WELCOME TO LONDON HEALTH RESEARCH DAY 2018

Welcome to the seventh annual London Health Research Day, the region’s premier research showcase event. Groundbreaking work from tomorrow’s leaders in health research will be highlighted today, with hundreds of abstracts submitted from top learners.

London Health Research Day 2018 features a broad range of innovative research projects. The award competitions include the top 40 abstracts selected for platform presentations and over 350 abstracts selected for poster presentations.

We are pleased to welcome back presenters joining us from the Faculty of Health Sciences who have become a valued part of this event. We look forward to learning more about the research they are conducting.

This year’s event was preceded by a forum on the evening of May 9 – Exchange: A London Health Research Day Forum on Diversity & Inclusivity. Exchange was a chance to foster conversations around equity, diversity and inclusivity in the careers of health researchers. This is a topic of critical importance to our learners as the next generation of health scientists.

Dr. Janet Smylie, an international leader in the field of Indigenous health and health research, was one of many presenters at Exchange. Dr. Smylie will also join us at today’s lunch as our 2018 keynote speaker, presented as part of The Lucille & Norton Wolf Health Research Lecture Series. Dr. Smylie holds a Canadian Institutes of Health Research (CIHR) Applied Public Health Research Chair in Indigenous Health Knowledge and Information and is Director of the Well Living House Applied Research Centre for Indigenous Infant, Child and Family Health. She will deliver a presentation titled “Our Health Counts: Community Partnered Approaches to Indigenous Health Assessment and Response.”

We thank the Bernard & Norton Wolf Family Foundation for their continued support of medical education and research through the Lecture Series. We are also grateful to the Wolf family for supporting Exchange and presenting the third annual Lucille & Norton Wolf London Health Research Day Trainee Publication Awards. Up to four awards will be presented to the top-scoring trainee peer-reviewed publications that have appeared in press during 2017.

London Health Research Day 2018 will also include a special presentation from Dr. Alastair Buchan, a world-renowned neurologist and stroke medicine researcher at the University of Oxford. Dr. Buchan’s presentation will be titled “Precision Stroke Medicine and the Role of Translational Clinical Research.”

The day will conclude with the Networking and Awards Reception where we will recognize the event’s top presenters. Prizes for those learners who complete an LHRD Poster Passport card will be also announced.

We extend a special note of gratitude to the committed volunteers, academic judges, corporate sponsors, and dedicated faculty and staff for contributing to the success of this event.

London Health Research Day unites young researchers from across our city and showcases London, Ontario as a leader for innovation, education and collaboration in the health sciences. We hope you will enjoy the day’s offerings and engage in the brilliance offered by all the participating trainees.

David J. Hill, DPhil, FCAHS
Scientific Director, Lawson Health Research Institute
Integrated Vice President, Research
London Health Sciences Centre and St. Joseph’s Health Care London

Michael J. Strong, MD, FRCP(C), FAAN, FCAHS
Dean, Schulich School of Medicine & Dentistry
Distinguished University Professor, Western University
AGENDA: LONDON HEALTH RESEARCH DAY 2018

Thursday, May 10, 2018

7:30 a.m. Registration Opens
Main Floor Foyer, First Floor
*Registration will remain open throughout the day

8:30 - 10:15 a.m. Poster Presentations – Morning Session
Ballroom A, Second Floor
*Morning posters must be removed by 12:00 p.m.

10:15 - 10:30 a.m. Coffee Break
Ballroom Foyer, Second Floor

10:30 - 11:45 a.m. Feature Platform Presentations – Morning Session
First Floor: Salon B, Salon B1, Salon D, Salon E

11:45 a.m - 1:30 p.m. Lunch
Ballroom B, Second Floor
*Guest Registration available (Main Floor Foyer, First Floor)

The Lucille & Norton Wolf Health Research Lecture Series
Featuring Dr. Janet Smylie
“Our Health Counts: Community Partnered Approaches to Indigenous Health Assessment and Response”

1:30 - 3:15 p.m. Poster Presentations – Afternoon Session
Ballroom A, Second Floor
*Afternoon posters must be removed by 5:45 p.m.

3:15 - 3:30 p.m. Coffee Break
Ballroom Foyer, Second Floor

3:30 - 4:45 p.m. Feature Platform Presentations – Afternoon Session
First Floor: Salon B, Salon B1, Salon D, Salon E

4:45 - 5:30 p.m. Special Presentation by Dr. Alastair Buchan
“Precision Stroke Medicine and the Role of Translational Clinical Research”
Ballroom B, Second Floor

5:30 - 6:15 p.m. Networking and Awards Reception
Ballroom B, Second Floor
# TABLE OF CONTENTS

6  THE LUCILLE & NORTON WOLF HEALTH RESEARCH LECTURE SERIES

7  SPECIAL PRESENTATION BY DR. ALASTAIR BUCHAN

8  POSTER PRESENTATIONS – MORNING SESSION – BALLROOM A, SECOND FLOOR

9  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

10 Advancing health services provision and health policy

11 Detection, screening and diagnosis of health and disease

13 Determinants of health

13 Early life programming and development

14 Mechanisms of disease

18 Populations, public health and education

19 Prevention of diseases and health conditions and promotion of well-being

20 FEATURE PLATFORM PRESENTATIONS – MORNING SESSION – FIRST FLOOR

21 10:30 - 10:45 a.m. presentations – Salon B, Salon B1, Salon D, Salon E

25 10:45 - 11:00 a.m. presentations – Salon B, Salon B1, Salon D, Salon E

29 11:00 - 11:15 a.m. presentations – Salon B, Salon B1, Salon D, Salon E

33 11:15 - 11:30 a.m. presentations – Salon B, Salon B1, Salon D, Salon E

37 11:30 - 11:45 a.m. presentations – Salon B, Salon B1, Salon D, Salon E

41 POSTER PRESENTATIONS – AFTERNOON SESSION – BALLROOM B, SECOND FLOOR

42 Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

43 Advancing health services provision and health policy

44 Detection, screening and diagnosis of health and disease

46 Determinants of health

47 Early life programming and development

47 Mechanisms of disease

51 Populations, public health and education

52 Prevention of diseases and health conditions and promotion of well-being

53 FEATURE PLATFORM PRESENTATIONS – AFTERNOON SESSION – FIRST FLOOR

54 3:30 - 3:45 p.m. presentations – Salon B, Salon B1, Salon D, Salon E

58 3:45 - 4:00 p.m. presentations – Salon B, Salon B1, Salon D, Salon E

62 4:00 - 4:15 p.m. presentations – Salon B, Salon B1, Salon D, Salon E

66 4:15 - 4:30 p.m. presentations – Salon B, Salon B1, Salon D, Salon E

70 4:30 - 4:45 p.m. presentations – Salon B, Salon B1, Salon D, Salon E

74 APPENDIX A – POSTER PRESENTATIONS OVERVIEW

81 APPENDIX B – PLATFORM PRESENTATIONS OVERVIEW
THE LUCILLE & NORTON WOLF
HEALTH RESEARCH LECTURE SERIES

The Lucille & Norton Wolf Health Research Lecture Series is the keynote lecture presented during the luncheon at London Health Research Day. The Series has been established thanks to the generosity of the Bernard & Norton Wolf Family Foundation.

The Bernard & Norton Wolf Family Foundation

With a commitment to philanthropy, Norton Wolf created The Bernard & Norton Wolf Family Foundation which supports education, the arts, and medical research and treatment. In London, the Foundation has contributed to such projects as the Wolf Performance Hall, Museum London Sculpture Garden, Robarts Research Institute, Western University, Norton and Lucille Wolf Breast Care Centre at St. Joseph’s Health Care London, Pre- and Peri-Operative Clinics at LHSC’s University Hospital and Wolf Orthopedic BioMechanical Lab.

Norton Wolf passed away in March of 2015. The Family Foundation he established continues to give shape to his personal vision of philanthropy and community support.

Dr. Janet Smylie
2018 Keynote Speaker

“Our Health Counts: Community Partnered Approaches to Indigenous Health Assessment and Response”

Dr. Janet Smylie is a family physician and public health researcher. She currently works as a research scientist in Indigenous health at St. Michael’s Hospital, Centre for Urban Health Solutions (CUHS), where she directs the Well Living House Applied Research Centre for Indigenous Infant, Child and Family Health. Her primary academic appointment is as a Professor in the Dalla Lana School of Public Health, University of Toronto. She maintains a part-time clinical practice with Inner City Health Associates at Seventh Generation Midwives Toronto.

Dr. Smylie has practised and taught family medicine in a variety of Aboriginal communities, both urban and rural. She is a member of the Métis Nation of Ontario, with Métis roots in the prairies.

Her research interests are focused in the area of addressing the health inequities that challenge Indigenous infants, children and their families through applied health services research. Dr. Smylie currently leads multiple research projects in partnership with First Nations, Inuit and Métis communities/organizations. She holds a Canadian Institutes of Health Research (CIHR) Applied Public Health Research Chair in Indigenous Health Knowledge and Information and was honoured with a National Aboriginal Achievement (Indspire) Award in Health in 2012. A Métis woman, Dr. Smylie acknowledges her family, teachers and lodge.

The Lucille & Norton Wolf London Health Research Day Trainee Publication Awards

With thanks to the generosity of the Bernard & Norton Wolf Family Foundation, and the personal interest of Lucille and Norton Wolf, we are pleased to present the annual Lucille & Norton Wolf London Health Research Day Trainee Publication Awards. These awards were created to recognize outstanding research discoveries by graduate trainees and postdoctoral scholars who are participating in London Health Research Day.

Up to four awards are provided to the top-scoring trainee peer-reviewed publications that have appeared in press from January 1 to December 31, 2017. Recipients will receive their award at The Lucille & Norton Wolf Health Research Lecture Series.
SPECIAL PRESENTATION BY DR. ALASTAIR BUCHAN

London Health Research Day 2018 will feature a special presentation from Dr. Alastair Buchan, world-renowned neurologist and stroke medicine researcher from the University of Oxford.

“PRECISION STROKE MEDICINE AND THE ROLE OF TRANSLATIONAL CLINICAL RESEARCH”

4:45 - 5:30 p.m.
Ballroom B, Second Floor

Dr. Alastair Buchan

Professor Buchan is currently Pro-Vice-Chancellor and the Head of Brexit Strategy at the University of Oxford. A neurologist and neuroscientist in stroke medicine and neurology since 1988, he is now the George Pickering Professor of Stroke Medicine in the Radcliffe Department of Medicine at the John Radcliffe Hospital where he is an honorary consultant neurologist. He is a Professorial Fellow in Medicine at Corpus Christi College, Oxford.

He was educated at Repton School, Cambridge, Oxford and Harvard. He undertook clinical medical training at Oxford and neurology specialist training at Oxford; the Schulich School of Medicine & Dentistry, Western University in London, Ontario; and Cornell University Medical College in New York. Since completing his training, he has worked at Schulich Medicine & Dentistry, Western University, University of Ottawa, and University of Calgary. He has been a consultant neurologist at London Health Sciences Centre’s University Hospital, the Civic Hospital in Ottawa, and the Foothills Hospital in Calgary where he held the Heart and Stroke Foundation Professorship in Stroke.

In Oxford since 2005, he has established the Acute Stroke Programme and has been the Translational Research Director for the UK Stroke Research Network. He was the founding Director of the Acute Vascular Imaging Centre, and the Director of the Oxford NIHR Biomedical Research Centre. He was appointed Head of the Medical Sciences Division of the University of Oxford in December 2007 and served as Dean of Medicine from October 2008 to October 2017. He is a Fellow of the Academy of Medical Sciences (FMedSci), a senior investigator of the NIHR and holds a DSc from the University of Oxford.

Dr. Buchan sits on the board of the Berlin Health Institute and Charité Hospital Stroke Programme in Berlin, Germany. He is the Senior Member of the Osler House Club in Oxford and a member of the Athenaeum Club in London, England. He is currently a Governor of Repton School. He was awarded an honorary degree of Laws from the University of Calgary in May 2009.

Professor Buchan’s research interests are in thrombolysis and neuroprotection for stroke. His laboratory focuses on selective neuronal vulnerability and understanding mechanisms that might lead to neuroprotection in the clinic. His clinical research interests are in managing hyperacute stroke and effective prevention following transient ischemic attack (TIA). He has run a number of trials including CASES and FASTER and is the inventor of ASPECTS.

Professor Buchan teaches students neuroscience, neurology and stroke. He supervises medical students, trainees and fellows. He also lectures to departments in the faculty of Physiological Sciences about neuroprotection and stroke. He supervises DPhil students in his laboratory.
### POSTER PRESENTATIONS
#### MORNING SESSION
8:30 - 10:15 a.m.
Ballroom A, Second Floor

<table>
<thead>
<tr>
<th>Category</th>
<th>Poster Number</th>
<th>Page Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>1 - 28</td>
<td>9 - 10</td>
</tr>
<tr>
<td>Advancing health services provision and health policy</td>
<td>29 - 37</td>
<td>10 - 11</td>
</tr>
<tr>
<td>Detection, screening and diagnosis of health and disease</td>
<td>38 - 71</td>
<td>11 - 13</td>
</tr>
<tr>
<td>Determinants of health</td>
<td>72 - 79</td>
<td>13</td>
</tr>
<tr>
<td>Early life programming and development</td>
<td>80 - 89</td>
<td>13 - 14</td>
</tr>
<tr>
<td>Mechanisms of disease</td>
<td>90 - 152</td>
<td>14 - 18</td>
</tr>
<tr>
<td>Populations, public health and education</td>
<td>153 - 162</td>
<td>18</td>
</tr>
<tr>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>163 - 176</td>
<td>19</td>
</tr>
</tbody>
</table>
ADVANCES IN STRUCTURAL AND PHYSIOLOGICAL TREATMENT OF DISEASE AND THERAPEUTIC INTERVENTION (INCLUDES SURGERY AND DRUGS)

Poster Number: 1
Name: Brandon Baer
Degree: PhD Candidate
Abstract Title: Using the wet bridge transfer system to assess exogenous surfactant as a pulmonary drug delivery vehicle
Supervisor(s): C. Yamashita, R. Veldhuizen

Poster Number: 2
Name: Angela Chang
Degree: Research Assistant/Associate
Abstract Title: Silica nanoparticles as a novel ophthalmic drug delivery system
Supervisor(s): C. Hutnik, W. Wan

Poster Number: 3
Name: Joshua Choi
Degree: PhD Candidate
Abstract Title: Glycolipid stimulation of invariant NKT cells mobilizes precursors of mature NK cells and potentiates their participation in immune surveillance against metastatic cancer
Supervisor(s): S.M. Haeryfar

Poster Number: 4
Name: Jim Denstedt
Degree: Medical Student
Abstract Title: The functional response of Tenon’s capsule fibroblast cells to treatment with fibrotic growth factors in a 3D collagen lattice
Supervisor(s): C. Hutnik

Poster Number: 5
Name: Charles-Antoine Dion
Degree: MSc Candidate
Abstract Title: A novel approach to revision total knee arthroplasty using 3D printed titanium augments: A biomechanical cadaveric study
Supervisor(s): J. Howard, M. Teeter, R. Willing, B. Lanting

Poster Number: 6
Name: Mohammadreza Faieghi
Degree: PhD Candidate
Abstract Title: A computationally efficient algorithm to construct micro-level finite element models
Supervisor(s): R. Eagleson, R. Tutunea-Fatan

Poster Number: 7
Name: Ailsa Gan
Degree: Medical Student
Abstract Title: Nerve sparing in retroperitoneal lymph node dissection for testicular cancer prevents post-operative retrograde ejaculation: A Canadian perspective
Supervisor(s): N. Power

Poster Number: 8
Name: Sherain Harricharan
Degree: PhD Candidate
Abstract Title: Overlapping frontoparietal networks in response to oculomotion during traumatic autobiographical memory recall
Supervisor(s): R. Lanius

Poster Number: 9
Name: Daphne Hui
Degree: MSc Candidate
Abstract Title: Investigating the utility of current steering for treating parkinsonian gait
Supervisor(s): M. Jog

Poster Number: 10
Name: Kenneth Ip
Degree: MSc Candidate
Abstract Title: Development of an in vitro intrinsically loaded temporomandibular force simulator and fast computational model based on method of external approximations
Supervisor(s): L. Ferreira, C. Moore

Poster Number: 11
Name: Kaitlyn Jacobs
Degree: MSc Candidate
Abstract Title: Sex differences in heart rate response to isometric handgrip exercise with concurrent contralateral forearm somatosensory stimulation
Supervisor(s): K. Shoemaker

Poster Number: 12
Name: Kunmo Kim
Degree: MSc Candidate
Abstract Title: Attenuation of sepsis in mice models by annexin A5 and mutant annexin A5-C316S
Supervisor(s): Q. Feng

Poster Number: 13
Name: Cecilia Kramar
Degree: Postdoctoral Scholar
Abstract Title: The role of astrocytes on memory
Supervisor(s): L. Saksida, T. Bussey

Poster Number: 14
Name: Chris Leclerc
Degree: MSc Candidate
Abstract Title: Formation of a vascular regenerative microenvironment within implantable human decellularized adipose tissue bioscaffolds
Supervisor(s): D. Hess, L. Flynn

Poster Number: 15
Name: Shabna Mohideen
Degree: MSc Candidate
Abstract Title: Use of current steering in deep brain stimulation of the subthalamic nucleus to improve appendicular motor symptoms
Supervisor(s): M. Jog
**Poster Number: 16**  
Name: Hareem Nisar  
Degree: MSc Candidate  
**Abstract Title:** Ultrasound image registration for intra-cardiac interventions  
**Supervisor(s):** T. Peters

**Poster Number: 17**  
Name: Nivin Nyström  
Degree: PhD Candidate  
**Abstract Title:** A novel MRI reporter gene for tracking breast cancer growth in preclinical animal models  
**Supervisor(s):** J. Ronald, T. Scholl

**Poster Number: 18**  
Name: Aanal Patel  
Degree: Research Assistant/Associate  
**Abstract Title:** Effect of GDF15 on renal ischemia injury during kidney transplant  
**Supervisor(s):** X. Zheng

**Poster Number: 19**  
Name: Alex Peidl  
Degree: PhD Candidate  
**Abstract Title:** CCN3: A novel antifibrotic therapy?  
**Supervisor(s):** A. Leask

**Poster Number: 20**  
Name: Alireza Rohani  
Degree: Postdoctoral Scholar  
**Abstract Title:** Optimal parameters for cochlear implant visualization using in-line synchrotron-radiation phase-contrast imaging  
**Supervisor(s):** H. Ladak, S. Agrawal

**Poster Number: 21**  
Name: Mayank Sharma  
Degree: MSc Candidate  
**Abstract Title:** Experimental measurement of the cutting forces generated during glenoid reaming procedure  
**Supervisor(s):** L. Ferreira, O. Tutunea-Fatan

**Poster Number: 22**  
Name: Naveed Siddiqui  
Degree: MSc Candidate  
**Abstract Title:** The conserved mitochondrial calcium uniporter regulator-1 (MCUR1) matrix domain is highly alpha-helical homotrimer sensitive to divalent cations  
**Supervisor(s):** P. Stathopoulos

**Poster Number: 23**  
Name: Christopher Smith  
Degree: MSc Candidate  
**Abstract Title:** Do dominant intraprostatic lesions receive sufficient dose in high dose rate brachytherapy?  
**Supervisor(s):** A. Ward, D. Hoover

**Poster Number: 24**  
Name: Lauren Solomon  
Degree: Postdoctoral Scholar  
**Abstract Title:** Estrogen effects on Th2 cell phenotype: Key to severe asthma in women?  
**Supervisor(s):** L. Cameron

**Poster Number: 25**  
Name: Emilie Woehrle  
Degree: MSc Candidate  
**Abstract Title:** Posture modifies neuro-cardiac heart rate responses at the onset of moderate intensity isometric handgrip exercise  
**Supervisor(s):** J. Shoemaker, J. P. Dickey

**Poster Number: 26**  
Name: Wenyao Xia  
Degree: PhD Candidate  
**Abstract Title:** Specular highlight removal for endoscopic images  
**Supervisor(s):** T. Peters

**Poster Number: 27**  
Name: Guangju Zhao  
Degree: Postdoctoral Scholar  
**Abstract Title:** Partial depletion of regulatory T cells prevents secondary pseudomonas aeruginosa infection post sepsis by enhancing host inflammatory response  
**Supervisor(s):** T. Peng

**Poster Number: 28**  
Name: Kevin Zhou  
Degree: Research Assistant/Associate  
**Abstract Title:** Controlled repopulation of a decellularized murine lung scaffold by malignant human lung epithelial cells targeted with a doxycycline-inducible regulatory construct  
**Supervisor(s):** P. Duchesneau, M. Ahmadipour, C. Monetti, A. Nagy, G. Karoubi, T. Waddell

ADVANCING HEALTH SERVICES PROVISION AND HEALTH POLICY

**Poster Number: 29**  
Name: Cadence Baker  
Degree: MSc Candidate  
**Abstract Title:** Frequency response of magnetophosphene perception in humans exposed to extremely low frequency magnetic fields and associated occipital cortex electroencephalography  
**Supervisor(s):** A. Legros

**Poster Number: 30**  
Name: Sierra Barrett  
Degree: Medical Student  
**Abstract Title:** An evaluation of utilization patterns and appropriateness of laboratory tests among new referrals to rheumatologists: Choosing Unwisely!  
**Supervisor(s):** J. Pope

**Poster Number: 31**  
Name: Nicolas Bouisset  
Degree: PhD Candidate  
**Abstract Title:** Is postural control sensitive to time varying magnetic fields up to 100 mT?  
**Supervisor(s):** A. Legros
Poster Number: 32
Name: Sarah Janssen
Degree: PhD Candidate
Abstract Title: A systematic review of exercise prescription for community-dwelling adults diagnosed with Type 2 diabetes
Supervisor(s): D. Connelly

Poster Number: 33
Name: Samuel Jumbo
Degree: MSc Candidate
Abstract Title: Burden experience of formal and informal caregivers of older adults with hip fracture in southern Nigeria
Supervisor(s): J. MacDermid

Poster Number: 34
Name: Faiza Rab
Degree: PhD Candidate
Abstract Title: Effectiveness of strategies for implementing childhood vaccination programs in fragile countries
Supervisor(s): A. Thind

Poster Number: 35
Name: Sadiq Raji
Degree: PhD Candidate
Abstract Title: EMR integration: Physician perspectives on use and impact of electronic health information in south west Ontario
Supervisor(s): C. Gibson

Poster Number: 36
Name: Aref Sleiman
Degree: MSc Candidate
Abstract Title: Novel bioactive and injectable co-polymer-reinforced bone cement for bone augmentation
Supervisor(s): A.S. Rizkalla, D.W. Hamilton

Poster Number: 37
Name: Thaksha Thavam
Degree: MSc Candidate
Abstract Title: The Impact of the diabetes management incentive on hospitalizations in Ontario
Supervisor(s): S. Sarma

DETECTION, SCREENING AND DIAGNOSIS OF HEALTH AND DISEASE

Poster Number: 38
Name: Androu Abdalmalak
Degree: PhD Candidate
Abstract Title: Towards a robust, optical brain-computer interface for communicating with patients with brain injuries
Supervisor(s): K. St. Lawrence, A. Owen

Poster Number: 39
Name: Juweliya Ahmed
Degree: MSc Candidate
Abstract Title: Identifying biomarkers of delusions and hallucinations in Alzheimer’s disease: A neuroimaging and genetic analysis study
Supervisor(s): E. Finger

Poster Number: 40
Name: Ryan Alfano
Degree: MSc Candidate
Abstract Title: Development of a computer aided diagnosis model for prostate cancer classification on multi-parametric MRI
Supervisor(s): A. Ward

Poster Number: 41
Name: Hayden Atkinson
Degree: PhD Candidate
Abstract Title: Acute quantitative MRI response of tibiofemoral articular cartilage in knees at risk for osteoarthritis after challenged walking
Supervisor(s): T. Birmingham

Poster Number: 42
Name: Jeffrey Brooks
Degree: PhD Candidate
Abstract Title: Development and validation of a novel high-speed video system for measuring saccadic eye movement
Supervisor(s): J. Dickey

Poster Number: 43
Name: James Charbonneau
Degree: Resident
Abstract Title: Diagnostic accuracy of referrals in an academic consultation-liaison service
Supervisor(s): M. Mak

Poster Number: 44
Name: Spencer Christiansen
Degree: PhD Candidate
Abstract Title: Blood clot hematocrit and age differentiation in vitro using R2* and quantitative susceptibility mapping
Supervisor(s): M. Drangova

Poster Number: 45
Name: John Demarco
Degree: MSc Candidate
Abstract Title: Creating a 3D histological atlas of subcortical nuclei using ultra-high field MRI registration: A model for DBS surgical planning
Supervisor(s): J. Lau, A. Khan

Poster Number: 46
Name: Sarah Donnelly
Degree: MSc Candidate
Abstract Title: Magnetic resonance imaging of microbiome using MagA-expressing Escherichia coli
Supervisor(s): J. Burton, D. Goldhawk

Poster Number: 47
Name: Saravanan Esakki
Degree: PhD Candidate
Abstract Title: Rasch analysis of the Patient-Rated Wrist Evaluation questionnaire
Supervisor(s): J. MacDermid
Poster Number: 48
Name: Fang Zhou Ge
Degree: Research Assistant/Associate
Abstract Title: MALDI Mass spectrometry imaging of gangliosides on formalin-fixed tissue
Supervisor(s): S. Whitehead, K. Yeung

Poster Number: 49
Name: Rob Gray
Degree: MSc Candidate
Abstract Title: Image based comparison between the bilateral symmetry of the distal radii through new and established clinical measures
Supervisor(s): E. Lalone, D. Holdsworth

Poster Number: 50
Name: Wenchao Han
Degree: PhD Candidate
Abstract Title: Automatic prostate cancer detection and localization on digital histopathology imaging
Supervisor(s): A. Ward

Poster Number: 51
Name: Michael Iacocca
Degree: MSc Candidate
Abstract Title: Adaptation of ACMG/AMP guidelines for standardized variant interpretation in familial hypercholesterolemia
Supervisor(s): R. Hegele

Poster Number: 52
Name: Peter Jeon
Degree: MSc Candidate
Abstract Title: Echo time comparison for detecting human brain glutamate changes using 7-Tesla functional magnetic resonance spectroscopy
Supervisor(s): J. Théberge, L. Palaniyappan

Poster Number: 53
Name: Mahro Khalid
Degree: MSc Candidate
Abstract Title: Monitoring absolute cerebral blood flow using a self-calibrated software-based DCS system
Supervisor(s): K. St. Lawrence

Poster Number: 54
Name: Jenna Kitz
Degree: MSc Candidate
Abstract Title: Epithelial-to-mesenchymal transition of prostate cancer circulating tumor cells effects detection and enumeration by the CellSearch System®
Supervisor(s): A. Allan

Poster Number: 55
Name: Fiona Li
Degree: PhD Candidate
Abstract Title: Personalization of cancer drug dosage based on kidney function: A preliminary study
Supervisor(s): T. Lee, J. Koropatnick

Poster Number: 56
Name: Isabelle McKillop
Degree: Research Assistant/Associate
Abstract Title: Correlation between the tumour’s location and treatment outcome for liver cancer patients
Supervisor(s): J. Vickress, S. Yartsev

Poster Number: 57
Name: Patrick Murphy
Degree: MSc Candidate
Abstract Title: Characterization of Wilms’ Tumour 1 (WT-1) as a fibrotic biomarker for Duchenne Muscular Dystrophy
Supervisor(s): L. Hoffman

Poster Number: 58
Name: Tomi Nano
Degree: PhD Candidate
Abstract Title: Apodized-aperture Pixel: A novel x-ray detector design to improve cancer detection in mammography
Supervisor(s): I. Cunningham

Poster Number: 59
Name: Daiana Pur
Degree: Research Assistant/Associate
Abstract Title: Influence of cortical thickness on BOLD signal variability
Supervisor(s): R. Eagleson, S. de Ribaupierre

Poster Number: 60
Name: Omar Raslan
Degree: Postdoctoral Scholar
Abstract Title: Prevalence of thrombophilia in suspected stroke patients
Supervisor(s): A. Lazo-Langner

Poster Number: 61
Name: Michael Robinson
Degree: PhD Candidate
Abstract Title: Psychometric evaluation of the SCAT symptom evaluation in adolescents using Rasch analysis
Supervisor(s): J. MacDermid

Poster Number: 62
Name: Roni Shanoada
Degree: Research Assistant/Associate
Abstract Title: Relationship between cerebral blood flow and arterial blood pressure during the dying process
Supervisor(s): M. Slessarev

Poster Number: 63
Name: Nicole Smith
Degree: MSc Candidate
Abstract Title: Testicular hemangioma mimicking cystic dysplasia of the testis
Supervisor(s): N. Chan

Poster Number: 64
Name: Olivia Stanley
Degree: PhD Candidate
Abstract Title: Comparing cortical layer activation using gradient echo with phase regression and spin echo in the human visual cortex
Supervisor(s): R. Menon
Poster Number: 65
Name: Qin Sun
Degree: MSc Candidate
Abstract Title: Using the magnetosome model to refine gene-based iron contrast for magnetic resonance imaging
Supervisor(s): D. Goldhawk, F. Prato

Poster Number: 66
Name: Claire Vannelli
Degree: MSc Candidate
Abstract Title: Accuracy of mitral valve imaging with dynamic computed tomography
Supervisor(s): T. Peters

Poster Number: 67
Name: Andrew Westcott
Degree: MSc Candidate
Abstract Title: Longitudinal imaging biomarkers of severe emphysema in AATD and COPD
Supervisor(s): G. Parraga

Poster Number: 68
Name: Robin Wigen
Degree: Medical Student
Abstract Title: Improving control of mineral bone disease in peritoneal dialysis patients
Supervisor(s): E. Qirjazi

Poster Number: 69
Name: Yee (Michelle) Wong
Degree: MSc Candidate
Abstract Title: Working memory and falls risk in older adults
Supervisor(s): L. Nagamatsu

Poster Number: 70
Name: Eric Wright
Degree: PhD Candidate
Abstract Title: Investigating the impact of scan duration on CT perfusion-derived hemodynamic parameters and infarct or penumbra volumes
Supervisor(s): T. Lee

Poster Number: 71
Name: Lawrence Yip
Degree: PhD Candidate
Abstract Title: Intraoperative tumour margin assessment through photoacoustic imaging in breast conserving surgery
Supervisor(s): J. Carson

DETERMINANTS OF HEALTH

Poster Number: 72
Name: Bishal Gautam
Degree: Postdoctoral Scholar
Abstract Title: Outcome of preterm infants after delivery room cardiopulmonary resuscitation: A retrospective observational study
Supervisor(s): O. da Silva, B. S. Richardson, M. Miller

