volunteers for contributing their time, ideas, and enthusiasm to this conference. Their commitment and efforts made all of this possible

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Thank you to the CUREs class volunteers. The Cancer Undergraduate Research Education class was first offered by the Faculty of Science at the University of Windsor in Fall 2018. This course exposes students to the importance of multidisciplinary research and brings them together into collaborative teams to design and implement tools to communicate cancer research to stakeholders. Students discuss ethical fundraising and marketing of research and develop projects that are shared with the community on a Cancer Education Day. It is the hands on learning experience where the students' ideas will directly contribute to moving cancer research forward in Windsor-Essex.

THANK YOU!

Thank you to all the registrants, speakers, students, sponsors, and community members for joining us for WCRG's 5th Biennial International Cancer Research Conference – Virtual Edition. It was through your support and participation that we were able to transition the conference to a virtual format this year. We look forward to connecting at WCRG's 6th Biennial International Cancer Research Conference in 2022!

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