Poster Number: 73
Name: Matthew Kewin
Degree: MSc Candidate
Abstract Title: Confirmation of a derivative hyperspectral NIRS method for measuring oxygen saturation by comparison to time-resolved NIRS
Supervisor(s): K. St. Lawrence, S. de Ribaupierre

Poster Number: 74
Name: Nicholas Kim
Degree: MSc Candidate
Abstract Title: Heterotypic compatibility of human Connexin37 with vascular connexins
Supervisor(s): D. Bai

Poster Number: 75
Name: Artur Miranda
Degree: PhD Candidate
Abstract Title: Temperature effects on Vj-gating kinetics of Cx40 and Cx45 gap junctions
Supervisor(s): D. Bai

Poster Number: 76
Name: Conrad Pitts
Degree: Research Assistant/Associate
Abstract Title: The hepatic metabolic signature and outcomes of lifelong exposure to Western Diet (high fat/ high sugar) in young adult guinea pigs
Supervisor(s): C. McKenzie, T. Regnault

Poster Number: 77
Name: Adam Rankin
Degree: PhD Candidate
Abstract Title: Augmented reality in image-guided therapy
Supervisor(s): T. Peters, G. Fichtinger

Poster Number: 78
Name: Imran Syed
Degree: MSc Candidate
Abstract Title: Gender and the risk perceptions of smoking: A systematic review
Supervisor(s): S. Wells, T. Elton-Marshall

Poster Number: 79
Name: Braeden Terpou
Degree: MSc Candidate
Abstract Title: Pulvinar functional connectivity patterns in PTSD and its dissociative subtype at rest
Supervisor(s): R. Lanius

EARLY LIFE PROGRAMMING AND DEVELOPMENT

Poster Number: 80
Name: Jamie Ching
Degree: MSc Candidate
Abstract Title: The role of Pannexin 1 in human induced pluripotent stem cell differentiation in vitro
Supervisor(s): D. Laird
Poster Number: 81  
Name: Anish Engineer  
Degree: PhD Candidate  
Abstract Title: MicroRNA-122 contributes to diabetes-induced congenital heart defects  
Supervisor(s): Q. Feng

Poster Number: 82  
Name: Mohamed Gatie  
Degree: PhD Candidate  
Abstract Title: Glucose metabolism: Fueling energy expenditure and directing stem cell fate through epigenetic regulation  
Supervisor(s): G. Kelly

Poster Number: 83  
Name: Elizabeth Greco  
Degree: MSc Candidate  
Abstract Title: The effect of nicotine on fetal heart development  
Supervisor(s): Q. Feng, D. Jones

Poster Number: 84  
Name: Faraj Haddad  
Degree: PhD Candidate  
Abstract Title: Sensory processing deficits in a developmental prenatal immune activation model  
Supervisor(s): S. Schmid

Poster Number: 85  
Name: Gargi Jaju  
Degree: MSc Candidate  
Abstract Title: Histone deacetylases play a vital role in syncytiotrophoblast development  
Supervisor(s): S. Renaud

Poster Number: 86  
Name: Marcus Lo  
Degree: Research Assistant/Associate  
Abstract Title: Behavioral, motor, and neurodevelopmental outcomes in children born with low grade vs. high grade intraventricular hemorrhage  
Supervisor(s): S. de Ribaupierre

Poster Number: 87  
Name: Shelby Oke  
Degree: MSc Candidate  
Abstract Title: Elevated MicroRNA-140 in low protein IUGR offspring with postnatal catch-up growth leads to decreased hepatic Pin1: Mechanism of premature senescence?  
Supervisor(s): D. Hardy

Poster Number: 88  
Name: Sandra Szlapinski  
Degree: PhD Candidate  
Abstract Title: A failure to adaptively increase β-cell mass during pregnancy is associated with maternal glucose intolerance after parturition  
Supervisor(s): D. Hill

Poster Number: 89  
Name: Phyo Win  
Degree: MSc Candidate  
Abstract Title: Beta-cell β1 integrin deficiency during second transition of fetal pancreas development on islet growth.  
Supervisor(s): R. Wang

MECHANISMS OF DISEASE

Poster Number: 90  
Name: Sanna Abbasi  
Degree: PhD Candidate  
Abstract Title: Characterizing protein interactions with the Ku70 von Willebrand A domain in non-homologous end-joining and the DNA damage response pathway  
Supervisor(s): C. Schild-Poulter

Poster Number: 91  
Name: Khadija Ahmed  
Degree: MSc Candidate  
Abstract Title: The characterization of Cx31 in keratinocytes and link to erythrokeratoderma variabilis.  
Supervisor(s): D. Laird

Poster Number: 92  
Name: Akina Au  
Degree: MSc Candidate  
Abstract Title: The absence of ATF3 reduces oncogenic KRAS Activity and overall tumour progression  
Supervisor(s): C. Pin

Poster Number: 93  
Name: Nawab Azizi  
Degree: MSc Candidate  
Abstract Title: rAAV-mediated gene delivery as a tool for investigating bone sialoprotein function in vivo  
Supervisor(s): H. Goldberg, F. Beier

Poster Number: 94  
Name: Kevin Bartman  
Degree: PhD Candidate  
Abstract Title: An investigation into T cell mediated drug hypersensitivity reactions  
Supervisor(s): M. Rieder

Poster Number: 95  
Name: Christine Caron  
Degree: MSc Candidate  
Abstract Title: Investigating the role of Inositol-Requiring Enzyme 1 (IRE1) in regulating the Unfolded Protein Response (UPR), stress adaptation, and chronological aging  
Supervisor(s): P. Lajoie

Poster Number: 96  
Name: Sarah Chadwick  
Degree: PhD Candidate  
Abstract Title: The role of non-alveolar cells in regulating lung development and function  
Supervisor(s): D. Laird
Poster Number: 97
Name: Tyler Dexter
Degree: MSc Candidate
Abstract Title: Role of prefront GABA in rodent touchscreen tasks of executive function: Assessing attention and working memory in mice
Supervisor(s): T. Bussey, L. Saksida

Poster Number: 98
Name: Mihai Dumbrava
Degree: Research Assistant/Associate
Abstract Title: Oxidative stress and Parkinson’s disease associated mutations alter DJ-1 Structure, function, and its interactions
Supervisor(s): M. Duennwald

Poster Number: 99
Name: Karen Dunkerley
Degree: PhD Candidate
Abstract Title: Effects of Parkinson’s disease mutations on the Ubiquitin-like domain of the E3 ligase Parkin
Supervisor(s): G. Shaw

Poster Number: 100
Name: Rachel Eddy
Degree: PhD Candidate
Abstract Title: What is the minimal clinically important difference for MRI ventilation defects?
Supervisor(s): G. Parraga

Poster Number: 101
Name: Roseane Franco
Degree: PhD Candidate
Abstract Title: Cognitive assessment in alpha-synuclein PD mouse models using automate touchscreen tasks
Supervisor(s): F. Beraldo

Poster Number: 102
Name: Ingrid Gan
Degree: PhD Candidate
Abstract Title: Endothelial cell necroptosis and cardiac allograft rejection can be regulated by mitochondrial dysfunction mediators
Supervisor(s): Z. Zhang, A. Jevnikar

Poster Number: 103
Name: Liliana German-Castelan
Degree: PhD Candidate
Abstract Title: Cholinergic regulation of Alzheimer’s-like pathology
Supervisor(s): V. Prado, M. Prado

Poster Number: 104
Name: Hayley Good
Degree: PhD Candidate
Abstract Title: The role of cyclooxygenase in colitis-associated cancer
Supervisor(s): S. Asfaha

Poster Number: 105
Name: Chidambra Halari
Degree: PhD Candidate
Abstract Title: Decorin-dependent expression and function of Connexin-43 in the trophoblast: Implications for pre-eclampsia
Supervisor(s): P. Lala, S. Renaud

Poster Number: 106
Name: Amber Harnett
Degree: MSc Candidate
Abstract Title: Identifying and targeting cancer stem cells in colorectal cancer
Supervisor(s): S. Asfaha

Poster Number: 107
Name: Seana Hill
Degree: MSc Candidate
Abstract Title: Expanded census of human genomic regions with chromatin accessibility differences between homologous metaphase chromosomes based on legacy gene mapping data
Supervisor(s): J. Knoll

Poster Number: 108
Name: Jacklyn Hurst
Degree: MSc Candidate
Abstract Title: The role of streptococcus pyogenes virulence factors in colonization and rheumatic heart disease
Supervisor(s): J. Hurst, K. Kasper, J. McCormick

Poster Number: 109
Name: Aoi Ichiyama
Degree: MSc Candidate
Abstract Title: Stress habitation: Elucidating the neural encoding of stressor significance
Supervisor(s): W. Inoue, B. Allman

Poster Number: 110
Name: Victoria Jaremek
Degree: Research Assistant/Associate
Abstract Title: Assessing cardiac dysfunction post-stroke in the insular cortex ischemic stroke rat model
Supervisor(s): J. Melling, L. Sposato, S. Whitehead

Poster Number: 111
Name: Mariyan Jeyarajah
Degree: MSc Candidate
Abstract Title: Syndecan-4 regulates extravillous cytotrophoblast cell migration by coordinating protein kinase C-alpha activation.
Supervisor(s): S. Renaud

Poster Number: 112
Name: Yuwei Jiang
Degree: PhD Candidate
Abstract Title: Sfp1 integrates TORC1 signaling and composition of the SAGA acetyltransferase complex in Huntington’s disease
Supervisor(s): P. Lajoie
Poster Number: 113  
Name: Hinissan Kohio  
Degree: PhD Candidate  
Abstract Title: Novel insights into the genomic integration site landscape of HIV  
Supervisor(s): S. Barr

Poster Number: 114  
Name: Fatima Kudaeva  
Degree: MSc Candidate  
Abstract Title: Infections and the risk of rheumatoid arthritis  
Supervisor(s): M. Speechley, J. Pope

Poster Number: 115  
Name: Rachel Lackie  
Degree: PhD Candidate  
Abstract Title: The Role of Stress-Inducible Phosphoprotein 1 (STI1) in cellular resilience and Alzheimer's disease  
Supervisor(s): M. Prado, V. Prado

Poster Number: 116  
Name: Jasper Lee  
Degree: MSc Candidate  
Abstract Title: The role of KIM-1 in the pathogenesis of renal cell carcinoma  
Supervisor(s): L. Gunaratnam

Poster Number: 117  
Name: Elaine Liu  
Degree: MSc Candidate  
Abstract Title: Mechanisms of antigen processing and presentation following efferocytosis  
Supervisor(s): B. Heit

Poster Number: 118  
Name: Asad Lone  
Degree: PhD Candidate  
Abstract Title: p66Shc activation promotes increased oxidative phosphorylation and renders CNS cells more vulnerable to amyloid beta toxicity  
Supervisor(s): R. Cumming

Poster Number: 119  
Name: Matthew Maksoud  
Degree: PhD Candidate  
Abstract Title: The effect of nitric oxide on calcium entry and phagocytosis in cortical microglia  
Supervisor(s): W. Lu

Poster Number: 120  
Name: Raanan Marants  
Degree: PhD Candidate  
Abstract Title: How does dialysate cooling affect liver hemodynamics during hemodialysis? A CT perfusion study  
Supervisor(s): T. Lee, C. McIntyre

Poster Number: 121  
Name: Alexander Moszczynski  
Degree: Research Assistant/Associate  
Abstract Title: Comorbid pathologies in neurodegeneration: Synergistic toxicity greater than the sum of their parts  
Supervisor(s): M. Strong

Poster Number: 122  
Name: Maedeh Naghibosadat  
Degree: MSc Candidate  
Abstract Title: Characterization and the role of GHSR in the vasculature of Duchenne muscular dystrophy  
Supervisor(s): S. Dhanvantari, L. Hoffman

Poster Number: 123  
Name: Niharika Nagrani  
Degree: Research Assistant/Associate  
Abstract Title: Glucose caused downregulation of mitochondrial SIRTs through specific microRNAs (miRs)  
Supervisor(s): N. Nagrani, E. Roudbari, B. Feng, S. Chen, S. Chakrabarti

Poster Number: 124  
Name: Geoffrey Ngo  
Degree: MSc Candidate  
Abstract Title: Investigating cortical thickness markers in relapse remitting multiple sclerosis  
Supervisor(s): R. Menon

Poster Number: 125  
Name: Rufina Ning  
Degree: MSc Candidate  
Abstract Title: Endothelial glycocalyx shedding in diabetic ketoacidosis  
Supervisor(s): D. Fraser, G. Cepinskas

Poster Number: 126  
Name: Teresa Nunez de Villavicencio Diaz  
Degree: PhD Candidate  
Abstract Title: Regulatory role of lysine and acetyllysine in CK2-mediated phosphorylation  
Supervisor(s): D. Litchfield

Poster Number: 127  
Name: Amanda Oakie  
Degree: PhD Candidate  
Abstract Title: The effects of c-Kit and IR interplay on beta cell proliferation and intracellular signalling in INS-1 cells  
Supervisor(s): R. Wang

Poster Number: 128  
Name: Cathy Ong Ly  
Degree: Research Assistant/Associate  
Abstract Title: How much do you change? An evaluation of the anatomical consequences of maxillomandibular advancement surgery  
Supervisor(s): T. Wilson, B. Rubin

Poster Number: 129  
Name: Daniel Palmer  
Degree: Postdoctoral Scholar  
Abstract Title: Prefrontal contributions to metacognition: Development of a new behavioural test for rodent models of disease  
Supervisor(s): T. Bussey, L. Saksida
Poster Number: 130
Name: Pirunthan Perampalam
Degree: PhD Candidate
Abstract Title: DREAM as a novel regulator of ovarian cancer cell dormancy
Supervisor(s): F. Dick, G. DiMattia

Poster Number: 131
Name: Kia Peters
Degree: PhD Candidate
Abstract Title: Effects of low dose niacin and vitamin D on vascular regeneration under lipotoxic conditions
Supervisor(s): N. Borradaile

Poster Number: 132
Name: Martin Prusinkiewicz
Degree: PhD Candidate
Abstract Title: E1A affects adenovirus induced metabolic changes
Supervisor(s): J. Mymryk

Poster Number: 133
Name: Michael Racanelli
Degree: MSc Candidate
Abstract Title: Fibroblast specific PTEN deficiency in mice promotes melanoma metastasis due to a fibrotic microenvironment
Supervisor(s): A. Leask

Poster Number: 134
Name: Jess Rhee
Degree: MSc Candidate
Abstract Title: The role of PU.1 in lipid metabolism and cell cycle regulation in myeloid progenitor cells
Supervisor(s): R. DeKoter

Poster Number: 135
Name: Megan Rowland
Degree: PhD Candidate
Abstract Title: Thyroxine supplementation ameliorates hypomyelination in ATRX deficient mice
Supervisor(s): N. Bérubé, F. Beier

Poster Number: 136
Name: Julliane Santos
Degree: PhD Candidate
Abstract Title: Analysis of neuromuscular alterations using a new transgenic mouse model with cholinergic dysfunction selective to motoneurons
Supervisor(s): M. Prado, V. Prado

Poster Number: 137
Name: Vidhyasree Shyam
Degree: MSc Candidate
Abstract Title: The Role of TP53INP2 in neuronal mitophagy
Supervisor(s): S. Cregan

Poster Number: 138
Name: Akshay Sule
Degree: MSc Candidate
Abstract Title: Role of pili in nasopharyngeal colonization by Streptococcus pyogenes
Supervisor(s): J. McCormick

Poster Number: 139
Name: Rebecca Sullivan
Degree: PhD Candidate
Abstract Title: The growth hormone secretagogue receptor, ghrelin, and biochemical signaling processes in human heart failure
Supervisor(s): S. Dhanvantari

Poster Number: 140
Name: Julia Sunstrum
Degree: MSc Candidate
Abstract Title: Synaptic correlates for stress habituation
Supervisor(s): W. Inoue

Poster Number: 141
Name: Renee Tamming
Degree: PhD Candidate
Abstract Title: Investigating the role of ATRX in hippocampal pyramidal neurons
Supervisor(s): N. Bérubé

Poster Number: 142
Name: Kyle Taruc
Degree: MSc Candidate
Abstract Title: Elucidation of the signaling pathway of MERTK
Supervisor(s): B. Heit

Poster Number: 143
Name: Yodit Tesfagiorgis
Degree: PhD Candidate
Abstract Title: The unexpected differences of B and T cell infiltration in the inflamed central nervous system in an animal model of multiple sclerosis
Supervisor(s): S. Kerfoot

Poster Number: 144
Name: Anu Thomas
Degree: PhD Candidate
Abstract Title: H19 regulates glucose-induced EndMT in chronic diabetic complications
Supervisor(s): S. Chakrabarti

Poster Number: 145
Name: Christine Wardell
Degree: MSc Candidate
Abstract Title: Mucosa-associated invariant T cells enhance anti-Influenza A virus CD8+ T cell cytotoxicity
Supervisor(s): S.M. Haeryfar

Poster Number: 146
Name: Krystyna Wieczerzak
Degree: PhD Candidate
Abstract Title: Unraveling the role of NMDA receptor hypofunction in aberrant cortical oscillations and cognitive dysfunction: Implications for schizophrenia
Supervisor(s): B. Allman
**Poster Number:** 147  
**Name:** Meagan Wiederman  
**Degree:** MSc Candidate  
**Abstract Title:** The neuroanatomical origin of GABAergic synaptic inputs on corticotropin releasing hormone neurons determines GABA neurons responsiveness to noradrenaline using the same pathway as prostaglandin E2  
**Supervisor(s):** W. Inoue

**Poster Number:** 148  
**Name:** Rachel Wilson  
**Degree:** MSc Candidate  
**Abstract Title:** The effects of elongation factor 1A-1 (EEF1A1) inhibition on the progression of non-alcoholic fatty liver disease (NAFLD)  
**Supervisor(s):** N. Borradaile

**Poster Number:** 149  
**Name:** Ben Withers  
**Degree:** MSc Candidate  
**Abstract Title:** The Leucine-Rich domain of RGNEF as a protective modifier in a model of ALS pathology  
**Supervisor(s):** M. Strong

**Poster Number:** 150  
**Name:** Keertika Yogendirarajah  
**Degree:** MSc Candidate  
**Abstract Title:** Investigating the function of a novel porin in Helicobacter pylori  
**Supervisor(s):** C. Creuzenet

**Poster Number:** 151  
**Name:** Najwa Zebian  
**Degree:** Postdoctoral Scholar  
**Abstract Title:** Comprehensive analysis of HIV-1 Env glycosylation  
**Supervisor(s):** C. Creuzenet, E. Arts

**Poster Number:** 152  
**Name:** Anthony Ziccarelli  
**Degree:** MSc Candidate  
**Abstract Title:** Mechanistic processes of TβR3 in non-small-cell lung carcinoma  
**Supervisor(s):** J. Di Guglielmo

**POPULATIONS, PUBLIC HEALTH AND EDUCATION**

**Poster Number:** 153  
**Name:** Farah Abdulsatar  
**Degree:** Postdoctoral Scholar  
**Abstract Title:** Unsafe teething remedies in a mid-size city: A survey  
**Supervisor(s):** S. Taheri

**Poster Number:** 154  
**Name:** Ahmed Al-Jaishi  
**Degree:** PhD Candidate  
**Abstract Title:** The effect of non-differential misclassification of confounding variables on parameter estimates in comparative effectiveness research  
**Supervisor(s):** A. Garg

**Poster Number:** 155  
**Name:** Adrian Buttazzoni  
**Degree:** MSc Candidate  
**Abstract Title:** Investigating community stakeholder and key facilitator perspectives: A qualitative analysis of a regional ASRTS program  
**Supervisor(s):** J. Gilliland

**Poster Number:** 156  
**Name:** Kiersten Colbran  
**Degree:** Medical Student  
**Abstract Title:** Utilization of a web-based module (WBM) for caregiver pain management following fractures in children: A randomized controlled trial  
**Supervisor(s):** N. Poonai

**Poster Number:** 157  
**Name:** Alexandra Jackson  
**Degree:** MSc Candidate  
**Abstract Title:** The experience of loneliness in older adults living in retirement community settings: A research proposal  
**Supervisor(s):** D. Connelly

**Poster Number:** 158  
**Name:** Emily Kyle  
**Degree:** MSc Candidate  
**Abstract Title:** Implementation of novel educational techniques in Pathologists’ Assistant (PA) student/resident training  
**Supervisor(s):** N. Chan, J. Gomez

**Poster Number:** 159  
**Name:** Javeed Sukhera  
**Degree:** PhD Candidate  
**Abstract Title:** Reducing stigma through implicit bias recognition and management: A realist evaluation of implicit stigma reduction curricula for health professionals  
**Supervisor(s):** C. Watling

**Poster Number:** 160  
**Name:** Leah Taylor  
**Degree:** MSc Candidate  
**Abstract Title:** Examining children’s perceived barriers to physical activity in the varying environments of northern and southern Ontario  
**Supervisor(s):** J. Gilliland

**Poster Number:** 161  
**Name:** Stephanie Truelove  
**Degree:** PhD Candidate  
**Abstract Title:** Exploring the physical education-related needs and self-efficacy of elementary school teachers  
**Supervisor(s):** P. Tucker

**Poster Number:** 162  
**Name:** Helen Wang  
**Degree:** Research Assistant/Associate  
**Abstract Title:** Generating Phaeodactylum tricornutum auxotrophs using CRISPR-Cas9 and TevCas9  
**Supervisor(s):** D. Edgell
PREVENTION OF DISEASES AND HEALTH CONDITIONS AND PROMOTION OF WELL-BEING

Poster Number: 163
Name: Nick Bray
Degree: PhD Candidate
Abstract Title: The effect of high dose vitamin D on physical performance in frail older adults: A feasibility study
Supervisor(s): M. Montero-Odasso

Poster Number: 164
Name: Brett Coelho
Degree: MSc Candidate
Abstract Title: Micromechanics based modelling of in-vivo respiratory motion of the diaphragm muscle with the incorporation of optimized Z-disks mechanics
Supervisor(s): A. Samani

Poster Number: 165
Name: Ariel Frame
Degree: PhD Candidate
Abstract Title: Age dependent alterations in lactate dehydrogenase expression and activity promote memory impairment in Drosophila melanogaster
Supervisor(s): R. Cumming

Poster Number: 166
Name: Joyla Furlano
Degree: MSc Candidate
Abstract Title: Feasibility of a 6-month resistance training program on cognition and brain health in older adults at-risk for diabetes: A pilot study
Supervisor(s): L. Nagamatsu

Poster Number: 167
Name: Reza Khazaee
Degree: MSc Candidate
Abstract Title: Impacts of Fetal Growth Restriction (FGR) on the pulmonary surfactant system in response to sepsis
Supervisor(s): R. Veldhuizen, C. Yamashita

Poster Number: 168
Name: Dorota Klubowicz
Degree: MSc Candidate
Abstract Title: A novel idea in the use of ultrasound to guide nasolabial Hyaluronic Acid (HA) dermal filler injections to prevent alar nasal necrosis
Supervisor(s): K. Galil

Poster Number: 169
Name: Jeffrey Levine
Degree: MSc Candidate
Abstract Title: Impact of the 2015 nepal earthquakes on hair cortisol concentration and mental health in affected villagers
Supervisor(s): M. Rieder

Poster Number: 170
Name: Alexandra Pearce
Degree: MSc Candidate
Abstract Title: Don’t sweat it: Investigating the effects of exercise frequency on cognition
Supervisor(s): A. Owen

Poster Number: 171
Name: Scarlett Puebla-Barragan
Degree: PhD Candidate
Abstract Title: The use of probiotic bacteria and their metabolites for the reduction of malodor in the female urogenital tract: Project overview
Supervisor(s): G. Reid

Poster Number: 172
Name: Rachel Reingold
Degree: MSc Candidate
Abstract Title: Increased cholinergic signaling protects against cardiac injury
Supervisor(s): R. Gros

Poster Number: 173
Name: Ualina Tariq
Degree: MSc Candidate
Abstract Title: Levels and correlates of sugar intake among Canadian children and adolescents in 2004 and 2015
Supervisor(s): P. Wilk

Poster Number: 174
Name: Emiley Watson
Degree: MSc Candidate
Abstract Title: The role of Lactobacillus crispatus in female reproductive health
Supervisor(s): G. Reid

Poster Number: 175
Name: Samantha Whiteside
Degree: Postdoctoral Scholar
Abstract Title: Lactobacillus secretions for the prevention and treatment of Helicobacter pylori infections
Supervisor(s): C. Creuzenet

Poster Number: 176
Name: Rubai Zhou
Degree: PhD Candidate
Abstract Title: Identification of the social functional biomarker for patients with major depressive disorder through structural neuroimaging
Supervisor(s): Y. Fang, L. Palaniyappan, J. Wang
# London Health Research Day 2018

**Driving Innovation Through Collaboration**

## Feature Platform Presentations

**Morning Session**

10:30 - 11:45 a.m.
London Convention Centre - First Floor

*Times are approximate*

<table>
<thead>
<tr>
<th>TIME</th>
<th>SALON B</th>
<th>SALON B1</th>
<th>SALON D</th>
<th>SALON E</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:30 - 10:45 A.M.</td>
<td>Victoria Deveau</td>
<td>Erfan Aref-Eshghi</td>
<td>Kelly Baines</td>
<td>Frederico Faria</td>
</tr>
<tr>
<td>10:45 - 11:00 A.M.</td>
<td>Zachary Hawley</td>
<td>Carolina Batista</td>
<td>Nina Hunt</td>
<td>Tim Tian Han</td>
</tr>
<tr>
<td>11:00 - 11:15 A.M.</td>
<td>Devika Jayawardena</td>
<td>Jacob Fanous</td>
<td>Zhe Li</td>
<td>Bridget Irwin</td>
</tr>
<tr>
<td>11:15 - 11:30 A.M.</td>
<td>Jina Kum</td>
<td>Matthew Maitland</td>
<td>Kate Parham</td>
<td>Miljan Kuljanin</td>
</tr>
<tr>
<td>11:30 - 11:45 A.M.</td>
<td>Anish Naidu</td>
<td>Victoria Thorburn</td>
<td>Alice Shin</td>
<td>Kara Ruicci</td>
</tr>
</tbody>
</table>
THE ROLE OF HAND1 IN THE DEVELOPMENT OF THE LATERAL PLATE MESODERM IN XENOPUS

VICTORIA DEVEAU
MSc Candidate

Research Areas:
Molecular cellular
Early life programming and development

Supervisor(s):
T. Drysdale

Advisory Committee:
R. Veldhuizen, R. DeKoter, F. Beier

Abstract:
Introduction: The lateral plate mesoderm (LPM) gives rise to several lineages including the heart and circulatory system. This progression from a progenitor cell to endothelial cell lineage relies on early expression of specific transcription factors. The transcription factor of interest in this study is Hand1. In mouse, Hand1 is vital in development of the trophoblast, the extra-embryonic mesoderm and cardiomyocytes. However, the lethality of Hand1 null mice has made identification of the precise role of Hand1 during development difficult. In Xenopus, we observed that the Hand1 expression pattern correlates very closely with the size of the vascular plexus, suggesting Hand1 may play an additional role in within the embryonic vasculature.

Hypothesis: We hypothesize that Hand1 is required for the development of the lateral plate mesoderm in Xenopus. The objectives of my study are to utilize CRIPSR/Cas9 to generate Hand1 mutants, and develop an assay to characterize the phenotype of Hand1 mutants.

Materials and Methods: We generated a Hand1 knockout (KO) model in Xenopus using CRIPSR-Cas9 targeted to the Hand1 gene. Guide RNA and CRISPR protein were injected into Xenopus embryos at the one-cell stage. Water injected embryos were used as a control for the injection process itself. Sequencing of the Hand1 gene in injected embryos was performed to ensure Cas9 was targeting the correct area of the genome. Expression of genes that differentiate the vasculature and heart, assessed through whole mount in situ hybridization, were used to image those lineages in the intact embryo. The phenotype of the vasculature plexus was classified into 4 classes of defects, ranging from normal vasculature to no vasculature. These classifications were based on complexity of the vascular network and overall vascularized area of the embryo and a Chi-Square test was performed to determine significance between phenotype frequency. Area of the non-vascularized region and complexity of the vasculature were also measured, and a One-Way ANOVA was performed comparing water injected embryos, and either 1 or 3 guide RNA injected embryos, all targeting the Hand1 gene at different locations.

Results: Sequencing of Hand1 mutants confirms that Cas9 is targeting the correct area of the genome, with a 90% efficiency rate for all 3 guides that were used in the study. Hand1 mutants have defects in heart morphology, showing defective heart tube looping and an increase in cardiac edema. Hand1 mutants had a significant increase in the proportion of embryos displaying vasculature defects as compared to controls (p>0.001). Furthermore, when quantified Hand1 mutants had a significant increase in the area of the non-vascular zone, and a significant decrease in vascular complexity (p>0.01) compared to controls.

Discussion and Conclusions: The altered heart morphology seen in our Hand1 KOs are consistent with those of Hand1 mice KOs, and we also have shown a potential new role for Hand1 in the embryonic vasculature arising from the LPM. We conclude that Hand1 is necessary for the formation of a complex vasculature system within Xenopus. Further analysis to determine the targets of Hand1 will be critical in understanding the mechanisms by which Hand1 regulates these developmental pathways.
Salon B1

MOLECULAR DIAGNOSIS OF HEREDITARY SYNDROMES AND CANCER USING GENOMIC DNA METHYLATION

ERFAN AREF-ESHGHI
Postdoctoral Scholar

Research Areas:
Translational research
Detection, screening and diagnosis of health and disease

Supervisor(s):
B. Sadikovic

Advisory Committee:
N/A

Abstract:
Introduction: DNA methylation of the CpG dinucleotide plays an integral role in the regulation of the processes that control the normal development. Aberrant DNA methylation in early development leads to neurodevelopmental syndromes, while its disruption in somatic tissues is associated with carcinogenesis. As a relatively stable functional modification, genomic DNA methylation has been the focus of biomarker discovery. Currently, the diagnosis of neurodevelopmental syndromes is challenging due to complex and overlapping clinical presentations and un-interpretability of the genetic variants of unknown significance. Similarly, in prostate cancer for example, the current diagnostic methods based on pathology examination of prostate needle biopsies produce high false negative rates due to the temporospatial, molecular, and morphological heterogeneity of prostate adenocarcinoma.

Hypothesis: We hypothesize that both neurodevelopmental conditions and cancer generate specific DNA methylation epi-signatures which can be utilized in molecular diagnosis and disease screening.

Materials and Methods: Peripheral blood samples from disease-specific cohorts of patients with Mendelian neurodevelopmental syndromes caused by defects in epigenomic machinery, as well as archival prostate cancer and benign tissues were assessed for genome-wide methylation changes using Illumina Infinium 450k and EPIC arrays. Supervised and unsupervised machine learning techniques, including support vector machine, LASSO, and unsupervised hierarchical clustering, were used to develop predictive models for each disorder. We used independent cohorts to validate the performance of our prediction models.

Results: We identified highly sensitive and specific peripheral blood DNA methylation epi-signatures in a number of genetic conditions including Floating-Harbor Syndrome, autosomal dominant cerebellar ataxia, deafness, and narcolepsy, alpha thalassemia/mental retardation X-linked syndrome, Kabuki syndrome, Sotos syndrome, CHARGE syndrome, Claes-Jensen syndrome, Genitopattellar syndrome, and Coffin-Siris syndrome. Using a total of ~1,000 selected CpG probes from every epi-signature we trained a multi-class machine-learning-based prediction model, enabling concurrent classification of the mentioned disorders, with 100% accuracy as determined using multiple external and internal validating cohorts. We demonstrated the ability of the algorithm to identify undiagnosed cases in a screened cohort, resolve the ambiguous clinical cases carrying variants of unknown significance, or to assign a new diagnosis to patients with absence of previous or correct clinical diagnosis. Similarly, in prostate cancer, using four CpG loci, we achieved 96% sensitivity and 98% specificity in differentiating the tumor form benign prostate as confirmed using an external cohort of 234 tumors and 92 benign samples. We also showed that this method can sensitively detect metastatic lesions in bone, lymph node, and soft tissue.

Discussion and Conclusions: This study describes unique, machine learning-derived DNA methylation signatures, enabling highly sensitive and specific molecular diagnosis in both hereditary genetic syndromes as well as somatic/acquired disorders.
Salon D

UTERINE NATURAL KILLER CELLS PREVENT FETAL GROWTH RESTRICTION FOLLOWING MATERNAL POLYINOSINIC-POLYCYTIDYLIC ACID EXPOSURE IN RATS

KELLY BAINES
PhD Candidate

Research Areas:
Fetal, family development
Early life programming and development

Supervisor(s):
S. Renaud, D. Hardy

Advisory Committee:
P. Lala, D. Hamilton, G. Eastabrooke

Abstract:

Introduction: Fetal growth restriction (FGR) affects 8% of pregnancies, leading to both maternal and fetal mortality. Additionally, FGR can lead to chronic morbidities such as cardiovascular and metabolic disorders in the developing child. Excessive or chronic maternal inflammation is associated with FGR; however, the mechanisms of this relationship remain unclear. Our lab is interested in how uterine Natural Killer (NK) cells, the most prevalent immune cells in the uterus during early pregnancy, respond to inflammation and contribute to fetal and placental development.

Hypothesis: We hypothesize that inflammation will disrupt the normal function of uterine NK cells, leading to FGR.

Materials and Methods: We used a rat model which displays extensive trophoblast-mediated spiral arteriole remodeling and possesses an abundant uterine NK cell population. Maternal inflammation was induced by injection of polyinosinic-polycytidylic acid (polyI:C, 10mg/kg) on gestational day (GD) 8.5. To deplete NK cells, rats were injected with asialo GM1 antibodies on GD4.5. Changes in uterine cytokines were assessed by qRT-PCR, and placental morphology was analyzed by immunohistochemistry staining for cytokeratin (epithelial cells), and vimentin (endothelial cells). Fetal growth was assessed by measuring fetal weight, organ weight, and crown-rump length. Statistical significance was determined using Student’s t-test and ANOVA (p<0.05).

Results: Compared to saline-injected dams, polyI:C decreased fetal weight, fetal crown-rump length, and placental weight by 15%, 4%, and 12%, respectively, on GD13.5 (n≥6 dams, p<0.05). On GD18.5, polyI:C decreased fetal brain weight, fetal liver weight, fetal weight, and fetal crown-rump length by 9%, 10%, 9%, and 2%, respectively (n≥4 fetuses, p<0.05). Decreased fetal weight correlated with increased production of inflammatory markers: interferon-gamma (3-fold), tumor necrosis factor-alpha (5-fold), interleukin-6 (10-fold), and perforin (4-fold) six hours following polyI:C injection compared to saline (n≥6, p<0.05). At GD13.5, increased junctional zone depth (26%) and decreased labyrinth zone depth (24%) were observed following polyI:C (p<0.05). Immunodepletion of NK cells prior to polyI:C caused a 40% decrease in fetal weight compared to controls on GD13.5 (n≥4 dams, p<0.05).

Discussion and Conclusions: Administration of polyI:C to pregnant rats resulted in elevated cytokine production within the uterus and caused FGR throughout pregnancy that was exacerbated in dams lacking NK cells. These results suggest uterine NK cells have a protective role on fetal development following maternal inflammation. These experiments will provide a foundation to uncover potential therapies to improve fetal outcomes in FGR.
DUAL-TASKING ATTENUATES ANTICIPATORY GAIT ADJUSTMENTS TO NEGOTIATE AN OBSTACLE IN OLDER ADULTS WITH MILD COGNITIVE IMPAIRMENT: RESULTS FROM “THE GAIT & BRAIN STUDY”

FREDERICO FARIA
Postdoctoral Scholar

Research Area:
Musculoskeletal health and rehabilitation
Prevention of diseases and health conditions and promotion of well-being

Supervisor(s):
M. Montero-Odasso

Advisory Committee:
M. Borrie

Abstract:
Introduction: Falls are highly prevalent in older adults with Mild Cognitive Impairment (MCI) and frequently happen while walking forward negotiating obstacles. Although previous research have shown that anticipatory postural control to overcome an upcoming balance perturbation is impaired due to cognitive decline, it is currently unknown the impact of MCI on anticipatory gait control to avoid collisions with obstacles while navigating. In this study, we aimed to test whether obstacle negotiation is impaired in older adults with MCI while facing cognitive challenges, using the dual task paradigm (walking while talking).

Hypothesis: Individuals with MCI will perform less anticipatory gait adjustments before stepping over an obstacle compared with controls (normally aged cognition) when distracted by a mental task (dual-tasking).

Materials and Methods: An obstacle negotiation protocol was applied during single and dual-task (counting backwards by 1s from 100 while walking) conditions to Control and MCI participants. To assess the anticipatory gait control behaviour, a 6m electronic walkway embedded with sensors recorded foot prints to measure temporal and spatial gait variables, specifically gait speed and step length variability, during early phase (3 steps before the late phase) and late phase (3 steps before obstacle) before crossing an “ad hoc” obstacle, set at 15% of participant’s height.

Results: Seventy nine participants (mean age = 72.0 ±2.7 years) were included in this study (controls = 27; MCI = 52). A significant interaction between group and early and late phases (p=0.01) revealed that gait speed in the MCI group decelerated less before the obstacle than the control group when dual-tasking. Similarly, step length variability when dual-tasking increased less in MCI before the obstacle (p=0.05) compared to controls. Results remained significant after controlling for demographics, physical and medical confounders. 3% of individuals with MCI collided with the obstacle when dual-tasking; whereas obstacle collisions were not observed among Controls.

Discussion and Conclusions: Individuals with MCI had attenuated anticipatory gait adjustments before stepping over an obstacle while walking and dual-tasking. This behaviour is a strong sign of “posture second” strategy in which individuals set wrong task priorities while facing balance challenges. Underlying cognitive impairments appear to mediate obstacle negotiation behaviours and may be a factor for the high risk of falls seen in MCI.
MiR-105 IS A CENTRAL REGULATOR OF INTERMEDIATE FILAMENTS ASSOCIATED WITH AMYOTROPHIC LATERAL SCLEROSIS (ALS)

ZACHARY HAWLEY
PhD Candidate

Research Areas:
Molecular cellular
Mechanisms of disease

Supervisor(s):
M. Strong

Advisory Committee:
R. Hammond, S. Chakrabarti, S. Whitehead

Abstract:
Introduction: Amyotrophic Lateral Sclerosis (ALS) is an untreatable neurodegenerative disease defined by progressive motor neuron death causing mortality 2-5 years after onset. Common pathological features of ALS include cytoplasmic inclusions of intermediate filaments (IFs) within motor neurons, where loss of IF stoichiometry due to changes in mRNA steady-state levels is thought to drive IF pathogenesis. In particular, neurofilament light (NEFL), peripherin (PRPH), and alpha-internexin (INA) are IFs that have drastically reduced mRNA levels within spinal motor neurons of ALS patients. While the mechanism by which IF mRNA levels are altered in ALS is largely unknown, it is critical to elucidate this pathway if we are to identify new therapeutic targets for ALS patients in the future. Interestingly, it was recently identified that microRNAs (miRNAs)—critical regulators of mRNA metabolism via interactions with the 3’ untranslated region (UTR)—also show drastically reduced expression in spinal motor neurons of ALS patients, indicating that the disruption of NEFL, PRPH and INA mRNA steady-state levels may be caused by the dysregulation of miRNAs in ALS.

Hypothesis: Dysregulation of a specific pool of miRNAs in ALS disrupts the mRNA steady-state levels of ALS-linked IFs (NEFL, PRPH and INA).

Materials and Methods: MiRanda software was used to identify miRNA candidates predicted to regulate the expression of IFs of interest. Candidate miRNAs were tested for differential expression between ALS patients and healthy controls within the spinal cord using real-time PCR, while fluorescent in situ hybridization (FISH) determined where miRNAs were localized within the spinal cord. NEFL, PRPH, and INA 3’UTRs that are expressed in human spinal cord were isolated and individually cloned into a vector linking them to a reporter gene. Reporter gene assays were done to determine if candidate miRNAs could directly target each IF 3’UTR and regulate the mRNA metabolism of the reporter gene within cell culture models.

Results: MiRanda identified 9 miRNAs predicted to regulate IFs of interest; however, real-time PCR revealed only 3 of the miRNAs (miR-105, miR-140-5p and miR-9) were significantly down-regulated within the spinal cord of ALS patients. FISH analysis showed that these miRNAs were expressed within spinal motor neurons. While all three of these miRNAs were predicted to regulate all IFs of interest, only miR-105 was shown to regulate the mRNA metabolism of the reporter gene when it contained either the NEFL, PRPH or INA 3’UTRs. MiR-9 had similar effects on the reporter gene when it contained either the NEFL or PRPH 3’UTRs, but not when it contained the INA 3’UTR, while miR-140-5p did not bind to any of the 3’UTRs to regulate mRNA metabolism.

Discussion and Conclusions: In this study, we determined miR-105 to be significantly down-regulated in ALS, normally expressed within spinal motor neurons, and involved in regulating the mRNA metabolism of ALS-linked IFs—NEFL, PRPH and INA. Overall, this data indicates that miR-105 is a central regulator of IFs associated with ALS, and that the down-regulation of miR-105 in ALS likely plays a role in disrupting IF mRNA steady-state levels, potentially promoting IF pathogenesis. Based on the role of miR-105 in IF regulation, it could be a potential therapeutic target to slow the progression of the disease; however, further investigation within in vivo models is still needed.
DELETION OF GENES ENCODING PU.1 AND Spi-B LEADS TO B CELL ACUTE LYMPHOBLASTIC LEUKEMIA ASSOCIATED WITH DRIVER MUTATIONS IN JANUS KINASE 3

CAROLINA BATISTA
PhD Candidate

Research Areas:
Cancer biology
Mechanisms of disease

Supervisor(s):
R. DeKoter

Advisory Committee:
S. Kerfoot, D. Hess

Abstract:
Introduction: B-cell acute lymphoblastic leukemia (B-ALL) is often linked with the presence of genomic translocations and/or mutations in genes encoding transcription factors. Spi-B and PU.1 belong to the ETS-transcription factors family and have a critical role in B cell maturation regulating the activation of several genes during development. The double-deletion of Spi-B and PU.1 in B cells is known to cause B-ALL in mouse models, however, the genetic mechanisms underlying disease generation are still uncovered. To investigate this, we have developed and characterised an improved conditional deletion model using the Mb1-Cre mouse, in which the Spib and Spi1 (encoding PU.1) genes are deleted during early B cell development with high efficiency in the bone marrow. These mice develop severe B-ALL characterized by an accumulation of immature B cells in the pre-B cell stage in organs as the spleen and the thymus. Analysis of survival demonstrated that the latency period of B-ALL in Mb1-CreΔPB mice is 18 weeks, point in which these mice require euthanasia due to presenting signals of disease as elaborate breathing and lethargy. Therefore, this observation led us to hypothesize that secondary driver mutations are required for the development of leukemia in the absence of Spi-B and PU.1 transcription factors.

Hypothesis: We hypothesized that secondary driver mutations are required for B-cell acute lymphoblastic leukemia development in the absence of the ETS-transcription factors Spi-B and PU.1.

Materials and Methods: Seeking to characterize the mutational profile of these leukemias, we performed combined whole-exome sequencing (WES) and RNA-sequencing (RNA-seq) in thymic tumours extracted from Mb1-CreΔPB mice. Genomic DNA and mRNA were extracted from three different mouse leukemias and used as a template for WES and RNA-seq applications. Matched-tail DNA was also extracted and used as a control in the WES analysis. Single-Nucleotide Variants (SNVs) were identified by Strelka and Mutect and gene expression levels were determined using Cufflinks. Wild-type pro-B cells were infected with MIGR1-GFP vectors containing the SNVs identified WES. Cell proliferation and survival advantage was determined by cell counting and by flow cytometry, respectively.

Results: Combined WES and RNA-seq analysis revealed that two out of three samples analyzed presented SNVs in genes encoding AIOLOS and JAK3. Importantly, these genes also appeared to be highly expressed in these leukemias. Gene ontology pathway analysis revealed that most of the mutated and highly expressed genes were involved in cellular processes as “Proliferation”, “Cell cycle” and “Metabolism”. Further analysis characterizing the biological effect of the SNVs identified by WES within the JAK3 gene have shown amino acid change consequences as T844M, R653H and V670A. Wild type pro-B cells infected with MIGR1-GFP vectors containing one of the single mutations have shown increased proliferation and growth advantage in conditions of low (0.5%) IL-7.

Discussion and Conclusions: Our results suggest that leukemia in Mb1-CreΔPB mice is induced by acquired driver mutations that cooperate with the initial driver mutation in genes encoding PU.1 and Spi-B. Mutations in AIOLOS and JAK3 genes have been previously identified driver mutations in human pre-B cell acute lymphoblastic leukemia, therefore represent potential secondary drivers of leukemogenesis in the Mb1-CreΔPB mouse model.
INTERFERON-INDUCED HERC5 IS A NOVEL AND POTENT INHIBITOR OF EBOLA VIRUS-LIKE PARTICLE PRODUCTION

NINA HUNT
MSc Candidate

Research Areas:
Infection and immunity
Mechanisms of disease

Supervisor(s):
S. Barr

Advisory Committee:
J. Mymryk, I. Heinemann

Abstract:
Introduction: The ability of Ebola virus to trigger the host immune response and cause hemorrhaging, without triggering an effective antiviral response, demands critical analysis of the virus-host interplay during the course of Ebola viral infection. The major means by which Ebola virus avoids an effective antiviral response is through suppression of the Type I interferon (IFN) response. This suggests that the Type I IFN pathway may restrict Ebola virus replication in the absence of antagonism mediated by Ebola accessory proteins. One IFN-induced protein, HECT and RCC-1-like domain containing protein 5 (HERC5), warrants further investigation as it has been shown to inhibit the replication of evolutionarily diverse viruses through its E3 ligase dependent and independent mechanisms.

Hypothesis: We hypothesized that HERC5 can restrict the replication of Ebola virus-like particles (VLPs) and that Ebola virus possesses one or more proteins to antagonize HERC5 function.

Materials and Methods: We have developed an Ebola virus release assay using the Ebola structural protein VP40, which has previously been shown to form Ebola VLPs similar to wild-type virus in the absence of any other Ebola proteins. We measured the effect of overexpressed HERC5 on VP40 VLPs using Western blotting and quantitative PCR techniques. We also performed transmission electron microscopy and confocal microscopy to examine cells co-transfected with VP40 and either HERC5 or empty vector. Finally, we used co-immunoprecipitation to examine interaction partners of HERC5 and Ebola VP40.

Results: We have shown that HERC5 acts as a novel and potent inhibitor of Ebola virus by drastically reducing structural protein VP40 at both the protein and mRNA level. This resulted in a nearly complete block in Ebola VP40 VLP release from cells as shown through Western blotting, confocal and transmission electron microscopy. Mutagenesis of HERC5 showed that the RCC-1-like domain is necessary and sufficient for such restriction. This indicates an E3-ligase independent mechanism, as the HECT domain is responsible for E3 ligase activity. We have also identified zinc-finger antiviral protein (ZAP) as a potential partner involved in HERC5-mediated restriction. Co-immunoprecipitation assays demonstrated an interaction between ZAP and HERC5, while ZAP knockdowns demonstrated that HERC5-mediated restriction necessitates ZAP activity. Finally, we have shown Ebola’s ability to antagonize HERC5-mediated restriction through Western blotting and qPCR techniques. The presence of Ebola glycoprotein (GP) rescued Ebola VP40 protein and mRNA levels, suggesting that GP acts as an antagonist towards HERC5. Future studies aim to further elucidate the mechanism of GP antagonism, and to utilize an Ebola pseudovirus system to investigate HERC5-mediated activity in the presence of all other Ebola proteins.

Discussion and Conclusions: Overall, by showing that HERC5 targets Ebola VP40 at both the protein and mRNA level, we have identified a novel antiviral mechanism targeting Ebola RNA. Moreover, the ability of HERC5 to deplete viral RNA via its RCC-1 like domain and ZAP identifies a novel E3 ligase-independent antiviral mechanism for HERC5. Moving forward, this information is translatable to therapeutic strategies such as small molecule drugs that can mimic the effects of HERC5 while inhibiting antagonistic activity mediated by Ebola virus GP.
Salon E

DYNAMIC PRECONDITIONING OF ADIPOSE-DERIVED STROMAL/STEM CELLS ON ENGINEERED BIOSCAFFOLDS FOR THE REGENERATION OF SUBCUTANEOUS ADIPOSE TISSUE

TIM TIAN HAN
PhD Candidate

Research Areas:
Medical biophysics, engineering and imaging
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

Supervisor(s):
L. Flynn

Advisory Committee:
A. Allan, C. Seguin, M. Sandig

Abstract:
Introduction: Subcutaneous soft tissue defects can severely compromise quality of life. With the goal of advancing towards a predictable and long-term treatment strategy, our lab has pioneered a subcutaneous adipose tissue substitute by extracting the extracellular matrix of surgically discarded fat to obtain decellularized adipose tissue (DAT). We have previously shown that the DAT seeded with allogeneic adipose-derived stromal/stem cells (ASCs) can promote adipose tissue regeneration in immunocompetent rats through the secretion of beneficial paracrine factors and cytokines. However, the employed static cell-seeding method limits ASC attachment and infiltration, which may impede the efficacy of this cell-based therapy. In this study, we aim to precondition ASCs on the DAT in a 3-D perfusion bioreactor system to overcome these limitations and promote a pro-regenerative ASC phenotype.

Hypothesis: Preconditioning human ASC-seeded DAT in a 3-D perfusion bioreactor under hypoxia will enhance the regeneration of healthy host-derived adipose tissue following subcutaneous implantation in an immunocompromised murine model.

Materials and Methods: Human ASCs were seeded on the DAT at a density of 1x10⁶ cells/scaffold and cultured for 14 days either statically or dynamically in the perfusion bioreactor, under normoxic (~20% O₂) or hypoxic (2% O₂) conditions. These four experimental groups, 20% O₂ static, 20% O₂ dynamic, 2% O₂ static, 2% O₂ dynamic, together with ASC-seeded DAT control and DAT only controls, were implanted subcutaneously in nude mice (Nu-FoxN1nu) and harvested at 4 or 8 weeks for histological and immunohistochemical assessment. Human ASCs were tracked by immunostaining for human mitochondria. Angiogenesis was assessed using Masson’s trichrome staining to measure the diameter and depth of infiltration of blood vessels in the implants. Adipogenesis was measured using semi-quantitative analysis of perilipin immunostaining. The phenotype of infiltrating macrophages was assessed by co-labelling for the pan-macrophage marker, allograft inflammatory factor 1 (IBA1), and an anti-inflammatory marker, arginase 1 (Arg1).

Results: Similar numbers of human ASCs were observed in all ASC-seeded scaffolds after 4 and 8 weeks, regardless of the preconditioning strategy. Angiogenic assessment showed that the 2% O₂ dynamic group contained more blood vessels at both 4 and 8 weeks compared to all other groups. These vessels were larger in diameter and had greater infiltration at 8 weeks. Implants from the 2% O₂ dynamic group had more adipose tissue remodeling as compared to both control groups. Labelling of infiltrating macrophages suggested that the fraction of IBA1+ cells was similar across all preconditioned scaffolds at both 4 and 8 weeks. However, a greater proportion of IBA1+ cells were also Arg1+ at 8 weeks in the 2% O₂ dynamic scaffolds as compared to both control groups, suggesting that hypoxic preconditioning in the bioreactor may promote a more pro-regenerative macrophage response.

Discussion and Conclusions: The results of this study demonstrate that the 3-D preconditioning of human ASCs on the DAT under hypoxia promotes blood vessel formation, adipose tissue remodeling, and a more anti-inflammatory macrophage response. Further experiments will be performed to explore the mechanisms through which the dynamic culture strategy promotes a more pro-regenerative ASC phenotype.
11:00 - 11:15 A.M. PRESENTATIONS

Salon B

LOSS OF TIMP3 IS ASSOCIATED WITH INCREASED METALLOPROTEINASE ACTIVITY AND PULMONARY MICROVASCULAR ENDOTHELIAL CELL BARRIER DYSFUNCTION DURING SEPSIS

DEVNIKA JAYAWARDENA
PhD Candidate

Research Areas:
Circulatory
Mechanisms of disease

Supervisor(s):
S. Gill

Advisory Committee:
R. Veldhuizen, D. Fraser, L. Dagnino

Abstract:
Introduction: Sepsis, a systemic inflammatory response to infection leading to organ dysfunction, is one of the major causes of death in elderly people. Septic organ dysfunction is associated with microvascular endothelial cell (MVEC) dysfunction due to stimulation by soluble inflammatory mediators, including lipopolysaccharide (LPS) and cytokines (tumour necrosis factor [TNF] alpha, interleukin [IL] 1 beta, interferon [IFN] gamma). This septic MVEC dysfunction leads to a loss of MVEC barrier function and accumulation of protein-rich edema fluid, especially in the lungs. Metalloproteinases, including the matrix metalloproteinase (MMP) and a disintegrin and metalloproteinase (ADAM) families, may have a role in pulmonary MVEC (PMVEC) barrier dysfunction through degradation of inter-PMVEC junctional proteins. Further, tissue inhibitor of metalloproteinases (TIMP) 3 appears to be required for PMVEC barrier dysfunction as PMVEC lacking TIMP3 (Timp3-/- PMVEC) have increased leak under basal conditions. In this study, the role of TIMP3 in the regulation of septic PMVEC barrier dysfunction will be identified.

Hypothesis: TIMP3 is required to restrict metalloproteinase activity and PMVEC barrier dysfunction under septic conditions.

Materials and Methods: Wild type (WT) and Timp3-/- PMVEC will be stimulated with PBS or a combination of cytomix [equimolar TNFalpha, IL1beta, and IFNgamma] and lipopolysaccharide (LPS). Metalloproteinase activity (MMP12, MMP13 and ADAM17) will be assessed in conditioned media and cell lysate using commercially available assays and trans-PMVEC macromolecular flux assessed using Evans blue-labelled albumin. Further, confocal microscopy will be used to examine PMVEC surface localization of VE-cadherin (adherens junction) as well as occludin and claudin 5 (tight junction) on WT and Timp3-/- PMVEC under basal (PBS) and septic (cytomix/ LPS) conditions.

Results: Metalloproteinase activity was significantly increased in WT and Timp3-/- PMVEC under septic conditions leading to inter-PMVEC junctional protein degradation and PMVEC barrier dysfunction. Additionally, the metalloproteinase activity in Timp3-/- PMVEC appeared to be significantly augmented vs. WT PMVEC under both basal and septic conditions. Both WT and Timp3-/- PMVEC also had disrupted VE-cadherin and claudin 5 localization following septic stimulation. Further, in Timp3-/- PMVEC, the surface localization of VE-cadherin and claudin 5 appeared disrupted under both basal and septic conditions.

Discussion and Conclusions: Increased metalloproteinase activity under septic conditions is associated with disrupted inter-PMVEC junctional protein localization and loss of barrier function suggesting metalloproteinases are critical mediators of septic PMVEC barrier dysfunction. Further, PMVEC-derived TIMP3 appears to be an important regulator of metalloproteinase activity under septic conditions as loss of TIMP3 further increases metalloproteinase activity and subsequent PMVEC barrier dysfunction. Future studies will further examine the role TIMP3 in PMVEC barrier function by assessing metalloproteinase activity and degradation of inter-PMVEC junctional proteins in PMVEC overexpressing TIMP3. These studies will further define a novel endogenous homeostatic system promoting PMVEC barrier function that is disrupted in sepsis.
INVESTIGATING THE TRICEPS SURAE IN CHRONIC INFLAMMATORY Demyelinating Polyneuropathy Patients Using Magnetic Resonance Imaging

JACOB FANOUS
MSc Candidate

Research Areas:
Medical biophysics, engineering and imaging
Mechanisms of disease

Supervisor(s):
C. Rice

Advisory Committee:
N/A

Abstract:
Introduction: Chronic inflammatory demyelinating polyneuropathy (CIDP) is an acquired autoimmune disease, primarily characterized by peripheral nerve demyelination. Patients present with sensory and motor deficits, including but not limited to symmetrical diffused muscle weakness. Studies to date have mainly focused on the neuropathic aspects of CIDP and its involvement in paresis. Impairments in skeletal muscle quantity and quality may be a consequence of motor nerve deficits, but these aspects have not been investigated comprehensively. Thus, the purpose of this study was to use magnetic resonance imaging (MRI) to explore anatomical and functional differences in the triceps surae muscle complex in a group with CIDP with a healthy control group.

Hypothesis: We hypothesize that CIDP patients will have diminished plantar flexor strength compared with controls due to a reduction in both muscle quantity and quality in the triceps surae.

Materials and Methods: To date, five patients with CIDP and six healthy control subjects were matched on anthropomorphic characteristics. Both groups underwent isometric plantar flexion strength measurements on a custom dynamometer. On separate days, MRI (T1 and T2) of the leg musculature was acquired via serial axial plane scans in a 3.0-Tesla magnet. All MRI scans were analyzed using Osirix imaging processing software. Total muscle volume was computed using the T1 weighted anatomical images with a 3D FLASH sequence: (0.9mm slice thickness with slice separation of 1mm ranging from 280 to 400 slices). T2 weighted relaxation times were determined from images with a spin-echo sequence (5.0mm slice thickness; 16 echoes between 13.2-211.2ms). Both total volume (T1) and relaxation times (T2) were calculated separately for the three components of the triceps surae: soleus, medial gastrocnemius (MG), and lateral gastrocnemius (LG).

Results: CIDP patients had ~34% less plantar flexion strength compared with controls. CIDP patients had ~14%, ~28% and ~38% smaller muscle total volumes in the soleus, MG and LG, respectively, compared to controls. When strength was normalized to total triceps surae volume it was ~26% lower in CIDP. T2 relaxation times were significantly longer in CIDP with the soleus, MG and LG showing ~37%, ~31% and ~29% longer relaxation times, respectively.

Discussion and Conclusions: CIDP patients were significantly weaker compared to control subjects. When strength was normalized to total triceps surae volume the difference remained indicating CIDP patients have lower intrinsic muscle contractile quality. This is further reinforced by the significantly longer T2 relaxation times, which likely reflects an increase in intramuscular fat infiltration, in the CIDP group. The results indicate that muscle quantity and quality are affected by alterations in axonal function due to CIDP.

Supported by NSERC.
Abstract:

Introduction: Older, frail patients are at higher risk of adverse health outcomes. In perioperative settings, formal frailty assessment has been shown to reduce mortality. This study was to determine the cost-effectiveness of frailty assessment in older patients undergoing coronary artery bypass grafting (CABG) surgery, guide health policy decision-making regarding the incorporation of frailty screening in perioperative risk assessment, and quantify the value of further research in this area.

Hypothesis: We hypothesized that frailty assessment in older patients undergoing CABG is cost-effective, and health policy regarding the incorporation of frailty screening in perioperative assessment is promising.

Materials and Methods: We conducted a model-based cost-utility analysis for patients aged 65 years and older undergoing CABG in Ontario. A combined decision tree and Markov model was developed to compare costs and quality-adjusted life years (QALYs) over a 20-year time horizon associated with conducting frailty assessment prior to CABG with no frailty assessment. We used the estimates from the Frailty Screening Initiative Study, a time-series analysis of the 30, 180, and 365 days mortality among patients undergoing elective non-cardiac surgery. Evidence on the prevalence of frailty, sensitivity and specificity of frailty assessment, and mortality of CABG were searched in the literature and incorporated in the model. The perspective was the Ontario Ministry of Health and Long-Term Care, and we included hospital costs, physician costs, nursing, pharmacy, operating room, and laboratory costs. To characterize uncertainty, we conducted sensitivity analyses, scenario analyses, and probabilistic sensitivity analyses. We also conducted Expected Value of Perfect Information (EVPI) analysis to estimate the expected costs of uncertainty.

Results: The average lifetime cost per patient with frailty assessment was $251,891, more costly than no frailty assessment at $245,019. The quality-adjusted life years with frailty assessment was 8.836, 0.269 more effects than no frailty assessment. At the willingness-to-pay (WTP) threshold of $50,000, frailty assessment was cost-effective with an incremental cost-effectiveness ratio (ICER) of $25,510/QALY. Results were sensitive to annual cost one year after surgery and the average age of the patients. However, within the range of parameter distribution, ICERs remained cost-effective at a WTP threshold of $50,000/QALY. Scenario analyses in which health benefits for non-frail patients were removed demonstrated a marginal effect on the cost-effectiveness results. At a WTP threshold of $50,000/QALY, the probability of frailty assessment being cost-effective was 84.6%, and the EVPI per patient was $1,203.

Discussion and Conclusions: From the perspective of the Ontario Ministry of Health and Long-Term Care, frailty assessment can lead to improved health. The incorporation of frailty screening in perioperative risk assessment for older patients undergoing CABG may be cost-effective. Our study has several limitations. Mortality odds ratios were derived from patients undergoing elective, non-cardiac surgeries, which may not translate directly to the CABG patients. This study did not consider the effects of frailty assessment on complications such as readmission, stroke and major bleeding. To better understand the long-term benefits of frailty assessment, further studies are needed.
Salon E

PROMOTING HEALTHY DRINKING HABITS IN CHILDREN: RESULTS OF THE HEALTHY KIDS COMMUNITY CHALLENGE

BRIDGET IRWIN
MSc Candidate

Research Areas:
Population health and education
Prevention of diseases and health conditions and promotion of well-being

Supervisor(s):
J. Gilliland, M. Speechley

Advisory Committee:
C. O’Connor, P. Wilk

Abstract:

Introduction: Childhood obesity has emerged as a major public health concern of the 21st century and is associated with an increased risk of a number of non-communicable diseases and psycho-social problems. Sugar-sweetened beverage (SSB) consumption is a key risk factor for childhood obesity, contributing a substantial proportion of children’s daily calorie intake. As such, there is a growing movement among health practitioners and policymakers to replace SSBs in children’s diets with water.

Hypothesis: The objective of this study is to evaluate the effectiveness of London’s Healthy Kids Community Challenge (HKCC) ‘Water Does Wonders’ interventions on improving children’s beverage consumption habits. It is hypothesized that students in schools with new water infrastructure and nutrition and water literary interventions will demonstrate the greatest improvements in drinking habits, consuming more water and less SSBs, compared to those who receive only water infrastructure.

Materials and Methods: A quasi-randomized cluster trial enrolling grade 4-8 children at 16 elementary schools in 13 priority neighbourhoods across London, Ontario was conducted from September 2016 to June 2017. Clusters examined included: (1) schools receiving a water bottle filling station (5 schools, n=434); (2) schools receiving a water bottle filling station and a nutrition education intervention (5 schools, n=339); and (3) schools receiving a water bottle filling station and a water education intervention (6 schools, n=176). Socio-demographic, dietary, and nutrition and water knowledge information was collected using self-administered surveys at 2 time points. Generalized linear mixed models accounting for repeated measures and clustering were used to compare daily self-reported water and SSB consumption across the intervention groups before and after the interventions occurred.

Results: Baseline and follow-up data on 954 children were available. Preliminary analyses suggest an increase in daily water consumption across all groups from pre- to post-intervention, with the greatest difference observed in children receiving both a water bottle filling station and a nutrition education intervention, followed by those who received a water bottle filling station and a water education intervention, and those who received just a water bottle filling station. A slight increase in SSB consumption was also observed across all intervention groups, however this increase did not vary significantly between groups.

Discussion and Conclusions: The HKCC ‘Water Does Wonders’ activities in London, Ontario resulted in an increase in water intake among elementary school students, as well as an improvement in nutrition and water knowledge scores. These findings demonstrate that school-based environmental and education interventions may be effective at increasing children's water consumption. The observed increase in SSB consumption from pre- to post-intervention, however, suggests that consuming more water does not displace SSBs in children’s diets. Further interventions targeting SSBs specifically are thus needed. This study provides a greater understanding of the role of environmental and education interventions in improving children’s health behaviours. It will inform the development of future school-based water and SSB interventions to ensure students gain the maximum health benefit.
11:15 - 11:30 A.M. PRESENTATIONS

Salon B

GLUCOSE MODULATES TRANSFORMING GROWTH FACTOR SIGNALLING IN BONE MARROW-DERIVED PROGENITOR CELLS TO ENHANCE ADIPOGENESIS

JINA KUM
PhD Candidate

Research Areas:
Molecular cellular
Mechanisms of disease

Supervisor(s):
C. Howlett, Z. Khan

Advisory Committee:
E. Arany, N. Borradaile

Abstract:

Introduction: Enhanced marrow adiposity and skeletal fragility are chronic complications of diabetes mellitus, a prevalent disease characterized by hyperglycemia. We have shown that high glucose conditions enhance adipogenic differentiation of bone marrow-derived progenitor cells (bm-MPCs) while impairing osteoblastogenesis. Additionally, our laboratory has shown that the vascular dysfunction and inadequate repair in diabetes entail vasculogenic impairment due to the depletion of regenerative stem cells in the bone marrow. These findings suggest that changes in the marrow composition depletes the regenerative vasculogenic stem cells, and may lead to organ dysfunction in diabetes.

Hypothesis: Diabetes changes the cellular composition of the bone marrow, enhancing adipogenesis and suppressing osteogenesis, which alters the stem cell niche, ultimately depleting regenerative stem cells.

Materials and Methods: To identify signalling pathways that drive differentiation of marrow precursor cells into adipocytes and osteoblasts, gene expression changes were comprehensively profiled in primary human bm-MPCs upon differentiation into adipocytes and osteoblasts. Our profiles led to the discovery of a potential role of transforming growth factor-beta (TGF-beta) signalling pathway in marrow-derived cell differentiation. Hence, marrow-derived cells were challenged with exogenous TGF-beta 1 to assess for cellular and molecular alterations in both adipogenic and osteogenic induction media.

Results: Primary human bm-MPCs that were induced to differentiate into osteoblast downregulated the TGF-beta signalling pathway. However, exposure of marrow cells to high levels of glucose prevented this suppression, implicating normalization of the TGF-beta pathway as a mechanism of inhibited osteogenesis in diabetes. Interestingly, bm-MPCs that were challenged with exogenous TGF-beta 1 inhibited both adipogenic and osteogenic differentiation. To dissect the intracellular signalling proteins mediating the changes by TGF-beta 1, marrow-derived cells were cultured with various inhibitors of downstream proteins of the TGF-beta signalling pathway. We have identified that the inhibition of non-canonical TAK1-JNK axis essentially reversed the effect of TGF-beta 1 and normalized adipogenic differentiation. Interestingly, the canonical SMAD1/5 protein levels increased when cells were induced to differentiate into adipocytes, whereas exposing the cells to TGF-beta 1 normalized this induction.

Discussion and Conclusions: Our preliminary findings raise an interesting possibility that TGF-beta 1 may prevent cell differentiation by maintaining a precursor phenotype in marrow cells. Specifically, the precursor phenotype maintenance in marrow cells may be due to the active TAK1-JNK axis. In contrast, the induction of SMAD1/5 may play a role in cell differentiation. Our findings suggest that diabetes may fine-tune TGF-beta signalling to affect the balance between canonical and non-canonical pathways in marrow cells to induce differentiation. Specifically, with high levels of glucose evident in diabetes, the marrow cells will favour adipogenic differentiation. Future studies will build on these findings to determine whether altering this balance can inhibit enhanced adipogenesis in the marrow.
Salon B1

THE C-TERMINAL TO LisH (CTLH) COMPLEX IS A NOVEL MAMMALIAN E3 UBIQUITIN LIGASE

MATTHEW MAITLAND
PhD Candidate

Research Areas:
Molecular cellular
Mechanisms of disease

Supervisor(s):
C. Schild-Poulter, G. Lajoie

Advisory Committee:
G. DiMattia, G. Shaw

Abstract:
Introduction: RING domain E3 ubiquitin ligases mediate the transfer of ubiquitin to specific proteins, resulting in proteasomal degradation, conformational changes, or altered protein-protein interactions. This regulatory capacity and its presence in all tissue types have made ubiquitination a major research focus in almost all areas of human health and disease. The functionally uncharacterized C-terminal to LisH (CTLH) complex is the mammalian homologue of a yeast E3 ligase complex. CTLH complex subunit RMND5A has a RING domain that is conserved from yeast to mammals, but it is undetermined if this domain in mammalian RMND5A has ligase activity. Downregulation of RanBPM, a core complex member, contributes to cancer development in a mouse model and negatively regulates oncogenic signaling pathways, but its molecular mechanism to regulate these pathways is unknown.

Hypothesis: I hypothesize that the CTLH complex is an E3 ubiquitin ligase that regulates oncogenic signaling pathways.

Materials and Methods: Auto-ubiquitination assays: purified components of the ubiquitination mechanism were incubated with either RanBPM immunocomplexes for 30 minutes at 37 °C or bacterial extracts expressing GST-RMND5A for 2 hours at 37 °C and reactions were analyzed by direct imaging of fluorescent ubiquitin on a protein gel. Ubiquitination proteomics: protein extracts were made from control or RanBPM shRNA HeLa cells treated with 10 μM MG132 (proteasome inhibitor) for 4 hours. Trypsin digested ubiquitinated peptides (which have a unique diGLY motif) were enriched with a bead-conjugated diGLY antibody. Mass spectrometry analysis of the eluant was performed and label-free quantification of ubiquitin sites was completed using Maxquant and Perseus software.

Results: Using in vitro ubiquitination assays with either immunoprecipitated RanBPM or a bacterially expressed RMND5A, we show that the CTLH complex has E3 ubiquitin ligase activity. To gain insight into the functional outcomes of ubiquitination by the complex, we screened a panel of E2 conjugating enzymes and lysine ubiquitin mutants in the assays to determine the type of E2 that RMND5A pairs with and the poly-ubiquitin linkage that is generated, respectively. Next, we evaluated the function of RMND5A in human cells to compare it to the function of RanBPM. Consistent with RanBPM deficient cells, we found that RMND5A−/− HEK293 cells have increased cell growth and upregulation of oncogenic proteins. Finally, to identify ubiquitination targets of the complex, we used quantitative diGLY proteomics to measure ubiquitinated peptides in control and RanBPM shRNA HeLa cells. Over 800 ubiquitin sites are significantly downregulated in the knockdown compared to the control (n=4, p<0.05; fold change >2), which represent putative sites on proteins ubiquitinated by the CTLH complex. The most over-represented pathway in this list of proteins is glycolysis.

Discussion and Conclusions: We conclude that the CTLH complex is a new mammalian E3 ubiquitin ligase that has potentially hundreds of substrates in human cancer cells. The similarity between RMND5A and RanBPM deficient cells suggests that the complex as a whole has tumour suppressive functions. Next, we will explore the role of the complex in the ubiquitination of glycolytic enzymes and the regulation of cancer metabolism. Overall, the knowledge gained in this study on the function of the CTLH complex may lead to new opportunities for cancer therapeutics.

MATTHEW MAITLAND
PhD Candidate

THE C-TERMINAL TO LisH (CTLH) COMPLEX IS A NOVEL MAMMALIAN E3 UBIQUITIN LIGASE
**Salon D**

**CHARACTERIZATION OF PRE-GERMINAL CENTER B AND T CELL INTERACTIONS IN AUTO-ANTIGEN VERSUS FOREIGN ANTIGEN INDUCED IMMUNE RESPONSES**

**KATE PARHAM**  
Postdoctoral Scholar

**Research Areas:**  
Infection and immunity  
Mechanisms of disease

**Supervisor(s):**  
S. Kerfoot

**Advisory Committee:**  
N/A

**Abstract:**  
**Introduction:** Multiple Sclerosis is an autoimmune disease resulting from an immune attack directed against myelin in the central nervous system. This leads to demyelination of axons and over time, loss of neurons. The antomyelin autoimmune response is thought to be initiated within lymph nodes, where B and T cells specific for the same antigen (e.g. Myelin proteins) undergo cognate interactions leading to the initiation of a germinal center (GC) response, where B cells undergo terminal differentiation into memory B cells or plasma cells. These GC initiating B and T cell interactions have been elegantly characterized for foreign antigen immune responses, but little is known about them in the autoimmune context. In this study we aim to reveal differences in B and T cell interactions within the anti-myelin autoimmune response in comparison to foreign antigen induced immune responses, to identify potential autoimmune specific therapeutic targets.

**Hypothesis:** Pre-GC interactions between auto-antigen specific B and T cells are less robust than those between B and T cells specific for a foreign antigen.

**Materials and Methods:** To model B and T cell interactions we use two disparate model antigen systems; the model foreign antigen nitrophenyl conjugates-ovalbumin (NP-OVA) and the model auto-antigen myelin oligodendrocyte glycoprotein (MOG). Fluorescently labelled antigen specific B (GFP) and T (RFP) cells were transferred into dark recipient Smarta mice, which contain T cells specific for an irrelevant antigen (LCMV). Mice were immunized via the footpad with their respective antigen (NP-OVA or mMOGtag), and two days post immunization B and T cells within the popliteal lymph node were visualised in real-time using intravital multiphoton microscopy. Three dimensional movies of the developing immune response were generated and analyzed to identify interacting B and T cells and their interaction properties were then compared.

**Results:** Using the NP-OVA and MOG model antigen systems we have shown that the MOG GC response is not sustained and primarily produces B cells with a memory-like phenotype at the expense of plasma cell differentiation. Further, we show that this is in part controlled by the cognate T cells, indicating that B/T interactions are central to the outcome of the autoimmune response. In this study, multiphoton imaging was used to determine whether differences in pre-GC interactions could contribute to the differences in GC outcome. Analysis of 3D movies revealed, MOG-specific B and T cells interacted for a significantly shorter duration than B and T cells specific for the foreign-antigen NP-OVA. This was also evident in the proportion of B/T interactions that were greater than 15mins in duration (MOG - 0%, NP-OVA - 32.7%). The next step is to determine the interaction molecules mediating these conjugates in both model antigen systems by flow cytometry.

**Discussion and Conclusions:** We have shown that auto-antigen specific B and T cells have differential interaction kinetics when compared to those of foreign-antigen specific B and T cells at the pre-GC time point. Specifically, MOG-specific B and T cell interactions are significantly shorter than their foreign antigen counterparts, suggesting the interacting molecules that mediate these conjugates may be different. This difference in interacting molecules may provide novel therapeutic targets for autoimmune diseases, while leaving the helpful responses to foreign antigens untouched.
QUANTITATIVE PROTEOMICS PREDICTS THE THERAPEUTIC POTENTIAL OF HUMAN MULTIPOTENT STROMAL CELLS FOR β-CELL REGENERATION

MILJAN KULJANIN
Postdoctoral Scholar

Research Areas:
Translational research
Detection, screening and diagnosis of health and disease

Supervisor(s):
D. Hess, G. Lajoie

Advisory Committee:
D. Betts, D. Litchfield

Abstract:
Introduction: Human multipotent stromal cells (hMSC) have been described as one of the most versatile cell types for use in regenerative medicine applications. Most recently, it has been demonstrated that hMSC can stimulate the endogenous regeneration of damaged islets using hyperglycemic mouse models. However, the heterogeneous nature of hMSC populations isolated from different donors has been shown to impact β-cell regenerative potential. Assessing whether an hMSC line is regenerative reliant on lengthy in vivo assays and is inherently ineffective at screening a large number of donor derived hMSC lines for therapeutic potential relevant to β-cell regeneration. Therefore, examination of hMSC using high-throughput technologies are needed to improve existing therapies and to tailoring each hMSC line isolated for β-cell regeneration.

Hypothesis: β-cell regenerative hMSC secrete unique trophic factors that can be used to identify them via protein signature using high-throughput quantitative proteomics.

Materials and Methods: Media conditioned by hMSC was collected, filtered and quantified for co-culture and proteomic analyses. Proteomics was used to investigate previously characterized regenerative (R-hMSC) (3), non-regenerative (NR-hMSC) (2) and 5 uncharacterized (UC-hMSC) cell lines. Using quantitative proteomics and support vector machine (SVM) learning algorithms, an unbiased protein signature that could accurately distinguish R-hMSC and NR-hMSC was determined. This protein signature was validated using an additional 10 previously UC-hMSC lines by proteomics and was functionally validated using human islet in vitro assays and streptozotocin (STZ)-treated hyperglycemic mouse model in vivo.

Results: Quantitative proteomics was used to determine what factors could best identify and place UC-hMSC lines into either β-cell regenerative or non-regenerative classes. Unbiased mining of secreted proteins revealed that 16 proteins met a very stringent statistical cut-off (0.95) that could best segregate UC-hMSC lines into either R-hMSC or NR-hMSC classes. Proteins obtained from the SVM suggested that 12 proteins were upregulated (4-32-fold) in NR-hMSC while 4 were upregulated (16-32-fold) in β-cell R-hMSC lines. The accuracy of this protein signature was assessed using 10 UC-hMSC and was able to accurately assign 1 as R-hMSC and 9 as NR-hMSC lines. Functional validation of each UC-hMSC lines was achieved by using a human islet culture system and multiparametric flow cytometry. The total-live human β-cell number was significantly increased when cultured with β-cell R-hMSC compared to NR-hMSC. In addition, STZ-treated hyperglycemic mice that were transplanted with NR-hMSC showed no improvement of systemic blood glucose after 42 days, while mice transplanted with R-hMSC showed significant improvement in systemic blood glucose.

Discussion and Conclusions: Our study demonstrates that quantitative proteomics can be used to predict the β-cell regenerative potential in a high-throughput fashion of previously uncharacterized hMSC lines. Using machine learning algorithms, an unbiased protein signature of β-cell regenerative hMSC could be efficiently determined and used to reliably predict the regenerative potential of 15 UC-hMSC samples. Following functional validation, using in vivo models, this protein signature proved to be robust and offers an additional tool to be used in an attempt to increase regeneration of β-cells in patients with diabetes.
11:30 - 11:45 A.M. PRESENTATIONS

Salon B

PREFRONTAL REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION (rTMS) FOR IMPROVING MOBILITY AND COGNITION IN OLDER ADULTS WITH EXECUTIVE DYSFUNCTION: METHODOLOGY AND PRELIMINARY RESULTS

ANISH NAIDU
Medical Student

Research Areas:
Neuroscience
Prevention of diseases and health conditions and promotion of well-being

Supervisor(s):
M. Montero-Odasso, A. Burhan

Advisory Committee:
N/A

Abstract:
Introduction: Mobility impairment leading to falls is not uncommon among seniors, and this is a significant cause of morbidity and mortality. Recent research shows that decline in cognition with age has a negative impact on mobility performance, specifically dual-task gait. Dual-task gait refers to walking while performing a cognitive challenge. A larger increase in gait variability from the baseline when dual-tasking is associated with a higher risk of falls. Emerging evidence suggests that decline in executive function is independently associated with poor dual-task gait performance and future falls. Repetitive transcranial magnetic stimulation (rTMS) is a safe, electrodeless technique to modulate the excitability of cortical areas depending on the field frequency and strength. rTMS has been shown to enhance motor learning and working memory, however, it has not yet been explored as a means to improve executive function mediated gait. This pilot study aims to explore the effect of high-frequency rTMS applied to the prefrontal cortex bilaterally on the gait performance in seniors with executive dysfunction.

Hypothesis: Application of prefrontal rTMS in older adults (> 60 y.o.) with executive dysfunction improves their dual-task gait performance and executive function measured at one week post treatment.

Materials and Methods: Forty community-dwelling seniors over the age of 60 with executive dysfunction (MoCA EIS < 11) will be recruited from geriatric medicine and psychiatry clinics in London, Ontario. Individuals with Alzheimer’s disease, severe depression, MSK or neurological disorders affecting gait, or contraindication to rTMS are excluded. Baseline clinical, cognitive and gait assessments are performed prior to randomizing participants to active vs. sham rTMS (45% tilted coil). The usual and dual-task gait performance is recorded using a pressure-sensitive gait mat. The participants receive five active or sham consecutive daily sessions of high-frequency rTMS (20 Hz, 1200 pulses) to the right then left prefrontal areas. The participants and assessors are blinded, and only the TMS operator is aware of the study assignment. Cognitive and gait assessments are repeated immediately after the last session (day 5) and at one week post intervention (day 12).

Results: Eight participants have completed the study thus far (4 active and 4 sham). rTMS was well tolerated with only one drop out due to head discomfort. The active and sham groups are similar in the baseline characteristics of age, sex, and number of medications and comorbidities. The improvement in dual-task (naming animals) gait variability at day 12 compared to day 1 was appreciably higher in the active vs. the sham group (103.8 ± 97.5 vs. 38.4 ± 90.4; p=0.08). Also, a significantly greater improvement in short-term memory (RAVLT-5 min word recall) was noted in the active vs. the sham group (6.5 ± 2.5 vs. 0.0 ± 4.1; p=0.04).

Discussion and Conclusions: The preliminary results suggest that prefrontal high-frequency rTMS shows promise in improving gait performance in seniors. This pilot study will allow the measurement of the effect size and enable the design of a larger, well-powered study to confirm the utility of rTMS in treating prefrontal cortex mediated gait impairment in seniors. If rTMS is found to be effective, it may help slow down mobility decline in seniors, reduce their risk of falls, and improve their quality of life.
Salon B1

DEVELOPMENT OF AN INSULAR ISCHEMIC STROKE ANIMAL MODEL TO STUDY THE PATHOPHYSIOLOGY OF ATRIAL FIBRILLATION DETECTED AFTER STROKE (AFDAS)

VICTORIA THORBURN
MSc Candidate

Research Areas:
Neuroscience
Mechanisms of disease

Supervisor(s):
S. Whitehead, L. Sposato

Advisory Committee:
P. Walton, R. Rajakumar

Abstract:
Introduction: Atrial fibrillation (AF) is associated with a 5-fold increased risk of ischemic stroke. Recent clinical evidence suggests that stroke can initiate a neurogenic cause of AF, referred to as AF detected after stroke (AFDAS). Specifically, it is thought that stroke involving the insular cortex (IC) contributes to this development of AFDAS, as the IC maintains important regulation of heart rhythm. However, the exact mechanisms underlying this brain-heart paradigm remain unknown. As a first step of an overall experimental initiative to evaluate the pathophysiology of AFDAS, we aimed to develop a rat model of focal insular ischemic stroke to better understand the downstream consequences of insular stroke.

Hypothesis: We hypothesize that AFDAS is the consequence of insular cortex damage occurring after stroke, which disrupts autonomic regulation of heart rhythm.

Materials and Methods: Focal ischemic stroke was induced into either the right (n=8) or left (n=8) insular cortex of 6-month-old male Wistar rats through stereotaxic injection of endothelin-1 (ET-1). Control groups received saline injection (n=7 right IC / n=7 left IC) or no injection (n=6). At 28 days post-stroke, rats were euthanized. Heart tissue was collected and histologically analyzed for left atrial fibrosis (using Masson’s Trichrome stain). Brain tissue was collected and analyzed to identify the extent of local and remote cerebral inflammation, visualized by the presence of activated microglia (using an OX-6 immunohistochemistry stain).

Results: Left atrial fibrosis was greater in animals with right IC stroke (5.8±1.2%) compared to those with right IC saline injection (0.6±0.1%; p=0.021) and no injection (0.6±0.1%; p=0.004), as well as in animals with left IC stroke (4.5±1.3%) compared to those with left IC saline injection (0.7±0.1%; p=0.020) and no injection (0.6±0.1%; p=0.007). Statistical analysis was performed using a one-way ANOVA followed by a Bonferroni post-hoc test. Additionally, qualitative results indicate a widespread neuroinflammatory response present within the forceps minor of the prefrontal cortex, anterior commissure, striatum, corpus callosum (ipsilateral and contralateral to injection) and posterior ventromedial thalamus (ipsilateral to injection) of both right IC stroke and left IC stroke animals, compared to control groups.

Discussion and Conclusions: We have successfully developed a focal insular ischemic stroke animal model and identified several downstream consequences of insular stroke. Rats subjected to ET-1 induced insular ischemic stroke show significantly greater left atrial fibrosis and secondary neuroinflammation. These findings provide insight into potential mechanisms of post-stroke atrial fibrillation, serving as possible future therapeutic targets for AFDAS.
ROLE OF DOUBLECORTIN-LIKE KINASE 1 (Dclk1) POSITIVE TUFT CELLS IN COLITIS-ASSOCIATED COLORECTAL CANCER

ALICE SHIN
PhD Candidate

Research Areas:
Cancer biology
Mechanisms of disease

Supervisor(s):
S. Asfaha

Advisory Committee:
F. Dick, Z. Khan

Abstract:
Introduction: Colorectal cancer (CRC) is the second leading cause of cancer death in Canada, with the major risk factor being chronic inflammation. However, how inflammation leads to cancer is not well understood. Our recent work has focused on a colonic epithelial cell known as the tuft cell that uniquely expresses the protein doublecortin-like kinase 1 (Dclk1). Using Cre-dependent lineage tracing of Dclk1-expressing cells, we previously showed that Dclk1 labels long-lived quiescent cells in the colon that serve as a cellular origin of CRC upon dextran sulfate sodium (DSS)-induced inflammatory injury.

Hypothesis: The aim of this study was to determine the generalizability of inflammation-induced tumor promotion from genetically susceptible Dclk1+ cells and explore the mechanism by which inflammation induces tuft cell cancer initiation. We hypothesized that colonic inflammatory insults lead to dedifferentiation of Dclk1+ tuft cells to a stem cell state susceptible to tumor initiation.

Materials and Methods: To investigate the various forms of injury or infection that can activate quiescent tuft cells, we generated tamoxifen-inducible Cre transgenic mice that allow for Dclk1+ cell lineage tracing and cell-specific knock-out of the tumor suppressor adenomatous polyposis coli (APC) (Dclk1-CreERT2/R26-tdTom/APCfl/fl). Following tamoxifen induction, mice were administered colitis-inducing agents dextran sodium sulfate (DSS), trinitrobenzene sulfonic acid (TNBS), oxazolone or Citrobacter rodentium. To examine the role of dedifferentiation in colonic tumor initiation, we ablated Lgr5+ intestinal stem cells (ISCs) post DSS-induced colitis in our Dclk1/APCf/f mice. To further investigate the mechanism by which inflammation contributes to tuft cell activation, we constitutively activated IKKβ, a positive regulator of NF-κB signaling, in APC-mutated Dclk1+ cells (Dclk1/APCf/f/R-IKKβca).

Results: Treatment with DSS, TNBS, oxazolone, or C. rodentium induced colonic inflammation as detected by significantly increased myeloperoxidase (MPO) activity and histologic analysis. DSS administration led to Dclk1+ cell-derived colonic tumors as previously reported. Surprisingly, administration of TNBS, oxazolone, or C. rodentium in Dclk1/APCf/f mice did not lead to colonic tumorigenesis up to 52 weeks following induction of colitis. Interestingly, ablation of Lgr5+ ISCs post colitis significantly reduced colonic tumors in DSS-treated Dclk1/APCf/f mice. Furthermore, constitutive activation of IKKβ in Dclk1+ cells resulted in reduced epithelial injury in DSS-induced colitis and fewer colonic tumors.

Discussion and Conclusions: Our data suggests that an inflammatory response unique to DSS-induced colitis, and not TNBS, oxazolone or C. rodentium infection, results in colonic tumor formation. Interestingly, the colonic transformation of Dclk1+ tuft cells in DSS colitis appears to be mediated through Lgr5-expressing cells. Dclk1+ cell-specific NF-κB signaling appears to affect the severity of colitis and tumorigenesis. These findings provide insight into the molecular mechanisms by which Dclk1-derived colonic tumors arise.
Salon E

TAM RECEPTORS ACTIVATE P90RSK/mTORC1 SIGNALLING TO MEDIATE ACQUIRED RESISTANCE TO PI3K INHIBITION IN HEAD AND NECK SQUAMOUS CELL CARCINOMA

KARA RUICCI
PhD Candidate

Research Areas:
Cancer biology
Mechanisms of disease

Supervisor(s):
A. Nichols

Advisory Committee:
T. Shepherd, F. Dick

Abstract:

Introduction: Head and neck squamous cell carcinoma (HNSCC) is the sixth most common cancer worldwide, with an incidence of >600,000 new cases per year, and a 50% mortality rate. PIK3CA—which encodes the PI3K alpha (α) isoform of PI3-kinase—is the most frequently altered actionable target in HNSCC. Small-molecule PI3K inhibitors are under active investigation and have shown early promise in clinical trials; however, the development of resistance limits the utility of these agents over time. In the present study, we aimed to elucidate mechanisms of acquired resistance to the PI3K inhibitor Alpelisib (BYL719) in HNSCC, in order to identify potential secondary therapeutic targets.

Hypothesis: Prolonged PI3K inhibition will lead drug resistance in vitro and in vivo, causing systems to develop new signalling pathway dependencies for survival.

Materials and Methods: We developed multiple BYL719-resistant HNSCC cell lines by escalating drug treatment over time. Simultaneously, we have established multiple patient derived xenograft (PDX) models of acquired drug resistance by treating xenografts with BYL719 (50mg/kg) for a prolonged (>100 days) period, which, on its own, is a novel experimental model. Using reverse phase protein arrays (RPPA) we examined differentially-expressed proteins and phospho-proteins and validated these findings in vitro using immunoblotting, flow cytometry and quantitative real-time PCR (qRT-PCR).

Results: Over time, both cell line and PDX models acquired resistance to BYL719, highlighting the likelihood of this phenomenon when PI3K inhibitors are used clinically. We identified TAM family receptor tyrosine kinases (RTKs) Tyro3, Axl and Mertk to be upregulated in BYL719-resistant models. Downstream, we observed hyper-activation of ERK/P90RSK signalling, leading to sustained, PI3K-independent mTORC1 activation and cell survival.

Discussion and Conclusions: We have interrogated the landscape of acquired resistance to PI3K inhibitor BYL719 in HNSCC and identified upregulation of TAM RTKs as a mechanism of acquired resistance. We have further explored TAM-mediated activation of ERK/P90RSK signalling and found this pathway to be involved with resistance to BYL719. Our findings highlight novel targets for either combinatorial, or second-line therapies that may prevent or delay therapeutic resistance to PI3K inhibitors.
# POSTER PRESENTATIONS

## AFTERNOON SESSION

1:30 - 3:15 p.m.  
Ballroom A, Second Floor

<table>
<thead>
<tr>
<th>Category</th>
<th>Poster Number</th>
<th>Page Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>1 - 28</td>
<td>42 - 43</td>
</tr>
<tr>
<td>Advancing health services provision and health policy</td>
<td>29 - 37</td>
<td>43 - 44</td>
</tr>
<tr>
<td>Detection, screening and diagnosis of health and disease</td>
<td>38 - 70</td>
<td>44 - 46</td>
</tr>
<tr>
<td>Determinants of health</td>
<td>71 - 79</td>
<td>46</td>
</tr>
<tr>
<td>Early life programming and development</td>
<td>80 - 89</td>
<td>47</td>
</tr>
<tr>
<td>Mechanisms of disease</td>
<td>90 - 151</td>
<td>47 - 51</td>
</tr>
<tr>
<td>Populations, public health and education</td>
<td>152 - 161</td>
<td>51</td>
</tr>
<tr>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>162 - 175</td>
<td>52</td>
</tr>
</tbody>
</table>
ADVANCES IN STRUCTURAL AND PHYSIOLOGICAL TREATMENT OF DISEASE AND THERAPEUTIC INTERVENTION (INCLUDES SURGERY AND DRUGS)

Poster Number: 1
Name: Ehsan Abolhasani
Degree: MSc Candidate
Abstract Title: Operant conditioning of short-latency stretch reflex
Supervisor(s): A. Pruszynski

Poster Number: 2
Name: David Axford
Degree: MSc Candidate
Abstract Title: Development of a 3D force measurement system for a glenoid reaming simulator
Supervisor(s): L. Ferreira, J. Johnson

Poster Number: 3
Name: Sumit Chaudhari
Degree: Resident
Abstract Title: Sudarshan Kriya Yoga (SKY) in post-traumatic stress disorder: A feasibility study
Supervisor(s): K. Vasudev

Poster Number: 4
Name: Lucy Chu
Degree: Resident
Abstract Title: Immunosuppression for primary Sjogren’s syndrome: Is it time for change?
Supervisor(s): J. Pope

Poster Number: 5
Name: Brendan Daisley
Degree: MSc Candidate
Abstract Title: Lactobacilli indirectly modulate in vitro expression of xenobiotic-metabolizing CYP3A4 via biotransformation of primary bile acids
Supervisor(s): G. Reid

Poster Number: 6
Name: Andrew Deweyert
Degree: PhD Candidate
Abstract Title: Intratumoral modulation therapy for diffuse intrinsic pontine glioma: A novel electrotherapeutic strategy for patient-derived DIPG cells
Supervisor(s): M. Hebb, S. Schmid

Poster Number: 7
Name: Asmahan Elsariti
Degree: MSc Candidate
Abstract Title: Ambroxol increases GM1 in neurons and protects against oxygen glucose deprivation
Supervisor(s): S. Whitehead

Poster Number: 8
Name: Benjamin Fuhrmann
Degree: MSc Candidate
Abstract Title: Regulation of NK cell cytotoxicity by TEC expression of Clr proteins in kidney Ischemia Reperfusion Injury (IRI)
Supervisor(s): Z. Zhang, A. Jevnikar

Poster Number: 9
Name: Derek Gillies
Degree: PhD Candidate
Abstract Title: Making it easier to see: Applicator segmentation for interventional liver therapies in three-dimensional ultrasound
Supervisor(s): A. Fenster

Poster Number: 10
Name: Nadezda Ivanova
Degree: PhD Candidate
Abstract Title: Negative impact of Ketoprofen-lysine treatment on cerebral pathology and cognition in Hypercaloric diet-induced metabolic syndrome and Alzheimer’s disease rat study
Supervisor(s): D. Cechetto

Poster Number: 11
Name: Ankita Kambli
Degree: MSc Candidate
Abstract Title: Utilizing cats to study HIV: Generation of a novel animal model
Supervisor(s): R. Troyer, E. Arts

Poster Number: 12
Name: Andrew Khalil
Degree: MSc Candidate
Abstract Title: Avoiding painful palatal injections at the dentist - articaine buccal injection anesthetizes the palatal mucosa: A cadaveric study on the maxillary alveolar bone
Supervisor(s): K. Galil, T. Wilson

Poster Number: 13
Name: Esther Lau
Degree: Postdoctoral Scholar
Abstract Title: Impact of robotic-assistance on mental workload and cognitive performance of surgical trainees performing a complex minimally invasive suturing task
Supervisor(s): N. Alkhamesi, C.M. Schlachta

Poster Number: 14
Name: Ji Yun Lee
Degree: PhD Candidate
Abstract Title: Donor Kidney Molecule-1 promotes graft recovery by regulating systemic necroinflammation
Supervisor(s): L. Gunaratnam

Poster Number: 15
Name: Xinyi Li
Degree: PhD Candidate
Abstract Title: A multifunctional nano-on-micro drug delivery system
Supervisor(s): W. Wan

Poster Number: 16
Name: Borna Mahmoudian
Degree: MSc Candidate
Abstract Title: Role of primate amygdala in gaze direction discrimination
Supervisor(s): J. Martinez
Poster Number: 17
Name: Pascal Morissette Martin
Degree: PhD Candidate
Abstract Title: Adipose-derived bioscaffolds enhance the survival and angiogenic phenotype of human chronic wound dermal fibroblasts relative to collagen-derived bioscaffolds
Supervisor(s): L. Flynn

Poster Number: 18
Name: Martina Mudri
Degree: MSc Candidate
Abstract Title: Using a surgical rabbit model of congenital diaphragmatic hernia to explore the effects of tracheal occlusion on fetal lung development
Supervisor(s): A. Bütter, T. Regnault

Poster Number: 19
Name: Nathan Orlando
Degree: PhD Candidate
Abstract Title: Quantifying needle deflection in high dose rate prostate cancer brachytherapy
Supervisor(s): A. Fenster

Poster Number: 20
Name: Darshit Patel
Degree: MSc Candidate
Abstract Title: Leveraging antigen-specific B cells for targeted endogenous production of anti-HIV broadly neutralizing antibodies
Supervisor(s): Y. Gao

Poster Number: 21
Name: Alexander C Roy
Degree: PhD Candidate
Abstract Title: Improving the versatility of the Tev-Cas9 dual endonuclease
Supervisor(s): D. Edgell

Poster Number: 22
Name: Olivia Samotus
Degree: PhD Candidate
Abstract Title: Investigating the therapeutic and transcortical effects of spinal cord stimulation for gait dysfunction in Parkinson’s disease patients
Supervisor(s): M. Jog

Poster Number: 23
Name: Corey Smith
Degree: PhD Candidate
Abstract Title: Development of force-space navigation for surgical robotics
Supervisor(s): L. Ferreira

Poster Number: 24
Name: Nicholas Tonial
Degree: PhD Candidate
Abstract Title: Effect of chronic kidney disease on expression and activity of CYPs and OATPs in rodent models and clinical studies
Supervisor(s): B. Urquhart, M. Weir

Poster Number: 25
Name: Charles Treford
Degree: PhD Candidate
Abstract Title: The role of sequestosome 1 in mediating TGF-β dependent EMT and autophagy
Supervisor(s): J. Di Guglielmo

Poster Number: 26
Name: Jason Vickress
Degree: PhD Candidate
Abstract Title: Online assessment of dose changes in head and neck radiotherapy without dose re-computation using deformable image registration.
Supervisor(s): S. Yartsev, R. Barnett

Poster Number: 27
Name: Annette Wong
Degree: Research Assistant/Associate
Abstract Title: The effectiveness of video discharge instructions for acute otitis media in children: A randomized controlled trial
Supervisor(s): N. Poonai

Poster Number: 28
Name: Cuilin Zhu
Degree: PhD Candidate
Abstract Title: The impact of GDF15 in renal ischemia reperfusion injury
Supervisor(s): X. Zheng

ADVANCING HEALTH SERVICES PROVISION AND HEALTH POLICY

Poster Number: 29
Name: Megan Chang
Degree: Research Assistant/Associate
Abstract Title: The burden of ICU survivorship at LHSC
Supervisor(s): M. Slessarev

Poster Number: 30
Name: Mojgan Farahani
Degree: PhD Candidate
Abstract Title: Listener comfort with alaryngeal voices: A paired comparison and slider rating study
Supervisor(s): P. Doyle, V. Parsa

Poster Number: 31
Name: Nima Gheisarzadeh
Degree: MSc Candidate
Abstract Title: Off-label use of second generation antipsychotics in primary care in southwestern Ontario
Supervisor(s): D. Lizotte, K. Anderson

Poster Number: 32
Name: Markus Gulilat
Degree: PhD Candidate
Abstract Title: Interpatient variation in apixaban plasma concentrations in routine care
Supervisor(s): R. Kim
**Poster Number: 33**  
**Name:** Boniface Harerimana  
**Degree:** PhD Candidate  
**Abstract Title:** Developing and evaluating an addiction care model of patients’ motivation for engagement and retention in the addiction recovery process in Rwanda: A prospective predictive study  
**Supervisor(s):** M. Kerr  

**Poster Number: 34**  
**Name:** Kaitlyn Hayes  
**Degree:** Medical Student  
**Abstract Title:** Evolving patterns of reactive arthritis  
**Supervisor(s):** J. Pope  

**Poster Number: 35**  
**Name:** Michael Hong  
**Degree:** MSc Candidate  
**Abstract Title:** After-hours access to primary care and emergency department utilization: A systematic review  
**Supervisor(s):** S. Sarma  

**Poster Number: 36**  
**Name:** Elizabeth Muchiri  
**Degree:** PhD Candidate  
**Abstract Title:** Patients’ perspectives of bedside handover: An integrated review  
**Supervisor(s):** R. Booth, M. Kerr  

**Poster Number: 37**  
**Name:** Michael Riddle  
**Degree:** MSc Candidate  
**Abstract Title:** Wearable technology  
**Supervisor(s):** E. Lalone, L. Ferreira  

**DETECTION, SCREENING AND DIAGNOSIS OF HEALTH AND DISEASE**

**Poster Number: 38**  
**Name:** Parvaneh Abbasalipour  
**Degree:** PhD Candidate  
**Abstract Title:** Listeners with unilateral conductive hearing loss suffer from abnormal loudness summation  
**Supervisor(s):** E. Macpherson  

**Poster Number: 39**  
**Name:** Khalid Abdalla  
**Degree:** MSc Candidate  
**Abstract Title:** Using magnetic resonance imaging to characterize the infarcted myocardium by R2+ mapping  
**Supervisor(s):** D. Goldhawk, N. Gelman  

**Poster Number: 40**  
**Name:** Monisha Basu  
**Degree:** Research Assistant/Associate  
**Abstract Title:** The Canadian collaboration on neurodegeneration and aging – Platform 1 – COMPASS-ND study: Planning and implementation  
**Supervisor(s):** M. Borrie, J. Wells, S. Best  

**Poster Number: 41**  
**Name:** Amanda Berberich  
**Degree:** MSc Candidate  
**Abstract Title:** Copy number variation analysis in the diagnosis of MODY  
**Supervisor(s):** R. Hegele  

**Poster Number: 42**  
**Name:** Riley Bloomfield  
**Degree:** PhD Candidate  
**Abstract Title:** Quantifying knee range of motion using wearable sensors in osteoarthritis patients  
**Supervisor(s):** M. Teeter, K. McIsaac  

**Poster Number: 43**  
**Name:** Charmaine Cruje  
**Degree:** PhD Candidate  
**Abstract Title:** Lanthanide nanoparticles as vascular contrast agents for pre-clinical computed tomography  
**Supervisor(s):** M. Drangova, E. R. Gillies  

**Poster Number: 44**  
**Name:** Praveen Dassanayake  
**Degree:** MSc Candidate  
**Abstract Title:** Monitoring inflammation using THP-1 monocytes and magnetic resonance  
**Supervisor(s):** D. Goldhawk  

**Poster Number: 45**  
**Name:** Sergio Dempsey  
**Degree:** MSc Candidate  
**Abstract Title:** Determining In-Silico left ventricular contraction force of myocardial infarct tissue using a composite material model  
**Supervisor(s):** A. Samani  

**Poster Number: 46**  
**Name:** Janice Gomes  
**Degree:** MSc Candidate  
**Abstract Title:** Development of a microparticle-based tool as an indicator of the effectiveness of dialysis treatment  
**Supervisor(s):** C. McIntyre  

**Poster Number: 47**  
**Name:** Amanda Hamilton  
**Degree:** Research Assistant/Associate  
**Abstract Title:** Engineering non-integrating lentiviral vectors for safe reporter-based imaging of mesenchymal stem cells  
**Supervisor(s):** J. Ronald  

**Poster Number: 48**  
**Name:** Andrew Harris  
**Degree:** PhD Candidate  
**Abstract Title:** Image registration for monitoring of neonatal intraventricular hemorrhage using 3D ultrasound  
**Supervisor(s):** A. Fenster
Poster Number: 49
Name: Khalil Hetou
Degree: Resident
Abstract Title: Mean of maximum standardized uptake value (SUV max) from PET imaging: A possible predictive parameter for locally advanced prostate cancer
Supervisor(s): J. Chin, G. Bauman

Poster Number: 50
Name: James Hughes
Degree: PhD Candidate
Abstract Title: Modelling intracranial pressure with noninvasive measures and genetic programming
Supervisor(s): M. Daley

Poster Number: 51
Name: Seva Ioussoufovitch
Degree: MSc Candidate
Abstract Title: Assessing early treatment response in an animal model of rheumatoid arthritis by measuring joint blood flow
Supervisor(s): M. Diop

Poster Number: 52
Name: Jason Kai
Degree: MSc Candidate
Abstract Title: Investigating quantitative and structural differences in short association, U-shaped fibres in temporal lobe epilepsy
Supervisor(s): A. Khan

Poster Number: 53
Name: Nikolas Knowles
Degree: PhD Candidate
Abstract Title: Revision shoulder arthroplasty after failed shoulder arthroplasty: A systematic review and comparison of North American versus European outcomes and complications
Supervisor(s): L. Ferreira

Poster Number: 54
Name: Yong Lim
Degree: MSc Candidate
Abstract Title: Characterization of the metabolome and renal tubular cisplatin disposition in cisplatin induced acute kidney injury
Supervisor(s): B. Urquhart

Poster Number: 55
Name: Matthew Mouawad
Degree: PhD Candidate
Abstract Title: Large signal enhancement volume changes in DCE-MRI after single fraction SBRT of early stage breast cancer
Supervisor(s): S. Gaede, N. Gelman

Poster Number: 56
Name: Patrick Park
Degree: Research Assistant/Associate
Abstract Title: A framework for reproducible evaluation of geometric inhomogeneity in magnetic resonance images
Supervisor(s): A. Khan

Poster Number: 57
Name: Alexander Pavlosky
Degree: Medical Student
Abstract Title: Validation of an effective, low cost, free/open access 3D-printed stethoscope
Supervisor(s): T. Loubani

Poster Number: 58
Name: Ana-Bianca Popa
Degree: MSc Candidate
Abstract Title: Eliciting perceptual errors to detect cognitive changes in early psychosis
Supervisor(s): I. Johnsrude

Poster Number: 59
Name: Wen Qin
Degree: MSc Candidate
Abstract Title: Identifying non-histone lysine methylated proteins via AP-MS/MS
Supervisor(s): S. Li

Poster Number: 60
Name: Ajay Rajaram
Degree: PhD Candidate
Abstract Title: A novel optical neuromonitor for simultaneous quantification of cerebral perfusion and metabolism in preterm brain injury
Supervisor(s): K. St. Lawrence, M. Diop

Poster Number: 61
Name: Hossein Rejali
Degree: MSc Candidate
Abstract Title: Investigating anatomical regions in which myelin abnormalities occur in schizophrenia using quantitative R1 maps
Supervisor(s): A. Khan

Poster Number: 62
Name: Dillon Richards
Degree: PhD Candidate
Abstract Title: Comparison of SCAT-3 baseline testing and electroencephalograph from season to season in university football players
Supervisor(s): J. Dickey, T. Birmingham

Poster Number: 63
Name: Kavin Selvan
Degree: Research Assistant/Associate
Abstract Title: Monitoring tissue oxygenation in ICU patients using hyperspectral NIRS
Supervisor(s): M. Diop

Poster Number: 64
Name: Marudan Sivagurunathan
Degree: PhD Candidate
Abstract Title: Perceptions of preparedness and intimate partner violence screening practices amongst hand therapists
Supervisor(s): J. MacDermid
Poster Number: 65
Name: Ahmed Tanashi
Degree: MSc Candidate
Abstract Title: Evaluation of finger kinematics for analysis and improvement of joint protection programs
Supervisor(s): E. Lalone

Poster Number: 66
Name: Olivia Tong
Degree: MSc Candidate
Abstract Title: Non-contact imaging of breast surface for breast surgeries
Supervisor(s): M. Diop, J. Carson

Poster Number: 67
Name: Matthew Turk
Degree: MSc Candidate
Abstract Title: Physician Global assessments for disease activity in rheumatoid arthritis are all over the map!
Supervisor(s): J. Pope

Poster Number: 68
Name: Hui Wang
Degree: PhD Candidate
Abstract Title: Development of a holography camera for non-contact photoacoustic tomography of the breast
Supervisor(s): M. Diop, J. Carson

Poster Number: 69
Name: TianDuo Wang
Degree: MSc Candidate
Abstract Title: Developing tumour-activatable minicircles as a novel platform for prostate cancer detection
Supervisor(s): J. Ronald

Poster Number: 70
Name: Dae-Myoung (Danny) Yang
Degree: PhD Candidate
Abstract Title: Tumor response in non-small cell lung cancer after stereotactic ablative radiation therapy: Hybrid dynamic 18F-FDG PET and CT perfusion study
Supervisor(s): T. Lee

DETERMINANTS OF HEALTH

Poster Number: 71
Name: Natalie Au
Degree: MSc Candidate
Abstract Title: Exploring risk factors of non-severe hypoglycemia in Type 2 Diabetes Mellitus: Results from the InHypo-DM Study (Canada)
Supervisor(s): S. Harris

Poster Number: 72
Name: Dhwanil Bhatty
Degree: Research Assistant/Associate
Abstract Title: Predictors of vocational status among persons with multiple sclerosis
Supervisor(s): M. Blair, S. Morrow

Poster Number: 73
Name: Avyarthana Dey
Degree: MSc Candidate
Abstract Title: Searching for a stratification marker for antioxidant use in schizophrenia and bipolar disorder: A meta-analysis of MRS studies of anterior cingulate glutathione
Supervisor(s): L. Palaniyappan

Poster Number: 74
Name: Uzair Hussain
Degree: Postdoctoral Scholar
Abstract Title: Design and evaluation of a diffusion MRI fibre phantom using 3D printing
Supervisor(s): A. Khan, C. Baron

Poster Number: 75
Name: Stephen Klassen
Degree: PhD Candidate
Abstract Title: The role of the paravertebral ganglia in sympathetic vasoconstrictor neural discharge patterns
Supervisor(s): K. Shoemaker

Poster Number: 76
Name: Sophie Laramee
Degree: MSc Candidate
Abstract Title: The related Ets transcription factors Spi-B and Spi-C exert opposing roles in regulating terminal B cell differentiation
Supervisor(s): R. DeKoter

Poster Number: 77
Name: M. Erin Moir
Degree: PhD Candidate
Abstract Title: Cerebrovascular compliance is affected by posture
Supervisor(s): K. Shoemaker

Poster Number: 78
Name: Klajdi Puka
Degree: MSc Candidate
Abstract Title: Trajectories of depressive symptoms among mothers of children with newly diagnosed epilepsy: A longitudinal ten-year study
Supervisor(s): K. Speechley

Poster Number: 79
Name: Richard Sové
Degree: PhD Candidate
Abstract Title: Novel method for determining incident light intensity for the calculation of red blood cell oxygen saturation from in-vivo microscopy image
Supervisor(s): G. Fraser, C. Ellis
EARLY LIFE PROGRAMMING AND DEVELOPMENT

Poster Number: 80
Name: William Cho
Degree: MSc Candidate
Abstract Title: Role of PU.1 and C/EBPa in remodelling the Interleukin (IL)-1b enhancer-promoter interactions
Supervisor(s): S. Kim

Poster Number: 81
Name: Joshua Dierolf
Degree: PhD Candidate
Abstract Title: Delineating the metabolic role of pyruvate kinase muscle isoforms in mouse embryonic stem cell pluripotency
Supervisor(s): D. Betts

Poster Number: 82
Name: Zachary Easton
Degree: MSc Candidate
Abstract Title: The metabolic response of human villous trophoblasts to prolonged fatty acid exposure
Supervisor(s): T. Regnault

Poster Number: 83
Name: Leah Groves
Degree: MSc Candidate
Abstract Title: Guided ultrasound calibration: A method for the common user
Supervisor(s): T. Peters

Poster Number: 84
Name: Sommer Jarvis
Degree: MSc Candidate
Abstract Title: Examining the correlation between cell behaviour and morphology in the developing skull of Cx43I130T/+ mice
Supervisor(s): K. Willmore, G. Kelly

Poster Number: 85
Name: Alexandra Kozlov
Degree: PhD Candidate
Abstract Title: Sole fuel source selection strategy to enhance pluripotency
Supervisor(s): R. Cumming, D. Betts

Poster Number: 86
Name: Pinki Nandi
Degree: Postdoctoral Scholar
Abstract Title: Human trophoblast stem cell self-renewal and differentiation: Role of decorin
Supervisor(s): P. Lala

Poster Number: 87
Name: Bethany Radford
Degree: PhD Candidate
Abstract Title: A complicated relationship between fetal undernutrition, post-natal diet and adult metabolism
Supervisor(s): V. Han

MECHANISMS OF DISEASE

Poster Number: 88
Name: Bibek Saha
Degree: Research Assistant/Associate
Abstract Title: Maternal nutrient restriction causes neuroinflammation and upregulates amyloid related proteins in guinea pig offspring born growth restricted
Supervisor(s): B. Richardson, D. Hill

Poster Number: 89
Name: Alex Szpak
Degree: MSc Candidate
Abstract Title: Mechanisms which regulate the dorsal-ventral patterning of the developing pharynx in Xenopus laevis
Supervisor(s): T. Drysdale

Poster Number: 90
Name: Julia Abitbol
Degree: PhD Candidate
Abstract Title: Mice expressing an oculodentodigital dysplasia-linked Cx43 G60S mutant have hearing loss
Supervisor(s): D. Laird

Poster Number: 91
Name: Rasha Alsubaie
Degree: Resident
Abstract Title: Characterizing post-stroke autonomic functioning, Sub-study protocol of the clinical arm of PARADISE study
Supervisor(s): K. Kimpinsk, L. Sposato

Poster Number: 92
Name: Farzad Asadi
Degree: PhD Candidate
Abstract Title: Heterogenous glucagon-immunoreactive peptides as novel regulators of insulin secretion
Supervisor(s): S. Dhanvantari

Poster Number: 93
Name: Matthew Berg
Degree: PhD Candidate
Abstract Title: Isolating mistranslating serine tRNAs with a proline anticodon through an ambivalent intermediate
Supervisor(s): C. Brandl

Poster Number: 94
Name: Matthew Bernardinis
Degree: MSc Candidate
Abstract Title: Visual Displacement perception in Parkinson’s disease using a virtual reality toolbox
Supervisor(s): R. Patel, M. Jog

Poster Number: 95
Name: Saumik Biswas
Degree: PhD Candidate
Abstract Title: MALAT1 and HOTAIR: Key epigenetic regulators in diabetic retinopathy
Supervisor(s): S. Chakrabarti
Poster Number: 96  
Name: Adrian Buensuceso  
Degree: Postdoctoral Scholar  
Abstract Title: The metabolic stress mediator LKB1 is required for ovarian cancer metastasis  
Supervisor(s): T. Shepherd

Poster Number: 97  
Name: Diego Buitrago-Piza  
Degree: MSc Candidate  
Abstract Title: Perception of gaze direction using 3D virtual reality displays: Effect of sclera and head orientation  
Supervisor(s): J. Martinez-Trujillo

Poster Number: 98  
Name: Ting Cao  
Degree: PhD Candidate  
Abstract Title: Up-regulation of mitochondria-targeted calpain-1 induces dilated heart failure in transgenic mice: An important role of mitochondrial reactive oxygen species  
Supervisor(s): T. Peng

Poster Number: 99  
Name: Jacqueline Chevalier  
Degree: MSc Candidate  
Abstract Title: Microvascular architecture and phenotypes in patients with severe peripheral artery disease  
Supervisor(s): J. Pickering

Poster Number: 100  
Name: Steve Chung  
Degree: MSc Candidate  
Abstract Title: Effects of stromal interaction molecule coiled-coil variations on the mechanisms of store operated Ca2+ entry regulation  
Supervisor(s): P. Stathopulos

Poster Number: 101  
Name: Melissa Crawford  
Degree: PhD Candidate  
Abstract Title: Role of integrin-linked kinase in melanocyte development  
Supervisor(s): L. Dagnino

Poster Number: 102  
Name: Brennan Dirk  
Degree: PhD Candidate  
Abstract Title: Seeing is believing: Unraveling intermolecular complexes mediating HIV-1 immune evasion  
Supervisor(s): J. Dikeakos

Poster Number: 103  
Name: Mohammad Esmaeili  
Degree: Postdoctoral Scholar  
Abstract Title: Two faces of Parkin oxidation and misfolding: Gain- and loss-of-function in Parkinson’s disease  
Supervisor(s): M. Duennwald

Poster Number: 104  
Name: Lana Estafanos  
Degree: MSc Candidate  
Abstract Title: The Group A Streptococci bacteriocin SpbMN facilitates a competitive advantage through direct antimicrobial action  
Supervisor(s): J. McCormick

Poster Number: 105  
Name: Jay Fang  
Degree: MSc Candidate  
Abstract Title: Expression of hepatic cytochrome P450 drug-metabolizing enzymes in diabetes and diabetic nephropathy  
Supervisor(s): B. Urquhart

Poster Number: 106  
Name: Melissa Fenech  
Degree: PhD Candidate  
Abstract Title: Pancreas-specific secretory pathway calcium ATPase 2 affects store operated calcium entry  
Supervisor(s): C. Pin

Poster Number: 107  
Name: Niveen Fulcher  
Degree: PhD Candidate  
Abstract Title: NUAk1 Expression is regulated to alter cell growth, viability and adhesion in ovarian cancer spheroids  
Supervisor(s): T. Shepherd, G. DiMattia

Poster Number: 108  
Name: Matthew Hintermayer  
Degree: MSc Candidate  
Abstract Title: Tau protein pathology following traumatic brain injury: A longitudinal investigation  
Supervisor(s): M. Strong

Poster Number: 109  
Name: Rosetta Ho  
Degree: MSc Candidate  
Abstract Title: Identification of a novel IHH insertion causative of brachydactyly type A1  
Supervisor(s): R. Hegele

Poster Number: 110  
Name: Aja Hogan-Cann  
Degree: PhD Candidate  
Abstract Title: Regulation of microglia-derived neuroinflammation in Alzheimer’s disease  
Supervisor(s): M. Prado, V. Prado
Poster Number: 112
Name: Nadun Karunatilleke
Degree: PhD Candidate
Abstract Title: Nrf2, the key transcription factor that regulates the cellular response to oxidative stress, is intrinsically disordered
Supervisor(s): W. Choy

Poster Number: 113
Name: Yae Ram Kim
Degree: Research Assistant/Associate
Abstract Title: Investigation of chromosome 19 miRNA cluster microRNA (miR-512-3p) in human trophoblast migration
Supervisor(s): P. Nandi, P. Lala

Poster Number: 114
Name: Ornela Kljakic
Degree: PhD Candidate
Abstract Title: Developing novel mouse lines to investigate the roles of TAN-secreted ACh and Glu
Supervisor(s): V. Prado, M. Prado

Poster Number: 115
Name: Jason Knapp
Degree: MSc Candidate
Abstract Title: Evaluating the role of ApsS in promoting Staphylococcus aureus adaptation to acidic pH and antimicrobial unsaturated free fatty acids
Supervisor(s): M. McGavin

Poster Number: 116
Name: Jeremy Lant
Degree: PhD Candidate
Abstract Title: tRNA-dependent, codon re-coding adds a ‘third dimension’ to the genetic code in Escherichia coli and mammalian cells
Supervisor(s): P. O’Donoghue

Poster Number: 117
Name: Matthew Novello
Degree: MSc Candidate
Abstract Title: Developing novel mouse lines to investigate the roles of TAN-secreted ACh and Glu
Supervisor(s): V. Prado, M. Prado

Poster Number: 120
Name: Alexander Levit
Degree: PhD Candidate
Abstract Title: Age-dependent white matter inflammation and cognitive impairment in the TgAPP21 rat model of Alzheimer disease
Supervisor(s): V. Hachinski, S. Whitehead

Poster Number: 121
Name: Johnny Luo
Degree: MSc Candidate
Abstract Title: Role of glycosylation on the HIV transmitted/founder: Encountering the lectin trap in the recipient mucosa
Supervisor(s): E. Arts, C. Creuzenet

Poster Number: 122
Name: Vy Ngo
Degree: PhD Candidate
Abstract Title: Deletion of CHOP prevents necroptosis in diabetic cardiomyopathy
Supervisor(s): T. Peng

Poster Number: 123
Name: Kevin Nixon
Degree: PhD Candidate
Abstract Title: Maintenance of cell-type specific gene expression patterns in memory forming neurons in a Drosophila model for intellectual disability
Supervisor(s): J. Kramer, M. Daley

Poster Number: 124
Name: Daniel Nouri Nejad
Degree: MSc Candidate
Abstract Title: Characterization of PANX1 mutations from patient-derived melanoma tumours
Supervisor(s): S. Penuela

Poster Number: 125
Name: Matthew Novello
Degree: MSc Candidate
Abstract Title: S-nitrosylation suppresses stromal interaction molecule-2 activation and stability which exerts an effect on cardiovascular physiology
Supervisor(s): P. Stathopulos, Q. Feng
Poster Number: 128
Name: Ranjit Singh Padda
Degree: PhD Candidate
Abstract Title: Polysialic acid synthesis by polysialyltransferase ST8SiaIV in prostate cancer: Using glycans as a novel anti-cancer drug target
Supervisor(s): Z. Khan, C. Chakraborty

Poster Number: 129
Name: Katie Parkins
Degree: PhD Candidate
Abstract Title: Dissecting the impact of a primary tumour on metastatic outgrowth with multimodality molecular imaging
Supervisor(s): P. Foster, J. Ronald

Poster Number: 130
Name: Emily Pawlak
Degree: PhD Candidate
Abstract Title: The immune cell surface receptor CD28 is hijacked by the HIV-1 accessory proteins Nef and Vpu
Supervisor(s): J. Dikeakos

Poster Number: 131
Name: Eric Press
Degree: Research Assistant/Associate
Abstract Title: Induction of cell death and gain-of-function properties of connexin26 mutants predict severity of skin disease and hearing loss
Supervisor(s): D. Laird

Poster Number: 132
Name: Amanda Rampersaud
Degree: MSc Candidate
Abstract Title: The effect of high sugar and fatty acids on rat trophoblast stem cell differentiation
Supervisor(s): S. Renaud

Poster Number: 133
Name: Abdul Razzaq
Degree: MSc Candidate
Abstract Title: HSP90 and STI1 Involvement in TDP-43 toxicity and pathology in neurodegenerative disorders
Supervisor(s): F. Beraldo, M. Prado, V. Prado

Poster Number: 134
Name: Noor Salloum
Degree: PhD Candidate
Abstract Title: Identifying host gastric proteins interacting with the Helicobacter pylori virulence factor HcpE
Supervisor(s): C. Creuzenet

Poster Number: 135
Name: Rafael Sanchez
Degree: PhD Candidate
Abstract Title: Effect of N-glycosylation on Pannexin 2 subcellular localization and its interaction with Pannexin1
Supervisor(s): S. Penuela

Poster Number: 136
Name: Jenna Schulz
Degree: PhD Candidate
Abstract Title: Sodium nitroglycerin induces cerebrovascular vasodilation in patients with ischemic heart disease
Supervisor(s): T. Birmingham, F. Beier

Poster Number: 137
Name: Kaela Scott
Degree: PhD Candidate
Abstract Title: The role of the CNTNAP2 gene in auditory temporal processing deficits
Supervisor(s): B. Allman, S. Schmid

Poster Number: 138
Name: Blake Shannon
Degree: Research Assistant/Associate
Abstract Title: Role of streptolysin O and streptolysin S in Streptococcus pyogenes nasopharyngeal colonization
Supervisor(s): J. McCormick

Poster Number: 139
Name: Ali Sherazi
Degree: Research Assistant/Associate
Abstract Title: O-GlcNAcylation plays a role in galectin expression regulation in human cell lines
Supervisor(s): A. Timoshenko

Poster Number: 140
Name: Jessica Snyder
Degree: MSc Candidate
Abstract Title: Correlating white matter changes to executive dysfunction in a rat model of mediodorsal thalamic stroke
Supervisor(s): S. Whitehead, B. Allman

Poster Number: 141
Name: Zheng Song
Degree: MSc Candidate
Abstract Title: Deciphering the binding mechanism of Hsp90 with Keap1 and Nrf2
Supervisor(s): W. Choy

Poster Number: 142
Name: Anette Surmanski
Degree: MSc Candidate
Abstract Title: Identifying the role of G-Protein Signalling Modulator 3 in GPCR signalling
Supervisor(s): P. Chidiac, B. Urquhart

Poster Number: 143
Name: Michael Tauro
Degree: MSc Candidate
Abstract Title: Alpha-Synuclein interferes with mitochondrial homeostasis
Supervisor(s): M. Duennwald

Poster Number: 144
Name: Vasiliki Tellios
Degree: PhD Candidate
Abstract Title: nNOS in the regulation of calcium homeostasis of the PF-PN synapse in mice cerebella
Supervisor(s): W. Lu

Poster Number: 145
Name: Mathura Thiagarajah
Degree: Research Assistant/Associate
Abstract Title: Prevalence of autoimmune disorders in frontotemporal dementia and related disorders
Supervisor(s): E. Finger
Poster Number: 146  
Name: An Tran  
Degree: MSc Candidate  
Abstract Title: Structural consequences of oxidative stress on parkin  
Supervisor(s): G. Shaw

Poster Number: 147  
Name: Meerah Vijeyakumaran  
Degree: MSc Candidate  
Abstract Title: Effect of estrogen and glucocorticoid signaling on Th2 cells: Implications in severe asthma  
Supervisor(s): L. Cameron

Poster Number: 148  
Name: Yuning Wang  
Degree: PhD Candidate  
Abstract Title: Mechanisms of calcium signaling in dysferlin-mediated membrane repair  
Supervisor(s): G. Shaw

Poster Number: 149  
Name: David Wright  
Degree: PhD Candidate  
Abstract Title: Acetylation regulates thioredoxin reductase oligomerization and activity  
Supervisor(s): P. O’Donoghue

Poster Number: 150  
Name: Spencer Yeung  
Degree: MSc Candidate  
Abstract Title: Determining the relative transmission fitness of HIV-1 Subtypes in cervical and foreskin tissue  
Supervisor(s): E. Arts, J. Prodger

Poster Number: 151  
Name: Katrina Zmavc  
Degree: MSc Candidate  
Abstract Title: Neurogenesis in the adult hippocampus and its role in mood  
Supervisor(s): T. Bussey, L. Saksida

POPULATIONS, PUBLIC HEALTH AND EDUCATION

Poster Number: 152  
Name: Jessica Blom  
Degree: PhD Candidate  
Abstract Title: Advancing the understanding of research and science in medical education through collaborative learning: Collaboration of practitioners and researchers seminar series  
Supervisor(s): D. Jones

Poster Number: 153  
Name: Jordan Edwards  
Degree: PhD Candidate  
Abstract Title: The burden of mood and anxiety disorders among immigrant and refugee populations in Canada: A systematic review  
Supervisor(s): K. Anderson, A. Thind

Poster Number: 154  
Name: Ian Janes  
Degree: MSc Candidate  
Abstract Title: Investigating falls knowledge and falls prevention among total hip arthroplasty patients: A cross-sectional study  
Supervisor(s): S. Hunter

Poster Number: 155  
Name: Erin Kennedy  
Degree: PhD Candidate  
Abstract Title: Spoilt for choice: A discerning view of review types in the health sciences  
Supervisor(s): S. Cristancho

Poster Number: 156  
Name: Rohin Krishnan  
Degree: Research Assistant/Associate  
Abstract Title: Sutures versus staples for skin closure after orthopedic surgery: A meta-analysis of randomized controlled trials.  
Supervisor(s): J. Martin

Poster Number: 157  
Name: Evan Michaelov  
Degree: Medical Student  
Abstract Title: Medical students’ opinion on ophthalmology as a career: A survey of students at a Canadian medical school  
Supervisor(s): S. Sharan

Poster Number: 158  
Name: Patrick Murphy  
Degree: Resident  
Abstract Title: Impact of the surgeon on the management and outcomes of acute appendicitis: A population based analysis  
Supervisor(s): C. Vinden and K. Vogt

Poster Number: 159  
Name: Peyton Schroeder  
Degree: MSc Candidate  
Abstract Title: Real world motor vehicle collision head injury risk and car seat use for children under 8 years old  
Supervisor(s): M. Shkrum

Poster Number: 160  
Name: Chloe Stewart  
Degree: PhD Candidate  
Abstract Title: The psychophysiology of guilt  
Supervisor(s): E. Finger

Poster Number: 161  
Name: Sinthiya Thavam  
Degree: MSc Candidate  
Abstract Title: Threshold interaural time differences under optimal conditions  
Supervisor(s): M. Dietz
PREVENTION OF DISEASES AND HEALTH CONDITIONS AND PROMOTION OF WELL-BEING

Poster Number: 162
Name: Faizah Alotaibi
Degree: PhD Candidate
Abstract Title: Targeting CD5 and Fas receptor on CD8+ T cells to improve anti-tumour immunity
Supervisor(s): J. Koropatnick, W. Min

Poster Number: 163
Name: Sarah Best
Degree: MSc Candidate
Abstract Title: Jazzercise as an intervention for subjective cognitive decline in postmenopausal women: Pilot study rationale and methodology
Supervisor(s): M. Borrie

Poster Number: 164
Name: Ryan Cochrane
Degree: MSc Candidate
Abstract Title: The development of synthetic organelle genomes for commercial and scientific use
Supervisor(s): B. Karas, D. Edgell

Poster Number: 165
Name: Rebecca Fried
Degree: PhD Candidate
Abstract Title: Making the Grade: Building mental health resiliency through mentorship and physical activity – A course-based, smart, healthy campus project: Mentors’ perspectives
Supervisor(s): J. Irwin

Poster Number: 166
Name: Michael Greff
Degree: Postdoctoral Scholar
Abstract Title: Hair cortisol concentrations in war-affected adolescents: A prospective intervention trial
Supervisor(s): M. Rieder, S. van Uum

Poster Number: 167
Name: Nicole Guitar
Degree: PhD Candidate
Abstract Title: The effects of physical exercise on executive function in community-dwelling older adults living with Alzheimer’s-type dementia: A systematic review of randomized controlled trials
Supervisor(s): D. Connelly

Poster Number: 168
Name: Sara Holland
Degree: MSc Candidate
Abstract Title: The comparison of golf grips to hand forces in individuals with and without hand arthritis
Supervisor(s): E. Lalone, L. Ferreira

Poster Number: 169
Name: Becky Horst
Degree: MSc Candidate
Abstract Title: Understanding the relationship between memory self-efficacy, cognition and brain health in older adult females with probable mild cognitive impairment: A pilot study
Supervisor(s): L. Nagamatsu

Poster Number: 170
Name: Sahana Kukan
Degree: MSc Candidate
Abstract Title: Examining the relationship between internal drivers of motivation and functional outcomes in a cross-section of individuals with psychotic disorders
Supervisor(s): A. MacDougall, K. Anderson

Poster Number: 171
Name: Wai-Dun Lee
Degree: MSc Candidate
Abstract Title: Nanosizing of ginseng polysaccharides enhanced cellular activity in vitro
Supervisor(s): E. Lui

Poster Number: 172
Name: Shirin Modarresi
Degree: PhD Candidate
Abstract Title: A protocol for investigating the effects of auditory stimulation on gait in people with Alzheimer’s disease
Supervisor(s): S. Hunter

Poster Number: 173
Name: Nadya Morrow
Degree: MSc Candidate
Abstract Title: Nobiletin corrects intestinal lipid metabolism in Ldlr-/- Mice fed a high-fat diet
Supervisor(s): M. Huff

Poster Number: 174
Name: Elena Sheldrake
Degree: MSc Candidate
Abstract Title: The maintenance and adaptation of identity in older adults aging with Parkinson’s disease
Supervisor(s): C. McGrath

Poster Number: 175
Name: Robert Szabla
Degree: PhD Candidate
Abstract Title: The role of PprA in extreme DNA damage resistance of Deinococcus
Supervisor(s): M. Junop
**LONDON HEALTH RESEARCH DAY 2018**
DRIVING INNOVATION THROUGH COLLABORATION

**FEATURE PLATFORM PRESENTATIONS**
**AFTERNOON SESSION**
3:30 - 4:45 p.m.
London Convention Centre - First Floor

*TIMES ARE APPROXIMATE*

<table>
<thead>
<tr>
<th>TIME</th>
<th>SALON B</th>
<th>SALON B1</th>
<th>SALON D</th>
<th>SALON E</th>
</tr>
</thead>
<tbody>
<tr>
<td>3:30 - 3:45 P.M.</td>
<td>Simon Benoit</td>
<td>Patricia Johnson</td>
<td>Jennifer Chen</td>
<td>Salma Dammak</td>
</tr>
<tr>
<td>3:45 - 4:00 P.M.</td>
<td>Radu Gugustea</td>
<td>Amanda McIntyre</td>
<td>Allison Dilliot</td>
<td>Jacqueline Dron</td>
</tr>
<tr>
<td>4:00 - 4:15 P.M.</td>
<td>Samantha McRae</td>
<td>Patrick Rudak</td>
<td>Mohammad Haddara</td>
<td>Rajiv Jain</td>
</tr>
<tr>
<td>4:15 - 4:30 P.M.</td>
<td>Marwan Shahid</td>
<td>Michelle Shum</td>
<td>Andrew Nicholson</td>
<td>Daniel Milej</td>
</tr>
<tr>
<td>4:30 - 4:45 P.M.</td>
<td>Charles Yin</td>
<td>Nibene Somé</td>
<td>Jessica Rodgers</td>
<td>Fatemeh Mirshafiei</td>
</tr>
</tbody>
</table>
TRANSCRIPTOME-LEVEL GENETIC CHANGES IN THE BRAIN OF LIVING PARKINSON’S DISEASE PATIENTS: EVIDENCE FOR DYSREGULATION OF INFLAMMATORY PROCESSES INVOLVING THE NF-κB COMPLEX

SIMON BENOIT
PhD Candidate

Abstract:
Introduction: The development of next-generation sequencing platforms, particularly RNAseq, has made the possibility of sequencing the transcriptome in biological samples feasible at a fraction of the time and cost. For diseases like Parkinson’s disease (PD), that show widespread pathological changes in the central nervous system (CNS) including mitochondrial dysfunction; activation of immune mechanisms; defective protein processing; and decreased levels of neurotrophic factors; this opens up the possibility of capturing ongoing neurodegenerative processes at the transcriptional level. To date, examinations of the PD transcriptome have mostly been limited to non-CNS or cadaveric sources of tissue, where investigation is likely to be at best a proxy of the ongoing disease process in the living brain. Small volume brain biopsies that can be safely obtained during deep-brain stimulation surgery for PD offer a feasible and novel alternative to these more traditional biological specimens.

Hypothesis: Whole-transcriptome RNA sequencing of total RNA in cortical samples taken from living PD patients will show gene expression alterations compared to controls.

Materials and Methods: Total RNA was extracted from cortical biopsies in 6 patients with PD and 6 controls, then sequenced on the Illumina HiSeq 2500 platform using a stranded paired-end protocol. Reads, totaling approximately 90 million per sample, were trimmed to remove adapters and low quality bases, then aligned to the human genome (Hg19) using TopHat (v.1.8). Raw counts were generated using HTSeq (v.0.6.1p2); and analyzed for differential expression using edgeR (v.3.8.6). Pathway enrichment and induced network analyses were performed using the freely available analysis tools from the Gene Ontology Consortium (http://www.geneontology.org/), ConsensusPathDB (http://cpdb.molgen.mpg.de) and Cytoscape (v.3.6.0) for network visualization.

Results: At a false discovery rate threshold of <0.05, 763 differentially expressed genes were identified, with 347 upregulated and 416 downregulated in PD. Using only significant genes with a fold change greater than 1.5, pathway analysis showed enrichment of genes responsible for regulating inflammatory response processes (p= 3.22E-07); tumor necrosis factor production and cellular response (p= 1.58E-04 and 5.21E-04 respectively); the innate immune response (p= 5.97E-04) and negative regulation of apoptosis (p= 3.06E-04). Well-known disease relevant genes involved in these biological processes include CD44, Interleukin-10, GDNF, IL1β, Transcription factor AP-2-delta, and NF-kappa-B inhibitor alpha. Interestingly, induced network analysis revealed a robust network surrounding the NF-κB complex.

Discussion and Conclusions: To our knowledge this is the first demonstration of differential CNS gene expression and enrichment of numerous biological processes in cortical samples from living PD patients. Alterations in gene expression in ~4% of detected genes offer a wealth of data with potential to identify genetic biomarkers that may facilitate diagnosis and treatment for PD. Furthermore, significant results from pathway analysis and induced networks point to dysregulation of processes surrounding the NF-κB complex, which is a key regulator of the immune response previously implicated in inflammatory diseases. This lends further evidence to the growing body of literature relating neuroinflammation to PD and other neurodegenerative diseases.
MOTION CORRECTION IN MRI USING DEEP LEARNING

PATRICIA JOHNSON
PhD Candidate

Research Areas:
Medical biophysics, engineering and imaging
Detection, screening and diagnosis of health and disease

Supervisor(s):
M. Drangova

Advisory Committee:
T. Peters, C. McKenzie, J. Liu

Abstract:
Introduction: Subject motion in MRI remains an unsolved problem; motion during image acquisition may cause artefacts that severely degrade image quality. In the clinic, if an image with motion artefacts is acquired, it will often be reacquired. This provides a source from which a large number of motion-degraded images, along with their respective re-scans, could be collected. These pairs of images could be used to train a neural network to identify the mapping relationship between an image with motion artefacts and a high quality, artefact free image. Inspired by previous work demonstrating MR image reconstruction with machine learning,[1,2] our objective is to train a neural network to perform motion corrected image reconstruction on image data with simulated motion artefacts. We simulate motion in previously acquired brain images and use the image pairs (corrupted + original) to train a deep neural network (DNN).

Hypothesis: We hypothesized that a deep neural network could be trained to perform a motion corrected MR image reconstruction given the motion corrupted data in fourier space (k-space).

Materials and Methods: Data were obtained from an open source neuro MRI data set [3] comprising T2* weighted, magnitude and phase images for 53 patients, each with 128 non-overlapping image slices; the data set thereby provides thousands of unique 2D complex-valued images. Each 2D image from this data set was Fourier transformed to simulate the acquired k-space data. To simulate rigid motion, k-space lines were rotated and phase shifted, simulating the k-space inconsistencies that would occur if the subject were moving their head. The motion profiles were parameterized by the time, magnitude and direction of motion and were randomly generated with constraints to keep the motion within the realm of realistic head motion. A unique 3D motion profile was applied to each image. The DNN was developed and trained using the TensorFlow library [4]. The network consists of a densely connected layer followed by 4 convolutional layers. The input to the network has 5 channels; each channel contains the data from one k-space slice. The network training set consisted of 2,048 image pairs; 64 pairs were reserved for validation and testing. The network was trained for 4 hrs using the SHARCNET computing network.

Results: The images predicted by the DNN, from motion-corrupted k-space, have improved image quality compared to the motion-corrupted images. The mean absolute error (MAE) between the motion corrupted and ground-truth images was 32% of the image mean value, while the (MAE) between the DNN-predicted and ground-truth images was only 11%.

Discussion and Conclusions: Motion-corrected image reconstruction was successfully achieved on brain images with simulated motion artefacts. This work represents the first time machine learning has been used to perform motion correction of MR images. Improving the consistency of the networks performance is the focus of ongoing work.

Salon D

THE EFFECT OF INCREASING PASSIVE CYCLING INTENSITY ON GLOBAL HEMODYNAMICS, BRAIN AND HEART PERFUSION IN SEPTIC PATIENTS

JENNIFER CHEN
MSc Candidate

Research Areas:
Circulatory
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

Supervisor(s):
C. McIntyre

Advisory Committee:
C. McIntyre, M. Slessarev

Abstract:
Introduction: Sepsis is a global problem that is associated with high mortality and immense healthcare costs (Angus et al, 2006). Despite its fatality, and the myriad of long term complications that effect sepsis survivors (Shankar-Hari et al, 2016), early therapies to improve organ perfusion are lacking. Exercise, particularly early mobilization, is associated with shorter hospital stay and better functional outcomes in septic patients (Cameron et al, 2015). However, active exercise is often not feasible in the early phase of sepsis. Passive exercise, such as in-bed cycling, can be used as an alternative to active exercise to potentially improve organ perfusion in the acute phase of sepsis. However, its optimal intensity in septic patients is unknown. In this study, we assessed the effect of increasing the intensity of passive cycling on global hemodynamics, brain perfusion and cardiac function in a cohort of septic patients.

Hypothesis: 1) Passive exercise will not worsen global hemodynamics, brain perfusion and cardiac function in septic patients. 2) The optimal intensity of passive exercise will vary between patients.

Materials and Methods: We used Finapres® NOVA, transcranial Doppler, and speckle-tracking echocardiography to measure global hemodynamics, cerebral blood flow and cardiac function respectively during incremental increase in passive cycling intensity in septic patients. The protocol started and concluded with 5 minutes of baseline measurements at rest, followed by progressive increase in passive cycling intensity from 5 to 55 rotations per minute (RPM) in 10 RPM increments, with each experimental stage lasting 5 minutes. Mean values were calculated for all measured parameters during the last 2 minutes of each experimental stage and ANOVA was used to determine difference between experimental stages both within and between patients.

Results: Ten septic patients completed the protocol. Increase in the intensity of passive cycling had no effect on global hemodynamics or cardiac function, but resulted in dose-dependent decrease in cerebral blood flow (CBF), with maximal reduction of 5.7% below resting values at peak exercise intensity (55 RPM), followed by incomplete recovery to 2.4% during ensuing 5 minutes of recovery (0 RPM). This reduction in CBF was not uniform among patients, with two patients showing no changes, one patient showing an increase, and three patients showing biphasic responses in CBF, with increasing exercise intensity.

Discussion and Conclusions: In contrast to our findings in healthy volunteers (Chen et al, 2017), increasing intensity of passive cycling resulted in a dose-dependent reduction in CBF in our cohort of septic patients. This reduction in CBF occurred on the background of stable global hemodynamics and global cardiac function, and varied between patients, with some showing no change or even mild increase in CBF. Our results are concerning with respect to safety of passive cycling in septic patients, as observed reduction in CBF may induce cerebral ischemia, potentially worsening patient outcomes. The observed heterogeneity of CBF responses between patients highlights the need to consider individualized prescription of passive exercise dose in different patients and warrants further research in this area.
EARLY DETECTION OF LUNG CANCER RECURRENCE AFTER STEREOTACTIC ABLATIVE RADIATION THERAPY: RADIOMICS SYSTEM DESIGN

SALMA DAMMAK
MSc Candidate

Research Areas:
Medical biophysics, engineering and imaging
Detection, screening and diagnosis of health and disease

Supervisor(s):
A. Ward, D. Palma

Advisory Committee:
A. Ward, D. Palma, K. Kwan, D. Hoover

Abstract:
Introduction: Stereotactic ablative radiotherapy is a guideline-recommended treatment option for patients with Stage I non-small cell lung cancer who are inoperable or refuse surgery [1]. This treatment, however, has a high likelihood of introducing a benign radiation-induced lung injury (RILI) that can be difficult to differentiate from disease recurrence on computed tomography (CT) scans, which are used for treatment follow up in the regular clinical workflow. Radiomics is a method of quantifying the visual descriptors of an image called features. In a preliminary study with a small sample (n=45), we have shown that radiomics coupled with machine learning shows promise for differentiating RILI from recurrence early after treatment [2]. The system produced an area under the receiver operating characteristic curve (AUC) of 0.85 [2].

Hypothesis: We hypothesize that we can differentiate RILI from recurrence within 2–5 month after treatment with an AUC > 0.8 using a radiomics and machine learning system for a sample size of 81 patients.

Materials and Methods: This study was approved by our institutional human subjects’ research ethics board. We obtained contrast enhanced CT images from 81 patients at 2-5 months after treatment (2 RILI : 1 recurrence). We extracted 141 features from two regions of interest (ROIs) in those images: the solid region where the tumour was diagnosed before treatment, which is traditionally analyzed, and the area surrounding it, both of which we obtained using our published segmentation algorithm [3]. We reduced the number of features to 10 features using four criteria for minimization of error, obtaining 4 feature sets, and for each set we used 7 classifiers to classify our data into recurrence and RILI using 1 to 10 features using cross-validation with two different training set sizes. We found the best performing classifier and feature set system using area under the curve (AUC) error metric. We determined the most robust combination by finding the set of features with least variation in AUC across all classifiers, and chose the classifier with the best error metrics for that set.

Results: The best performing system had an AUC of 0.92 and used 10 features selected by the inter-intra distance (in-in) criterion, classified by the combined Adaboost and nu-type support vector machine (nuSVC) classifier. The most robust system had an AUC of 0.80 and used 5 features also selected by the in-in criterion, and classified with the nuSVC classifier. When comparing the performance of each of these systems using different size training sets, we found that the more robust one has less variability in error metrics with a changing sample size, suggesting that it is likely to be generalizable. Both systems used features from both ROIs to arrive at these results.

Discussion and Conclusions: Analysis of 81 patients confirmed that an approach of radiomics combined with machine learning provides a promising solution to differentiating RILI from recurrence within the first 2-5 months after treatment. Additionally, exploring other machine learning parameters can allow for the design of a system with less error and more robustness. Both systems relied on features extracted from both ROIs, suggesting that both are important for solving this problem.

3:45 - 4:00 P.M. PRESENTATIONS

Salon B

EFFECT OF ATRX INACTIVATION ON HIPPOCAMPAL SYNAPTIC PLASTICITY IN MICE

RADU GUGUSTEA
MSc Candidate

Research Areas:
Neuroscience
Mechanisms of disease

Supervisor(s):
L. Leung, N. Bérubé

Advisory Committee:
A. Brown, L. Leung, N. Bérubé, W. Inoue

Abstract:

Introduction: The hippocampus is a brain structure involved in learning and memory. Normal hippocampal development depends on the expression of chromatin-remodeling proteins, such as ATRX. ATRX mutations result in alpha thalassemia X-linked mental retardation (ATR-X) syndrome, a severe cognitive disorder which leads to impairments in learning and memory. Male mice with conditional ablation of the Atrx gene (ATRX- mice) specifically in postnatal forebrain pyramidal neurons were impaired in hippocampus-dependent memory tasks such as the Morris water maze and contextual fear conditioning (Tamming NRJ, unpublished). Long-term potentiation (LTP), or a long-lasting enhancement of synaptic transmission induced by tetanic stimulation, has been proposed as a mechanism of memory. We propose to study whether synaptic plasticity in the hippocampus in vivo is altered in ATRX-deficient mice as compared to wildtype (WT) mice. In vivo LTP may provide a physiologically relevant measure of synaptic plasticity that may correlate with behavioural results.

Hypothesis: Conditional ablation of the Atrx gene in postnatal forebrain pyramidal neurons disrupts hippocampal synaptic plasticity.

Materials and Methods: ATRX- mice were generated by conditional deletion of a floxed Atrx allele at postnatal day 20 in cortical and hippocampal pyramidal neurons using the CaMKII promoter to drive Cre recombinase expression. Adult ATRX- and WT mice (age 16-20 weeks), identified by PCR genotyping of tail biopsies, were anesthetized with urethane (1.25 mg/kg i.p.). A 16-channel recording probe was inserted into the CA1 region to record extracellular evoked potentials. A stimulating electrode was inserted into the CA3 region to evoke basal dendritic population excitatory postsynaptic potentials (pEPSPs) in CA1, through the Schaffer collaterals. Basal dendritic, rather than apical dendritic, LTP was attempted because it is larger and more readily induced in vivo. Responses evoked by the medial perforant path (MPP) served as a control pathway. After a stable baseline of 30 min, a high-frequency theta-burst stimulation (TBS; 10 trains at 10 s intervals, 10 bursts at 0.2 s intervals per train, 10 pulses at 100 Hz per burst) was given, and recordings continued for two hours post-TBS. Mouse brains were extracted after intracardial perfusion and used to verify electrode placement. Average evoked field potentials were analyzed by current source density analysis. The excitatory sink at the basal dendrites following CA3 stimulation, and that in the dentate gyrus (DG) molecular layer following MPP stimulation, were estimated and normalized by the respective baseline measure. Statistical analysis between the two groups used unpaired t-test or two-way repeated measures ANOVA.

Results: Preliminary data showed that basal dendritic LTP was induced in both ATRX- and WT mice, averaging ~160-200% of the baseline at 30-120 min post-TBS. Two-way repeated measures ANOVA of LTP showed no significant group or group x time effects. However, short-term potentiation (STP) at 15 min post-TBS showed a trend (0.1>P>0.05, t-test) to be higher in ATRX- mice (233 ± 11%, n=2) than WT mice (200 ± 5%, n=5). The non-tetanized MPP pathway to the DG did not show a significant change within the two groups.

Discussion and Conclusions: ATRX- mice may show higher STP and LTP than WT mice, which is contrary to our hypothesis. Future studies will increase sample size and assess LTP of other hippocampal pathways.
A COMPARISON OF HOME-BASED AND HOSPITAL-BASED OUTPATIENT REHABILITATION SERVICES IN ONTARIO, CANADA

AMANDA MCINTYRE
PhD Candidate

Research Areas:
Neuroscience
Advancing health services provision and health policy

Supervisor(s):
M. Kerr, R. Booth

Advisory Committee:
N/A

Abstract:

Introduction: Numerous studies have been conducted comparing hospital and home-based outpatient programs and the results remain inconclusive; however, little research has examined what occurs in a similar model system in a real-world setting.

Objective: The primary objective of this study is to determine what predicts the setting (hospital-based versus home-based) of outpatient therapy services provided to patients upon discharge from an inpatient stroke rehabilitation unit. The secondary objective is to assess the relationship between type of program attended and Functional Independence Measure (FIM) change.

Methods and Materials: This study was a retrospective chart review. Patients meeting the following inclusion were included for analysis: (1) admitted to the stroke inpatient rehabilitation unit between January 1, 2009 and March 1, 2016 then subsequently participated in either the home-based or hospital-based therapy program; (2) had a stroke diagnosis; (3) had an inpatient length of stay >1 day; and (4) lived within the geographical boundaries of the South West Health Network. For outpatient FIM change analyses, patients must have had complete admission and discharge outpatient FIM scores. Inpatient rehabilitation data was collected electronically from the National Rehabilitation Reporting System (e.g., age, gender, stroke type, FIM scores etc.). Outpatient program data were collected through each program’s administrative dataset (e.g., admission/discharge dates, FIM scores, postal code, etc.). Descriptive statistics were calculated for all variables collected. To determine predictors of outpatient therapy setting (home versus hospital) after inpatient rehabilitation a binary logistic regression was conducted. Significance was set a p<0.05. All data were organized and analyzed using SPSS version 23.0.

Results: A total of 721 inpatients attended one of the outpatient therapy programs following their inpatient rehabilitation stay; 360 and 361 inpatients attended the hospital and home-based programs, respectively. Those attending the hospital-based outpatient program were significantly younger, lived closer to the hospital, and had higher inpatient FIM scores at both admission and discharge. Compared to the hospital-based program, patients attending the home-based program had significantly greater FIM changes during outpatient therapy (6.9±8.5 versus 4.4±6.8, p=0.001). The mean total therapy visits were also significantly different between groups, with those in the hospital-based program receiving a greater number of therapy visits than those in the home-based program (41.2±31.7 versus 32.5±19.3, respectively; p=0.005). Participation in the home-based therapy program was associated with a 2.5 increase in total FIM change compared to the hospital-based (p=0.004), and this effect remained statistically significant after adjusting for potential confounding variables. The addition of total therapy visits into the model did not significantly impact the association.

Discussion and Conclusions: Identifying pathways to integrated, high-quality and efficient stroke care is necessary for optimizing health outcomes for patients. Patients receiving both hospital- and home-based therapy made significant gains as measured using the FIM, with the latter group demonstrating modestly greater gains. These findings can now be placed within the context of the entire stroke care continuum, providing insight into one of the least studied areas of the recovery trajectory.
Salon D

IDENTIFYING THE GENETIC BASIS OF VASCULAR COGNITIVE IMPAIRMENT USING A CUSTOM DESIGNED NEXT-GENERATION SEQUENCING-BASED GENE PANEL

ALLISON DILLIOTT
MSc Candidate

Research Areas:
Neuroscience
Mechanisms of disease

Supervisor(s):
R. Hegele

Advisory Committee:
G. Gloor, M. Montero-Odasso

Abstract:
Introduction: Vascular cognitive impairment (VCI) is characterized by cognitive decline resulting from stroke or cerebrovascular accident and is the second most common form of dementia, following Alzheimer’s disease (AD). It is estimated that approximately 30% of individuals who have experienced a stroke will develop VCI. The Ontario Neurodegenerative Disease Research Initiative (ONDRI) is a province-wide, observational cohort study characterizing five neurodegenerative diseases: 1) AD and mild cognitive impairment; 2) amyotrophic lateral sclerosis; 3) frontotemporal dementia; 4) Parkinson’s disease; and 5) VCI. Genetic testing for neurodegenerative disease is becoming more clinically relevant; however, most testing attempts only screen for specific disease-associated mutations. Due to the considerable heterogeneity of the etiology and presentation of these diseases, clinicians may be missing an opportunity to identify novel pathogenic variants in particular neurodegenerative disease-associated genes. The ONDRI genomics subgroup’s objective is to elucidate the often discounted, yet extremely important genetic landscape of these neurodegenerative diseases with the use of targeted next-generation sequencing (NGS). Particularly, in those with VCI we are interested in identifying genetic markers that may place individuals at risk for developing cognitive impairment post-stroke.

Hypothesis: Susceptibility to VCI following a stroke and the disease presentation is determined by a combination of both rare and common single nucleotide variants (SNVs).

Materials and Methods: ONDRI participants were sequenced with the custom NGS panel, ONDRISeq, which targets 80 genes previously associated with the five neurodegenerative diseases of interest. Rare, non-synonymous SNVs likely to be of clinical significance are identified using a custom bioinformatics workflow and manual curation. Additionally, ONDRISeq is able to genotype individuals for the APOE AD risk alleles (rs429358 and rs7412), that determine one’s APOE genotype. The APOE E4/4 genotype is the most commonly accepted genetic risk factor, with the largest effect size yet detected, for developing AD.

Results: The VCI cohort consists of 161 participants with a mean age of 70.2±7.5 years; 31.6% are female. Across the cohort, 255 unique non-synonymous rare variants were identified that may be of clinical significance, with 86% of individuals harbouring at least one of these variants. Interestingly, 12.5% of the unique variants occur in genes that have been previously associated with VCI or related cerebrovascular phenotypes. Analysis of rare variant burden is ongoing, including comparisons with age-matched controls. Further, genomics data will be consolidated with the data that is being compiled by other ONDRI platforms including, but not limited to, neuroimaging, neuropsychology, gait, and ocular testing. Multivariate regression analyses will be used to identify genetic biomarkers that correlate to various phenotypic measures encompassed by VCI.

Discussion and Conclusions: This work will identify known and novel variants, as well as networks of variants that are mediating susceptibility to and symptoms of VCI. The identification of genetic biomarkers associated with VCI onset, progression, and outcomes, may provide important early-diagnosis tools for interventions and possible therapeutic targets.
Abstract:

Introduction: Cardiovascular disease (CVD) is the primary cause of death globally and is responsible for one-third of deaths in Canada. Because the prevalence of CVD is expected to increase due to the obesity epidemic, there is a need to characterize CVD risk factors and develop medical interventions. Epidemiological studies have shown a direct correlation between triglyceride (TG) levels and CVD risk. In patients with extremely elevated TG phenotypes, namely hypertriglyceridemia (HTG), there is an increased prevalence of single-nucleotide variants (SNVs) in TG-associated genes; typically, rare and common genetic variants have large and small phenotypic effects, respectively. Due to past sequencing limitations, HTG has only been studied as a monogenic disorder, with only a fraction of HTG cases being genetically explained by rare SNVs. As a complex trait, TG levels are likely influenced by multiple genetic factors: rare and common SNVs, and copy-number variants (CNVs). To properly understand TG levels and clinically diagnosed HTG, research into additional genetic determinants is required to fully appreciate this CVD risk factor and uncover new, genetically-based strategies to reduce residual CVD risk. I seek to better characterize the genetic determinants driving HTG using next-generation sequencing (NGS) and multiple bioinformatic methods.

Hypothesis: HTG has a genetically diverse architecture comprised of combinations of rare and common variants, including SNVs and CNVs, across multiple genes, cumulatively demonstrating a polygenic effect.

Materials and Methods: 377 patients with severe HTG (TG ≥10 mmol/L) were sequenced for over 200 loci associated with dyslipidemias and metabolic disorders using our targeted NGS panel. For each patient, we screened genes involved in TG metabolism for rare SNVs and CNVs. The bioinformatic tool, VarSeq, was used to annotate and identify rare variants of interest. Next, to quantify the accumulation of common SNVs in patients, we developed a polygenic risk score (PRS) that quantifies the number and phenotypic impact of previously established, TG-associated common SNVs. Patients with extremely high PRSs have an abundance of common SNVs. The same genetic analysis was performed in 503 healthy individuals from the 1000 Genomes Project. Comparisons between groups were done using odds ratios (ORs) from 2x2 contingency tables. Significance was calculated using Fisher’s exact tests.

Results: 20.2% of HTG patients carried rare SNVs or CNVs with likely large phenotypic effects. 29.4% of HTG patients had extremely high PRSs, indicating an abundance of common SNVs. Taken together, 49.6% of HTG patients had either a rare variant or polygenic burden likely contributing towards their HTG phenotype. After performing the same analysis in the cohort of healthy individuals, 15.1% had either a rare variant or polygenic, common SNV burden. Overall, HTG patients are 5.5-fold (CI 95% [3.98-7.69]; P<0.0001) more likely to carry a genetic factor linked to TG, compared to healthy individuals.

Discussion and Conclusions: Our study demonstrates the complex genetic determinants underlying severe HTG and provides insight towards the prevalence of each genetic factor. As NGS technologies and bioinformatic tools improve, the polygenic assortment of factors contributing towards HTG will become even more completely characterized.
Abstract:

Introduction: Recent Canadian research suggests that psychiatric hospitalizations are increasing among adolescents aged 15 to 17. This growing demand will place a burden on the already constrained resources of hospitals, which may leave no other option but to accommodate youth in an adult psychiatry unit. The Ontario Network of Child & Adolescent Inpatient Psychiatry have recommended against this practice due to risks to safety, patient vulnerability, and the potential to exacerbate symptoms. However, there is currently a lack of information on the prevalence of youth who are admitted onto adult psychiatric units and associated impacts of this practice, both nationally and internationally.

Hypothesis: The purpose of this study is to explore the prevalence, determinants, and outcomes related to the hospitalization of youth aged 12 to 17 years on adult inpatient psychiatric units in Ontario. We hypothesize that adolescents admitted to adult psychiatric units will have a greater likelihood of being readmitted within 30-days, that they will have a longer length of stay, and have a higher likelihood of being discharged against medical advice.

Materials and Methods: A cohort of youth between the ages of 12 and 17 years who have experienced an inpatient psychiatric admission in Ontario between 2007 and 2010 was compiled using two health administrative databases containing data on all inpatient psychiatric admissions received from the Canadian Institute for Health Information. The cohort was further divided into two separate groups for comparison: (i) adolescents admitted onto a pediatric psychiatry unit or other non-psychiatry unit and (ii) adolescents admitted onto an adult psychiatry unit. To examine factors related to admission onto an adult psychiatric unit, prevalence ratios will be computed. Modified Poisson regressions will be used to estimate the impact of admission to an adult psychiatric unit on 30-day readmission and discharge against medical advice, and liner regression models will be used to estimate the effect on length of stay.

Results: There were a total of 29,410 adolescent inpatient admissions between April 1, 2007 and March 31, 2011. Of these admissions, 30.3% occurred in adult psychiatric inpatient units. Data analysis is currently in progress and the complete results will be available for the conference.

Discussion and Conclusions: Determining the extent to which adolescents are hospitalized in adult psychiatric units in Ontario and associated impacts will help fill a critical gap in the literature on inpatient mental health services for youth with mental disorders. To our knowledge, this will be the only Canadian study to examine the factors associated with and outcomes of admission in an adult psychiatric unit compared to pediatric/non-psychiatric unit. Youth mental health has been highlighted as a strategic target both provincially and nationally, and this information can be used to determine suitable resource allocation and provide appropriate access to care for adolescents in Ontario.
ENDOGENOUS GLUCOCORTICOIDS RELEASED DURING PROLONGED PSYCHOLOGICAL STRESS IMPAIR ANTI-TUMOUR IMMUNITY INITIATED BY INVARIANT NATURAL KILLER T CELLS

PATRICK RUDAK
PhD Candidate

Research Areas:
Infection and immunity
Mechanisms of disease

Supervisor(s):
S.M. Haeryfar

Advisory Committee:
S.M. Haeryfar, D. Jackson, K. Summers, W. Inoue

Abstract:
Introduction: The nervous system serves critical roles in the regulation of immunity. Consequently, psychological stress can disrupt certain aspects of the immune response, potentially impeding our abilities to mount adequate defenses against malignancies. Yet, the cellular and molecular mechanisms linking mediators of stress to immunological outcomes are poorly understood. Invariant natural killer T (iNKT) cells are innate-like T cells that rapidly trigger robust anti-tumour immune responses upon activation by glycolipid antigens (e.g., α-GalCer) and/or inflammatory cytokines (e.g., IL-12 and IL-18). This study is the first to define how stress impacts protective immune responses initiated by iNKT cells.

Hypothesis: We hypothesized that prolonged psychological stress would suppress iNKT cell-mediated immune responses against cancer.

Materials and Methods: We subjected C57BL/6 mice to prolonged psychological stress, in a well-established model of restraint, before intraperitoneal or intravenous administration of α-GalCer or the combination of IL-12 and IL-18, respectively. Subsequent inflammatory responses were measured by intracellular cytokine staining of isolated iNKT cells and by enzyme-linked immunosorbent assays of serum cytokine levels. Anti-cancer immunity was assessed by enumerating tumour nodules on the lungs 14 days following intravenous injection of B16-F10 metastatic melanoma cells. To extend our findings in vitro, we measured iNKT cell-mediated cytotoxicity against YAC-1 cancer cells using a radioactive chromium release assay. To determine how stress influences the cellular abundance of iNKT cells, we examined their frequencies and absolute numbers within the lymphoid organs of stressed mice by flow cytometry. In some experiments, the functions of glucocorticoids released during stress were inhibited using the glucocorticoid receptor antagonist RU-486. Statistical analyses were conducted using one- or two-way ANOVA or student’s t-test.

Results: We report that prolonged stress abrogates pro-inflammatory cytokine production by activated iNKT cells in vivo, an effect that is reversible by blockade of the glucocorticoid receptor. Accordingly, α-GalCer-stimulated iNKT cells in stressed mice exhibit a failure to protect against the formation of B16-F10 lung metastases in vivo. We also observe a diminished capacity of iNKT cells from stressed mice to initiate cytotoxicity against YAC-1 target cells. Finally, immunosuppression following stress is not due to cell death since, unlike conventional T cells, iNKT cells were resistant to glucocorticoid-induced apoptosis.

Discussion and Conclusions: In support of our hypothesis, we have demonstrated that glucocorticoids released during prolonged psychological stress abolish iNKT cell-mediated immune responses against cancer. Impaired reactivity within stressed individuals has broad implications for targeting iNKT cells for cancer immunotherapy, a topic that is under active clinical investigation. Intriguingly, iNKT cells are insensitive to glucocorticoid-induced apoptosis, potentially allowing for the reversal of their hyporesponsive states during stress. Future work will include a comparison of the transcriptome of iNKT cells from stressed and control mice, which may reveal molecular targets for restoring their protective functions. Ultimately, this research will uncover novel pathways that link the nervous system to immunological competence with relevance to human disease.
THE EFFECT OF WRIST POSITION ON TENDON LOADS FOLLOWING PULLEY SECTIONING AND OPERATIVE RECONSTRUCTION

MOHAMMAD HADDARA
PhD Candidate

Research Areas:
Musculoskeletal health and rehabilitation
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

Supervisor(s):
L. Ferreira

Advisory Committee:
N. Suh, R. Grewal, S. Chinchalkar

Abstract:
Introduction: Surgical reconstruction following pulley rupture is often required for preventing tendon bowstringing and restoring finger kinematics. Post-operative rehabilitation is vital and must balance potential rupture of the pulley reconstruction with aggressive therapy from overly cautious protocols. The purpose of this study is to identify the optimal wrist position required for rehabilitation following reconstruction using tendon load as a metric for strain at the pulleys.

Hypothesis: We believe that placing the wrist in a flexed position would decrease loads experienced within the tendons and therefore reduce strain at the pulley during rehabilitation following a surgical reconstruction of the pulley. This hypothesis contradicts what surgeons believe today where fixing the hand in a neutral position is deemed more beneficial for pulley healing.

Materials and Methods: Fourteen digits comprised of the index, long, and ring fingers were tested from five cadaveric specimens on a validated novel in vitro finger motion simulator devised to actively achieve full finger flexion and extension. Servo-motors were used to generate motion through the tendons under load or position control using a closed-loop feedback system. The simulator is designed to measure and record tendon forces, joint ROM, and tendon excursion. FDP loads were measured sequentially with native intact pulleys, A2 and A4 pulleys sectioned, and finally with reconstructed A2 and A4 flexor tendon pulleys. Each pulley condition was tested in wrist neutral, and 30 degrees of wrist flexion and extension. Using the simulator to measure FDP tendon load, the effects of wrist position on sectioned and reconstructed A2 and A4 pulleys were analyzed using repeated-measures ANOVA.

Results: With the wrist in neutral, FDP tendon loads were 8.5N, 6.2N, and 7.8N with pulleys intact, sectioned, and reconstructed, respectively. With a flexed wrist, the loads were 8.5N, 4.7N, and 5.4N. When the wrist was extended, the loads were 8.7N, 5.2N, and 6.7N. With pulleys reconstructed, the wrist position had a significant effect on tendon load (p=0.030). The flexed wrist position resulted in a 31% reduction of FDP load compared to the neutral wrist position (p=0.010). Wrist extension also produced an apparent reduction, though not statistically significant.

Discussion and Conclusions: Sectioning of the A2 and A4 pulleys resulted in a significant reduction in FDP tendon load in contrast to the intact state. Subsequent reconstruction however restored loads to within no significant difference of the intact state, which supports the decision to reconstruct. Placing the wrist in 30-degree flexion resulted in a reduction of tendon load after reconstruction. These results may suggest that rehabilitation of surgically reconstructed flexor tendon pulleys should be carried out with the wrist flexed in order to reduce strain on pulley reconstructions.
AUTOREACTIVE B CELLS PREFERENTIALLY DIFFERENTIATE INTO UNRESPONSIVE MEMORY B CELLS DURING GERMINAL CENTER RESPONSES

RAJIV JAIN
PhD Candidate

Research Areas:
Infection and immunity
Mechanisms of disease

Supervisor(s):
S. Kerfoot

Advisory Committee:
B. Heit, G. Dekaban

Abstract:
Introduction: B cells have recently been discovered to play a vital role in multiple sclerosis (MS), an autoimmune disease of the central nervous system. However the study of how pathogenic effector B cell subsets are produced in MS has remained unstudied. Here we study germinal center responses, which are composed of germinal center B cells and act as the source of long-lived antibody-secreting plasma cells and memory B cells, induced by immunization with the autoantigen Myelin Oligodendrocyte Glycoprotein (MOG). The objective of this study is to determine whether autoreactive germinal centers, which are normally suppressed, and foreign-antigen induced germinal centers differ in their development and to characterize autoimmune B cell subsets produced from these GCs.

Hypothesis: We hypothesize that germinal center responses induced against an autoantigen will be smaller and less stable than responses induced against a model foreign antigen as a result of properties intrinsic to autoreactive B and T cells.

Materials and Methods: To study germinal center responses we use two model antigen systems, nitrophenyl conjugated-ovalbumin (NP-OVA), a model foreign antigen, and MOG, a model autoantigen. To initiate germinal center responses, fluorescent antigen-specific B and T cells are transferred into non-fluorescent recipient mice that are then immunized and their draining lymph nodes are taken for analysis by flow cytometry, histology, and ELISpot analysis.

Results: While it was possible to generate a germinal center response against the MOG antigen, this response consistently appears to collapse in the germinal center B cell population relative to the NP-OVA response. Furthermore, while plasma cells are produced in abundance during NP-OVA immunization, their numbers are greatly reduced during MOG immunization and this affected long-lasting antibody production. Surprisingly, B cells in the anti-MOG germinal center response are preferentially directed towards a memory B cell like phenotype. Indeed, they can be seen throughout B cell follicles but are primarily excluded from the germinal center relative to the NP-OVA system consistent with memory B cell differentiation. When memory B cells were challenged in a secondary immune response, memory cells from the NP-OVA immune response were capable of responding during secondary immunization whereas memory cells from the MOG immune response could not.

Discussion and Conclusions: This study shows that germinal center responses induced against MOG, a commonly targeted antigen in MS, are characterized by germinal center collapse, inefficient production of plasma cells, and preferential differentiation of B cells into unresponsive memory B cells. The production of unresponsive memory B cells has been associated with chronic infections and autoimmune diseases and this study represents the first description of how these cells can be produced during an immune response.
4:15 - 4:30 P.M. PRESENTATIONS

Salon B

DEVELOPMENT OF A HYBRID OPTICAL SYSTEM FOR STUDYING THE DYNAMIC REGULATION OF BLOOD FLOW/METABOLISM IN THE HUMAN BRAIN

MARWAN SHAHID
MSc Candidate

Research Areas:
Medical biophysics, engineering and imaging
Determinants of health

Supervisor(s):
K. St. Lawrence, M. Diop

Advisory Committee:
K. St. Lawrence, M. Diop, C. Ellis

Abstract:
Introduction: The brain relies on cerebral blood flow (CBF) for a continuous supply of oxygen and energy substrates. Due to its high metabolic demand – 20% of the body’s total energy – and lack of its own energy stores, the brain is vulnerable to injury if CBF is significantly impeded, such as during a stroke. It has also been suggested that subtle changes to CBF regulation could contribute to age-related neurological diseases such as Alzheimer’s. However, this link remains speculative because of the lack of non-invasive methods to study dynamic regulation of blood flow and oxidative metabolism in the human brain. Optical techniques are promising as oxy and deoxyhemoglobin concentrations can be measured by near-infrared spectroscopy (NIRS), CBF can be measured by an emerging technology known as diffuse correlation spectroscopy (DCS), and the combination can be used to quantify cerebral oxidative metabolism. The goal of this project is to develop a hybrid NIRS/DCS system with sufficient temporal resolution to study dynamic coupling of CBF, oxygenation and energy metabolism. The first step is to modify existing NIRS and DCS systems in order to provide simultaneous measurements without crosstalk between the two optical measurements. This study presents initial tests that were conducted to assess the suitability of optical filters for isolating DCS and NIRS signals.

Hypothesis: Simultaneous DCS and NIRS measurements can be achieved using optical filters to prevent signal crosstalk between the two systems, which will enable real-time measurement of CBF, oxygenation and energy metabolism.

Materials and Methods: Because the hybrid system uses different emission lasers for DCS (855 nm) and NIRS (760 and 830 nm), it should be possible to isolate the signals from the two systems with the appropriate high/low pass optical filters. To test the required isolation, experiments were conducted that involved the acquisition of NIRS and DCS data at varying source-detector distances (SDD) since intensity is inversely proportional to distance. A 3D-printed probe holder held emission and detection fibres of both systems in place above a tissue-like liquid phantom. Trials were conducted by collecting DCS data while varying NIRS signal intensity. Likewise, NIRS detectors were set to acquire data with DCS source on.

Results: DCS emission resulted in an immediate saturation of the NIRS detector. Regarding DCS, there was no significant difference in measured relative blood flow when the NIRS lasers were on; however, the signal-to-noise ratio (SNR) diminished significantly with decreasing SDD. DCS acquisition of blood flow at SDD of 1 and 2 cm resulted in no significant difference when compared to control (p = 0.863 and 0.720 respectively) with the NIRS source was positioned 1 cm from the DCS detector. However, there was an increase in the standard deviation (49 and 79% respectively) which was absent when the NIRS was positioned 3 cm from the DCS detector.

Discussion and Conclusions: Preliminary results show that low-pass filtering will be required on the detection side of the NIRS system. In contrast, the NIRS light sources did not have the same effect on DCS detection. The experiments with the tissue-mimicking phantom indicated filters will not be required when operating at a SDD typical in human applications (3 cm). These initial studies indicate that filters will only be required for NIRS, which simplifies the design of the hybrid system.
LOCALIZATION AND TRAFFICKING DYNAMICS OF PANNEXIN 1 IN POLARIZED AND NON-POLARIZED CELLS

MICHELLE SHUM
MSc Candidate

Research Areas:
Molecular cellular
Mechanisms of disease

Supervisor(s):
D. Laird

Advisory Committee:
D. Hamilton, S. Penuela, P. Walton

Abstract:
Introduction: The channel-forming membrane protein Pannexin 1 (Panx1) is ubiquitously expressed in many mammalian tissues. Panx1 is most well-characterized as an ATP release channel and has been linked to over a dozen human pathologies. Along with protein-protein interactions and post-translational modifications, the function of Panx1 channels is highly dictated by its subcellular localization. Interestingly, Panx1 is reported to selectively localize to specific membrane compartments in polarized epithelial cells, a scenario not observed in non-polarized cells, which suggests that the protein may contain internal motifs that guide its trafficking and distribution. Despite this possibility, Panx1 trafficking has only been examined in non-polarized cell lines. Thus, the aims of the present study are: (1) to assess the localization of Panx1 in polarized and non-polarized cells in vitro; and (2) to identify motifs within the Panx1 polypeptide that may act to target Panx1 to specific membrane domains of polarized cells.

Hypothesis: We hypothesize that motifs within the Panx1 polypeptide direct its trafficking to distinct plasma membrane domains of polarized epithelial cells.

Materials and Methods: Wildtype (WT) or mutant (Y308F, LL-365/366-AA or C-terminally truncated) Panx1 was tagged with moxGFP and expressed in both Madin-Darby canine kidney (MDCK) cells and BICR-M1Rk cells, a line of non-polar rat mammary tumor cells. MDCK cells were cultured under polarizing and non-polarizing conditions. Trans-epithelial electrical resistance was used to assess MDCK cell polarization. To evaluate Panx1 localization, cells were fixed and immunostained for markers of the apical and basolateral plasma membrane domains, as well as resident proteins of intracellular compartments. Colocalization of Panx1-moxGFP with immunolabeled proteins was quantified using Manders coefficients.

Results: WT Panx1 was localized throughout the plasma membrane of non-polarized MDCK and BICR-M1Rk cells without spatial restriction; however, after MDCK cell polarization, Panx1 was preferentially distributed to the apical surface. The apical localization of Panx1 was further confirmed in three-dimensional MDCK cell spheroids. Preliminary experiments involving the mutation of the dileucine motif at the C-terminal domain of Panx1 into alanine residues (LL-365/366-AA) revealed no disruption in the ability of the protein to traffic to the apical membrane domain of polarized cells, indicating that this region of Panx1 may not be a key targeting motif. Studies examining the role of putative Panx1 phosphorylation sites and the C-terminal tail in selectively targeting the protein to the apical membrane of polarized cells are currently under way.

Discussion and Conclusions: Taken together, our data indicate that Panx1 acquires a predominantly apical distribution upon MDCK cell polarization, suggesting that it must contain a sorting signal to guide this selective trafficking. This polarized targeting appears independent of the classical dileucine-based sorting motif contained within the C-terminal tail of the protein. Understanding the molecular determinants of Panx1 trafficking in polarized cells is essential to uncovering the physiological roles of these membrane channels in the context of the epithelial cell microenvironment.
MACHINE LEARNING TO PREDICT POST-TRAUMATIC STRESS DISORDER AND ITS DISSOCIATIVE SUBTYPE

ANDREW NICHOLSON  
Postdoctoral Scholar

Research Areas:  
Mental health and wellness  
Detection, screening and diagnosis of health and disease

Supervisor(s):  
R. Lanius

Advisory Committee:  
N/A

Abstract:  
Introduction: Unique neurobiological correlates have been identified that distinguish patients with post-traumatic stress disorder (PTSD) from its dissociative subtype and healthy controls. However, these mass-univariate analyses are restricted in terms of their clinical applicability, as they cannot be used to accurately diagnose patients on the individual level. We present the first study to use resting-state fMRI data as inputs to machine learning algorithms in order to classify individual patients with PTSD and dissociative subtype PTSD psychopathology.

Hypothesis: We predicted that both resting-state activation, as well as amygdala complex seed-based functional connectivity, would accurately predict PTSD, dissociative subtype PTSD, and healthy control individual classification.

Materials and Methods: We obtained 6-minute resting-state fMRI scans from n=187 participants (82 non-subtype PTSD, 49 dissociative subtype PTSD, and 56 healthy controls). We calculated the mean amplitude-of-low-frequency-fluctuations (mALFF) for each participant, as well as functional connectivity maps for each of the bilateral amygdala complexes (basolateral, centromedial, and superficial amygdala complexes). These were used as inputs for multiclass Gaussian Process Classification (MGPC) multivariate pattern analyses (MVPA) implemented in PRoNTo software running in Matlab.

Results: The mALFF of participants was able to classify individuals with 90% balanced accuracy. Furthermore, amygdala complex functional connectivity maps were able to classify participants with 85% balanced accuracy. Here, mass-univariate analyses similarly identified key regions that were also central for classification in the machine learning analysis, which included the orbitofrontal cortex, globus pallidus, supramarginal gyrus and dorsolateral prefrontal cortex.

Discussion and Conclusions: The current study has significant clinical applications in terms of validating objective diagnostic measures that could be used to improve the early identification of PTSD and early intervention/treatment of the disorder.
VALIDATION OF A NON-INVASIVE OPTICAL METHOD FOR MEASURING CEREBRAL BLOOD FLOW DURING CRITICAL CARE

DANIEL MILEJ
Postdoctoral Scholar

Research Areas:
Medical biophysics, engineering and imaging
Detection, screening and diagnosis of health and disease

Supervisor(s):
K. St. Lawrence

Advisory Committee:
N/A

Abstract:
Introduction: A key management strategy for acute brain injury is to maintain cerebral blood flow (CBF) such that oxygen and glucose delivery matches metabolic demands. Patient-specific implementation of this strategy could be aided by the use of bedside CBF monitors for rapid detection of ischemic conditions. Herein, we validate a noninvasive optical technique (near-infrared spectroscopy, NIRS) for measuring CBF by comparison to concurrent measurements obtained using the magnetic resonance imaging perfusion technique known as arterial spin labeling (ASL). The major challenge to adopting this optical method to human adults is signal contamination from extracerebral tissue. This study implemented time-resolved (TR) detection and a layered model of the head to account for extracerebral signal contamination. Experiments involved healthy volunteers and were conducted in an MRI scanner to provide simultaneous CBF measurements by NIRS and ASL.

Hypothesis: Time-resolved NIRS can accurately measure absolute CBF values in adults.

Materials and Methods: Eleven healthy subjects (3 females, aged 22 to 54 years) with no history of neurological or psychiatric disorders were recruited. Experiments were performed in a 3T Verio system (Siemens Medical Systems, Germany). For each subject, two sets of simultaneous DCE-NIRS and ASL data were collected at normocapnia and hypercapnia to ascertain whether the method was sensitive to increased CBF. The latter was induced by having subjects breathe 6% CO2 through a facemask for a duration of 5 min. The NIRS technique for measuring CBF required an intravenous injection of an optical contrast agent (indocyanine green, ICG), followed by a continuous recording of TR NIRS data from optical probes placed on the subject’s forehead to monitor the washin and clearance of the dye from the brain. Each subject received two injection (one at normocapnia and one at hypercapnia) while ASL images of CBF were continuously recorded.

Results: Comparing the absolute CBF estimates from TR-NIRS and ASL revealed good agreement between the two techniques (R2 = 0.82, slope = 0.89 and a y-intercept of 4.45 ml/100 g/min). Bland-Altman analysis indicated a mean difference of -6.7 mg/100g/min, which was not significantly different from zero. The average resting CBF computed with DCE-NIRS and ASL were 68 ± 10 ml/100g/min and 75 ± 14 ml/100g/min, respectively (mean±SD). Both NIRS and ASL showed the expected increase in CBF during hypercapnia of 38 ± 21 ml/100g/min and 35 ± 15 ml/100g/min, respectively (mean±SD).

Discussion and Conclusions: This study showed that NIRS with depth-sensitive detection can quantify cerebral blood flow in adults. In addition to showing a good agreement in absolute values, these experiments also revealed that the technique was sensitive to flow changes. This is the first study to show that NIRS can measure CBF in adults, which highlights the potential of this technology for bedside monitoring of brain health in critical-care patients with neurological emergencies (e.g., acute stroke, traumatic brain injury).
Introduction: Atherosclerosis is a chronic inflammatory disease involving formation of lipoprotein-rich lesions in the arterial intima and infiltration of these lesions by macrophages, which then proceed to take up cholesterol and differentiate into pathological foam cells and drive disease progression. Macrophage accumulation and dysregulation are key initial steps in the pathogenesis of atherosclerosis. Animal models of atherosclerosis have identified several mechanisms driving pathological change in lesion-resident macrophages, but reproducing these results in human disease remains challenging. Our objective is to examine gene expression in macrophages isolated from patient coronary atherosclerotic lesions in order to better characterize macrophage dysfunction in human disease.

Materials and Methods: Aortic wall punch samples were obtained from coronary artery bypass graft surgery patients. Samples were sectioned and sections were stained using Oil Red O and Movat’s stain to establish gross sample pathology and stage atherosclerotic disease. Sections were further stained using an anti-CD163 antibody to identify lesion-resident macrophage populations, which were isolated through laser capture microdissection (LCM). Gene expression profiling was performed on LCM-dissected macrophage populations by human whole-exome microarray, with macrophages differentiated in vitro with M-CSF from peripheral blood mononuclear cell (PBMC) from healthy age-matched donors as a control.

Results: We established through Oil Red O and Movat’s staining that our samples contained evidence of intimal thickening in the absence of plaque and necrotic core formation, indicating an early stage of atherosclerotic disease. Macrophage cell populations were successfully isolated from punch samples and PBMCs of three patients and three healthy controls respectively. qPCR analysis demonstrated enrichment of macrophage-specific CD14 and diminishment of smooth muscle-specific αSMA in our dissected cell population compared to whole-section controls. Gene expression profiling revealed a total of ~5,300 differentially regulated genes in patient lesion-resident macrophages, with particular enrichment in pathways involving intracellular transport, phagocytosis and antigen processing. In particular, we identified upregulation of the hematopoietic transcription factor GATA2 (single nucleotide polymorphisms in GATA2 are associated with coronary artery disease and its risk factors) and several genes regulated by this transcription factor in atherosclerotic macrophages. qPCR analysis demonstrated that GATA2 was upregulated approximately 45× in patient macrophages compared to controls.

Discussion and Conclusions: This study is to our knowledge the first to assess the transcriptional profile of intima-infiltrating macrophages from the initial stages of coronary atherosclerosis in human subjects. We identify phagocytosis and antigen processing and presentation as potentially important pathways that become dysregulated in atherosclerotic macrophages, and are the first to identify a potential role for GATA2 in macrophage dysfunction in atherosclerosis.
PHYSICIANS’ PRODUCTIVITY UNDER FEE-FOR-SERVICE AND BLENDED FEE-FOR-SERVICE: EVIDENCE FROM ONTARIO NATURAL EXPERIMENT

NIBENE SOMÉ
Postdoctoral Scholar

Research Areas:
Population health and education
Advancing health services provision and health policy

Supervisor(s):
S. Sarma

Advisory Committee:
S. Sarma

Abstract:
Introduction: In the province of Ontario, the vast majority of family physicians have traditionally been paid according to a fee-for-service (FFS) payment scheme, under which a physician receives a fee for each service provided. In July 2003, the Government of Ontario introduced a blended FFS known as the Family Health Group (FHG) model, under which physicians receive 100 per cent FFS payments plus bonuses and incentives to provide comprehensive care and required to provide services during after-hours. Understanding how physicians respond to such changes in incentives has important policy implications in relation to the supply of healthcare services. This study evaluates the impact of switching family physicians from FFS to FHG payment on total services, comprehensive care services, after-hours services and other services.

Hypothesis: Based on the theoretical economics literature on physician behaviour, we hypothesize that the productivity of physicians who switched from FFS to FHG will be greater than the productivity of those who remained under FFS.

Materials and Methods: The data for this study come from the detailed information on Ontario family physicians’ billings data between April 1st, 2003 (year of FHG introduction) and March 31st, 2008. The data are obtained from several health administrative databases held at the Institute for Clinical and Evaluative Sciences (ICES) in Ontario, Canada. To estimate the impact of switching from FFS to FHG on physicians’ productivity, we use a two-step estimation strategy. In the first stage, we account for pre-reform observed differences between switchers and non-switchers as those switched to FHG may be systematically different from those remained in FFS, leading to potential selection bias. To address this concern, we use propensity score matching (PSM), and recent doubly robust statistical methods: covariate balancing propensity score (CBPS) and entropy balancing (EB). In the second stage, we use panel data models to account for both observed physician and practice characteristic and unobserved physician-specific heterogeneity affecting the productivity. In order to estimate the causal impact of switching from FFS to FHG, we use a weighted difference-in-difference regression model using inverse probability weighting technique.

Results: Our results show that physicians respond to incentives in a predictable manner. We find that, on average, switching from FFS to FHG increases the volume of total services, after-hours services and other services by about 4.0%, 5.4%, and 4.1%, respectively. Also, switching to FHG increases the comprehensive care services by 4.6%, suggesting a potential improvement in quality and continuity of care. These results are robust to CBPS and EB methodologies.

Discussion and Conclusions: We find that physicians who switched from the traditional fee-for-service to the blended fee-for-service increased their productivity in the neighbourhood of 4% to 5%. Our results have important policy implications for the provision of healthcare services. In an era of increased demand for medical services, governments can use financial incentive mechanisms to increase the supply of services. Although our results are robust to the alternative matching approaches employed, it is still based on the assumption that the matching equation in the first stage is correctly specified. This may not hold true if any unobservable factors influence selection into FHG.
INTRAOPERATIVE NEEDLE PLACEMENT VERIFICATION DURING INTERSTITIAL GYNECOLOGIC BRACHYTHERAPY NEEDLE INSERTION USING A 360° 3D TRANSVAGINAL ULTRASOUND SYSTEM

JESSICA RODGERS
PhD Candidate

Research Areas:
Medical biophysics, engineering and imaging
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)

Supervisor(s):
A. Fenster

Advisory Committee:
A. Ward, K. Surry, V. Velker

Abstract:
Introduction: High-dose-rate interstitial brachytherapy is a treatment option for gynecologic cancers where hollow needles are inserted through a perineal template and a radioactive source is placed at planned positions via these needles to allow higher radiation doses to be delivered to the tumor relative to nearby healthy tissues. To avoid overexposure of organs at risk (OAR), including the rectum, bladder, and bowel, and deliver optimal treatment, precise needle placement is necessary; however, there is currently no standard method to assess needles intraoperatively during the insertion procedure. Patients currently receive a post-insertion x-ray computed tomography (CT) scan to perform dose planning but implementation of an intraoperative needle verification tool would allow for OAR to be avoided and needle placements to be refined, potentially improving the quality of the implant. We have developed a 3D transvaginal ultrasound (TVUS) system that produces a 360° 3D image through a template-compatible sonolucent vaginal cylinder and propose its use for intraoperative needle placement verification during HDR interstitial gynecologic brachytherapy.

Hypothesis: We hypothesize that the use of 3D TVUS imaging for gynecologic brachytherapy needle insertion will allow for accurate intraoperative assessment of the implant by providing visualization and localization of needles with mean angular errors < 3 ° and mean distance errors < 5 mm.

Materials and Methods: We developed a 3D TVUS system that rotates a 2D side-fire transrectal ultrasound (US) probe 360° using a motorized mechanism, generating a ring-shaped 3D image. The 3D scan takes approximately 20 s and allows the user to view the image immediately after acquisition. Before acquisition, the probe is placed inside the hollow core of a sonolucent plastic cylinder that is inserted transvaginally, mimicking the current template vaginal cylinder. Three patients receiving interstitial gynecologic brachytherapy at the London Regional Cancer Program were imaged using the 3D TVUS system. For each needle placed, the entrance and exit points of the needle in the field-of-view were identified in the 3D TVUS and rigidly registered clinical CT images. Corresponding entrance and exit points between the modalities were established for each needle and used to assess angular and distance errors in the needle paths identified. The maximum distance error was calculated using the maximum distance value (entrance or exit) for each needle.

Results: Features of interest, including the patient’s rectum, urethra, vaginal wall, bowel, and bladder with an inserted Foley catheter, were clearly visualized in the 3D US images. For the 28 needles placed, the mean angular error between needle paths in the two modalities was 1.67 ± 0.75 ° and there were no trends in the direction of the errors relative to the image planes. The mean entrance point error was 1.98 ± 0.92 mm and the mean exit point error was 1.92 ± 0.81 mm, with a mean maximum distance error of 2.33 ± 0.78 mm.

Discussion and Conclusions: Based on this preliminary patient study, 360° 3D TVUS may be a feasible option for intraoperatively visualizing and localizing needles during interstitial gynecologic brachytherapy needle insertion. The 360° 3D TVUS images allowed OAR to be visualized intraoperatively and the visualization of needles provides the potential to improve implant quality in the future, with a 15 patient proof-of-concept study ongoing.
THE EFFECT OF ATF4 UPREGULATION ON NEURONAL GLUTATHIONE LEVELS

FATEMEH MIRSHAFIEI
MSc Candidate

Research Areas:
Neuroscience
Mechanisms of disease

Supervisor(s):
S. Cregan

Advisory Committee:
R. Cumming, P. Chidiac

Abstract:
Introduction: Activating Transcription Factor 4 (ATF4) is a proapoptotic transcription factor in neurons. The expression of ATF4, as well as its downstream gene targets has been implicated in both in vivo and in vitro models of Alzheimer’s and Parkinson’s Disease. Furthermore, the combined effect of ATF4 overexpression and oxidative stress has been correlated with a reduction in neuronal glutathione (GSH) levels. One of the important precursors for regeneration of GSH in the cells is NADPH produced in the Pentose Phosphate Pathway (PPP), which is the preferred pathway for glucose metabolism in neurons.

Hypothesis: We hypothesize that expression of ATF4 will cause neuronal cell death by affecting the regenerative ability of GSH in the cells.

Materials and Methods: Primary cortical neurons are extracted from embryonic day 13 (E13) wild type (WT) and ATF4-deficient mice. Neurons are treated with various compounds and different parameters such as GSH, reactive oxygen species (ROS), glutathione reductase (GR) activity and NADPH levels are measured and compared between the two genotypes.

Results: Treatment of neurons with 1 µM thapsigargin, an activator of ATF4, for 12 hours caused a significant decrease in GSH level in WT neurons (n=7) while the level was unchanged in ATF-deficient cell (n=8) (p-value<0.001). Neurons were then treated with BSO to block the synthesis of GSH. Although blocking de novo synthesis of glutathione caused a reduction in both WT (n=5) and ATF4-KO neurons (n=5) (p-value<0.001), the WT cells showed a more significant loss compared to the KO cells (p-value=0.002).

Discussion and Conclusions: Currently we have evidence that endogenous upregulation of ATF4 is sufficient to reduce GSH levels. We also show that ATF4-deficient neurons are more resistant to depletion of GSH reserves after blocking de novo synthesis of GSH using BSO. Future studies intend to elucidate the mechanism behind this progressive loss of GSH in the presence of ATF4 by specifically comparing the oxidative processes, the activity of glutathione reductase and the level of NADPH in both wildtype and ATF4-deficient neurons.
**APPENDIX A: POSTER PRESENTATIONS OVERVIEW**

**MORNING SESSION**

<table>
<thead>
<tr>
<th>Name</th>
<th>Research Area</th>
<th>Poster Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abbasi, Sanna</td>
<td>Mechanisms of disease</td>
<td>90</td>
</tr>
<tr>
<td>Abdalmalak, Androu</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>38</td>
</tr>
<tr>
<td>Abdul'satar, Farah</td>
<td>Population, public health and education</td>
<td>153</td>
</tr>
<tr>
<td>Ahmed, Juweirya</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>39</td>
</tr>
<tr>
<td>Ahmed, Khadija</td>
<td>Mechanisms of disease</td>
<td>91</td>
</tr>
<tr>
<td>Alfano, Ryan</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>40</td>
</tr>
<tr>
<td>Al-Jaishi, Ahmed</td>
<td>Population, public health and education</td>
<td>154</td>
</tr>
<tr>
<td>Atkinson, Hayden</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>41</td>
</tr>
<tr>
<td>Au, Akina</td>
<td>Mechanisms of disease</td>
<td>92</td>
</tr>
<tr>
<td>Azizi, Nawab</td>
<td>Mechanisms of disease</td>
<td>93</td>
</tr>
<tr>
<td>Baer, Brandon</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>1</td>
</tr>
<tr>
<td>Baker, Cadence</td>
<td>Advancing health services provision and health policy</td>
<td>29</td>
</tr>
<tr>
<td>Barrett, Sierra</td>
<td>Advancing health services provision and health policy</td>
<td>30</td>
</tr>
<tr>
<td>Bartman, Kevin</td>
<td>Mechanisms of disease</td>
<td>94</td>
</tr>
<tr>
<td>Bouisset, Nicolas</td>
<td>Advancing health services provision and health policy</td>
<td>31</td>
</tr>
<tr>
<td>Bray, Nick</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>163</td>
</tr>
<tr>
<td>Brooks, Jeffrey</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>42</td>
</tr>
<tr>
<td>Buttazzoni, Adrian</td>
<td>Population, public health and education</td>
<td>155</td>
</tr>
<tr>
<td>Caron, Christine</td>
<td>Mechanisms of disease</td>
<td>95</td>
</tr>
<tr>
<td>Chadwick, Sarah</td>
<td>Mechanisms of disease</td>
<td>96</td>
</tr>
<tr>
<td>Chang, Angela</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>2</td>
</tr>
<tr>
<td>Charbonneau, James</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>43</td>
</tr>
<tr>
<td>Ching, Jamie</td>
<td>Early life programming and development</td>
<td>80</td>
</tr>
<tr>
<td>Choi, Joshua</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>3</td>
</tr>
<tr>
<td>Christiansen, Spencer</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>44</td>
</tr>
<tr>
<td>Coelho, Brett</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>164</td>
</tr>
<tr>
<td>Colbran, Kiersten</td>
<td>Population, public health and education</td>
<td>156</td>
</tr>
<tr>
<td>Demarco, John</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>45</td>
</tr>
<tr>
<td>Denstedt, Jim</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>4</td>
</tr>
<tr>
<td>Dexter, Tyler</td>
<td>Mechanisms of disease</td>
<td>97</td>
</tr>
<tr>
<td>Dion, Charles-Antoine</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>5</td>
</tr>
<tr>
<td>Donnelly, Sarah</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>46</td>
</tr>
<tr>
<td>Dumbrava, Mihai</td>
<td>Mechanisms of disease</td>
<td>98</td>
</tr>
<tr>
<td>Dunkerley, Karen</td>
<td>Mechanisms of disease</td>
<td>99</td>
</tr>
<tr>
<td>Eddy, Rachel</td>
<td>Mechanisms of disease</td>
<td>100</td>
</tr>
<tr>
<td>Engineer, Anish</td>
<td>Early life programming and development</td>
<td>81</td>
</tr>
<tr>
<td>Esakki, Saravanan</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>47</td>
</tr>
<tr>
<td>Faieghi, Mohamadreza</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>6</td>
</tr>
<tr>
<td>Frame, Ariel</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>165</td>
</tr>
<tr>
<td>Franco, Roseane</td>
<td>Mechanisms of disease</td>
<td>101</td>
</tr>
<tr>
<td>Furlano, Joyla</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>166</td>
</tr>
<tr>
<td>Gan, Alisa</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>7</td>
</tr>
<tr>
<td>Gan, Ingrid</td>
<td>Mechanisms of disease</td>
<td>102</td>
</tr>
<tr>
<td>Gatie, Mohamed</td>
<td>Early life programming and development</td>
<td>82</td>
</tr>
<tr>
<td>Gautam, Bishal</td>
<td>Determinants of health</td>
<td>72</td>
</tr>
<tr>
<td>Ge, Fang Zhou</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>48</td>
</tr>
<tr>
<td>German-Castelan, Liliana</td>
<td>Mechanisms of disease</td>
<td>103</td>
</tr>
<tr>
<td>Good, Hayley</td>
<td>Mechanisms of disease</td>
<td>104</td>
</tr>
</tbody>
</table>
Gray, Rob  
Detection, screening and diagnosis of health and disease  
49

Greco, Elizabeth  
Early life programming and development  
83

Haddad, Faraj  
Early life programming and development  
84

Halari, Chidambra  
Mechanisms of disease  
105

Han, Wenchao  
Detection, screening and diagnosis of health and disease  
50

Harnett, Amber  
Mechanisms of disease  
106

Harricharan, Sherain  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
8

Hill, Seana  
Mechanisms of disease  
107

Hui, Daphne  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
9

Hurst, Jacklyn  
Mechanisms of disease  
108

Iacocca, Michael  
Detection, screening and diagnosis of health and disease  
51

Ichiyama, Aoi  
Mechanisms of disease  
109

Ip, Kenneth  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
10

Jackson, Alexandra  
Population, public health and education  
157

Jacobs, Kaitlyn  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
11

Jaju, Gargi  
Early life programming and development  
85

Janssen, Sarah  
Advancing health services provision and health policy  
32

Jaremek, Victoria  
Mechanisms of disease  
110

Jeon, Peter  
Detection, screening and diagnosis of health and disease  
52

Jeyarajah, Mariyan  
Mechanisms of disease  
111

Jiang, Yuwei  
Mechanisms of disease  
112

Jumbo, Samuel  
Advancing health services provision and health policy  
33

Kewin, Matthew  
Determinants of health  
73

Khalid, Mahro  
Detection, screening and diagnosis of health and disease  
53

Khazaee, Reza  
Prevention of diseases and health conditions and promotion of well-being  
167

Kim, Kunmo  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
12

Kim, Nicholas  
Determinants of health  
74

Kitz, Jenna  
Detection, screening and diagnosis of health and disease  
54

Klubowicz, Dorota  
Prevention of diseases and health conditions and promotion of well-being  
168

Kohio, Hinissan  
Mechanisms of disease  
113

Kramar, Cecilia  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
13

Kudaeva, Fatima  
Mechanisms of disease  
114

Kyle, Emily  
Population, public health and education  
158

Lackie, Rachel  
Mechanisms of disease  
115

Leclerc, Chris  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
14

Lee, Jasper  
Mechanisms of disease  
116

Levine, Jeffrey  
Prevention of diseases and health conditions and promotion of well-being  
169

Li, Fiona  
Detection, screening and diagnosis of health and disease  
55

Liu, Elaine  
Mechanisms of disease  
117

Lo, Marcus  
Early life programming and development  
86

Lone, Asad  
Mechanisms of disease  
118

Maksoud, Matthew  
Mechanisms of disease  
119

Marants, Raanan  
Mechanisms of disease  
120

McKillop, Isabelle  
Detection, screening and diagnosis of health and disease  
56

Miranda, Artur  
Determinants of health  
75

Mohideen, Shabna  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
15

Moszczyński, Alexander  
Mechanisms of disease  
121

Murphy, Patrick  
Detection, screening and diagnosis of health and disease  
57

Naghibosadat, Maedeh  
Mechanisms of disease  
122

Nagrani, Niharika  
Mechanisms of disease  
123

Nan, Tomi  
Detection, screening and diagnosis of health and disease  
58

Ngo, Geoffrey  
Mechanisms of disease  
124

Ning, Rufina  
Mechanisms of disease  
125

Nisar, Hareem  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  
16
<table>
<thead>
<tr>
<th>Name</th>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nunez de Villavicencio Diaz,</td>
<td>Mechnisms of disease</td>
<td>126</td>
</tr>
<tr>
<td>Teresa</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td></td>
</tr>
<tr>
<td>Nyström, Nivin</td>
<td>therapeutic intervention (includes surgery and drugs)</td>
<td>17</td>
</tr>
<tr>
<td>Oakie, Amanda</td>
<td>Mechnisms of disease</td>
<td>127</td>
</tr>
<tr>
<td>Oke, Shelby</td>
<td>Early life programming and development</td>
<td>87</td>
</tr>
<tr>
<td>Ong Ly, Cathy</td>
<td>Mechnisms of disease</td>
<td>128</td>
</tr>
<tr>
<td>Palmer, Daniel</td>
<td>Mechnisms of disease</td>
<td>129</td>
</tr>
<tr>
<td>Patel, Annal</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>18</td>
</tr>
<tr>
<td>Pearce, Alexandra</td>
<td>therapeutic intervention (includes surgery and drugs)</td>
<td></td>
</tr>
<tr>
<td>Peidl, Alex</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>19</td>
</tr>
<tr>
<td>Perampalam, Pirunthan</td>
<td>therapeutic intervention (includes surgery and drugs)</td>
<td></td>
</tr>
<tr>
<td>Peters, Kia</td>
<td>Mechnisms of disease</td>
<td>130</td>
</tr>
<tr>
<td>Pitts, Conrad</td>
<td>Determinants of health</td>
<td>76</td>
</tr>
<tr>
<td>Prusinkiewicz, Martin</td>
<td>Mechnisms of disease</td>
<td>132</td>
</tr>
<tr>
<td>Puebla-Barragan, Scarlett</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>171</td>
</tr>
<tr>
<td>Pur, Daiana</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>59</td>
</tr>
<tr>
<td>Rab, Faiza</td>
<td>Advancing health services provision and health policy</td>
<td>34</td>
</tr>
<tr>
<td>Racanelli, Michael</td>
<td>Mechnisms of disease</td>
<td>133</td>
</tr>
<tr>
<td>Raji, Sadiq</td>
<td>Advancing health services provision and health policy</td>
<td>35</td>
</tr>
<tr>
<td>Rankin, Adam</td>
<td>Determinants of health</td>
<td>77</td>
</tr>
<tr>
<td>Raslan, Omar</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>60</td>
</tr>
<tr>
<td>Reingold, Rachel</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>172</td>
</tr>
<tr>
<td>Rhee, Jess</td>
<td>Mechnisms of disease</td>
<td>134</td>
</tr>
<tr>
<td>Robinson, Michael</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>61</td>
</tr>
<tr>
<td>Rohani, Aliureka</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>20</td>
</tr>
<tr>
<td>Santos, Juliane</td>
<td>Mechnisms of disease</td>
<td>136</td>
</tr>
<tr>
<td>Shanoada, Roni</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>62</td>
</tr>
<tr>
<td>Sharma, Mayank</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>21</td>
</tr>
<tr>
<td>Siddiqui, Naveed</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>22</td>
</tr>
<tr>
<td>Sleiman, Aref</td>
<td>Advancing health services provision and health policy</td>
<td>36</td>
</tr>
<tr>
<td>Smith, Christopher</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>23</td>
</tr>
<tr>
<td>Smith, Nicole</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>63</td>
</tr>
<tr>
<td>Solomon, Lauren</td>
<td>Advances in structural and physiological treatment of disease and</td>
<td>24</td>
</tr>
<tr>
<td>Stanley, Olivia</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>64</td>
</tr>
<tr>
<td>Sukhera, Javeed</td>
<td>Population, public health and education</td>
<td>159</td>
</tr>
<tr>
<td>Sule, Akshay</td>
<td>Mechnisms of disease</td>
<td>138</td>
</tr>
<tr>
<td>Sullivan, Rebecca</td>
<td>Mechnisms of disease</td>
<td>139</td>
</tr>
<tr>
<td>Sun, Qin</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>65</td>
</tr>
<tr>
<td>Sunstrum, Julia</td>
<td>Mechnisms of disease</td>
<td>140</td>
</tr>
<tr>
<td>Syed, Imran</td>
<td>Determinants of health</td>
<td>78</td>
</tr>
<tr>
<td>Szlapinski, Sandra</td>
<td>Early life programming and development</td>
<td>88</td>
</tr>
<tr>
<td>Tamming, Renee</td>
<td>Mechnisms of disease</td>
<td>141</td>
</tr>
<tr>
<td>Tariq, Ulaina</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>173</td>
</tr>
<tr>
<td>Taruc, Kyle</td>
<td>Mechnisms of disease</td>
<td>142</td>
</tr>
<tr>
<td>Taylor, Leah</td>
<td>Population, public health and education</td>
<td>160</td>
</tr>
<tr>
<td>Terpou, Braeden</td>
<td>Determinants of health</td>
<td>79</td>
</tr>
<tr>
<td>Tesfagiorgis, Yodit</td>
<td>Mechnisms of disease</td>
<td>143</td>
</tr>
<tr>
<td>Thavam, Thaksha</td>
<td>Advancing health services provision and health policy</td>
<td>37</td>
</tr>
<tr>
<td>Thomas, Anu</td>
<td>Mechnisms of disease</td>
<td>144</td>
</tr>
<tr>
<td>Truelove, Stephanie</td>
<td>Population, public health and education</td>
<td>161</td>
</tr>
<tr>
<td>Vannelli, Claire</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>66</td>
</tr>
<tr>
<td>Wang, Helen</td>
<td>Population, public health and education</td>
<td>162</td>
</tr>
<tr>
<td>Name</td>
<td>Research Area</td>
<td>Poster Number</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Abbasalipour, Parvaneh</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>38</td>
</tr>
<tr>
<td>Abdalla, Khalid</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>39</td>
</tr>
<tr>
<td>Abibol, Julia</td>
<td>Mechanisms of disease</td>
<td>90</td>
</tr>
<tr>
<td>Abolhasani, Ehsan</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>1</td>
</tr>
<tr>
<td>Alotaibi, Faizah</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>162</td>
</tr>
<tr>
<td>Alsuaibie, Rasha</td>
<td>Mechanisms of disease</td>
<td>91</td>
</tr>
<tr>
<td>Asadi, Farzad</td>
<td>Mechanisms of disease</td>
<td>92</td>
</tr>
<tr>
<td>Au, Natalie</td>
<td>Determinants of health</td>
<td>71</td>
</tr>
<tr>
<td>Axford, David</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>2</td>
</tr>
<tr>
<td>Basu, Monisha</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>40</td>
</tr>
<tr>
<td>Berberich, Amanda</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>41</td>
</tr>
<tr>
<td>Berg, Matthew</td>
<td>Mechanisms of disease</td>
<td>93</td>
</tr>
<tr>
<td>Bernardinis, Matthew</td>
<td>Mechanisms of disease</td>
<td>94</td>
</tr>
<tr>
<td>Best, Sarah</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>163</td>
</tr>
<tr>
<td>Bhatti, Dhwanil</td>
<td>Determinants of health</td>
<td>72</td>
</tr>
<tr>
<td>Biswas, Saumik</td>
<td>Mechanisms of disease</td>
<td>95</td>
</tr>
<tr>
<td>Blom, Jessica</td>
<td>Population, public health and education</td>
<td>152</td>
</tr>
<tr>
<td>Bloomfield, Riley</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>42</td>
</tr>
<tr>
<td>Buenesuceso, Adrian</td>
<td>Mechanisms of disease</td>
<td>96</td>
</tr>
<tr>
<td>Buitrago-Piza, Diego</td>
<td>Mechanisms of disease</td>
<td>97</td>
</tr>
<tr>
<td>Cao, Ting</td>
<td>Mechanisms of disease</td>
<td>98</td>
</tr>
<tr>
<td>Chang, Megan</td>
<td>Advancing health services provision and health policy</td>
<td>29</td>
</tr>
<tr>
<td>Chaudhari, Sumit</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>3</td>
</tr>
<tr>
<td>Chevalier, Jacqueline</td>
<td>Mechanisms of disease</td>
<td>99</td>
</tr>
<tr>
<td>Cho, William</td>
<td>Early life programming and development</td>
<td>80</td>
</tr>
<tr>
<td>Chu, Lucy</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>4</td>
</tr>
<tr>
<td>Chung, Steve</td>
<td>Mechanisms of disease</td>
<td>100</td>
</tr>
</tbody>
</table>

**AFTERNOON SESSION**
Cochrane, Ryan  
Prevention of diseases and health conditions and promotion of well-being  

Crawford, Melissa  
Mechanisms of disease  

Cruje, Charmainne  
Detection, screening and diagnosis of health and disease  

Daisley, Brendan  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Dassanayake, Praveen  
Detection, screening and diagnosis of health and disease  

Dempsey, Sergio  
Detection, screening and diagnosis of health and disease  

Deweyert, Andrew  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Dey, Avarthanana  
Determinants of health  

Dierolf, Joshua  
Early life programming and development  

Dirk, Brennan  
Mechanisms of disease  

Easton, Zachary  
Early life programming and development  

Edwards, Jordan  
Population, public health and education  

Elsariti, Asmahan  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Esmaeili, Mohammad  
Mechanisms of disease  

Estafanos, Lana  
Mechanisms of disease  

Fang, Jay  
Mechanisms of disease  

Farahani, Mojgan  
Advancing health services provision and health policy  

Fenech, Melissa  
Mechanisms of disease  

Fried, Rebecca  
Prevention of diseases and health conditions and promotion of well-being  

Fritz, Jamie  
Mechanisms of disease  

Fuhrmann, Benjamin  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Fulcher, Niveen  
Mechanisms of disease  

Gheisarzadeh, Nima  
Advancing health services provision and health policy  

Gillies, Derek  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Gomes, Janice  
Detection, screening and diagnosis of health and disease  

Greff, Michael  
Prevention of diseases and health conditions and promotion of well-being  

Groves, Leah  
Early life programming and development  

Guitar, Nicole  
Prevention of diseases and health conditions and promotion of well-being  

Gulilat, Markus  
Advancing health services provision and health policy  

Hamilton, Amanda  
Detection, screening and diagnosis of health and disease  

Harerimana, Boniface  
Advancing health services provision and health policy  

Harris, Andrew  
Detection, screening and diagnosis of health and disease  

Hayes, Kaitlyn  
Advancing health services provision and health policy  

Hetou, Khalil  
Detection, screening and diagnosis of health and disease  

Hintermayer, Matthew  
Mechanisms of disease  

Ho, Rosettia  
Mechanisms of disease  

Hogan-Cann, Aja  
Mechanisms of disease  

Holland, Sara  
Prevention of diseases and health conditions and promotion of well-being  

Hong, Michael  
Advancing health services provision and health policy  

Horst, Becky  
Prevention of diseases and health conditions and promotion of well-being  

Hughes, James  
Detection, screening and diagnosis of health and disease  

Hussain, Uzair  
Determinants of health  

Ioussoufovitch, Seva  
Detection, screening and diagnosis of health and disease  

Ivanova, Nadezda  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Janes, Ian  
Population, public health and education  

Jarvis, Sommer  
Early life programming and development  

Kai, Jason  
Detection, screening and diagnosis of health and disease  

Kambli, Ankita  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  

Karunatilleke, Nadun  
Mechanisms of disease  

Kennedy, Erin  
Population, public health and education  

Khalil, Andrew  
Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)
Kim, Yae Ram  Mechanisms of disease  113
Klassen, Stephen  Determinants of health  75
Kljakic, Ornela  Mechanisms of disease  114
Knapp, Jason  Mechanisms of disease  115
Knowles, Nikolas  Detection, screening and diagnosis of health and disease  53
Kozlov, Alexandra  Early life programming and development  85
Krishnan, Rohin  Population, public health and education  156
Kuiack, Robert  Mechanisms of disease  116
Kukan, Sahana  Prevention of diseases and health conditions and promotion of well-being  170
Kusins, Jonathan  Mechanisms of disease  117
Lant, Jeremy  Mechanisms of disease  118
Laramee, Sophie  Determinants of health  76
Laski, Jeremi  Mechanisms of disease  119
Lau, Esther  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  13
Lee, Ji Yun  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  14
Lee, Wai-Dun  Prevention of diseases and health conditions and promotion of well-being  171
Levit, Alexander  Mechanisms of disease  120
Li, Xinyi  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  15
Lim, Yong  Detection, screening and diagnosis of health and disease  54
Luo, Johnny  Mechanisms of disease  121
Mahmoudian, Borna  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  16
Meadows, Adam  Mechanisms of disease  122
Michaelov, Evan  Population, public health and education  157
Modarresi, Shirin  Prevention of diseases and health conditions and promotion of well-being  172
Moir, M. Erin  Determinants of health  77
Morissette, Martin Pascal  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  17
Morrow, Nadya  Prevention of diseases and health conditions and promotion of well-being  173
Murphy, Patrick  Detection, screening and diagnosis of health and disease  55
Murray, Patrick  Detection, screening and diagnosis of health and disease  56
Murphy, Patrick  Population, public health and education  158
Murphy, Patrick  Detection, screening and diagnosis of health and disease  55
Murray, Patrick  Detection, screening and diagnosis of health and disease  56
Murphy, Patrick  Population, public health and education  158
Nandi, Pinki  Early life programming and development  86
Ngo, Vy  Mechanisms of disease  123
Ni, Rui  Mechanisms of disease  124
Nixon, Kevin  Mechanisms of disease  125
Nouri Nejad, Daniel  Mechanisms of disease  126
Novello, Matthew  Mechanisms of disease  127
Orlando, Nathan  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  19
Padda, Ranjit Singh  Mechanisms of disease  128
Park, Patrick  Detection, screening and diagnosis of health and disease  56
Parkins, Katie  Mechanisms of disease  129
Patel, Darshit  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  20
Pavlosky, Alexander  Detection, screening and diagnosis of health and disease  57
Pawlak, Emily  Mechanisms of disease  130
Polch, Ana-Bianca  Detection, screening and diagnosis of health and disease  58
Press, Eric  Mechanisms of disease  131
Puka, Kladji  Determinants of health  78
Qin, Wen  Detection, screening and diagnosis of health and disease  59
Radford, Bethany  Early life programming and development  87
Rajaram, Ajay  Detection, screening and diagnosis of health and disease  60
Rampersaud, Amanda  Mechanisms of disease  132
Razzaq, Abdul  Mechanisms of disease  133
Rejali, Hossein  Detection, screening and diagnosis of health and disease  61
Richards, Dillon  Detection, screening and diagnosis of health and disease  62
Riddle, Michael  Advancing health services provision and health policy  37
Roy, Alexander C.  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  21
Saha, Bibek  Early life programming and development  88
Salloum, Noor  Mechanisms of disease  134
Samotus, Olivia  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  22
Sanchez, Rafael  Mechanisms of disease  135
Schroeder, Peyton  Population, public health and education  159
Schulz, Jenna  Mechanisms of disease  136
Scott, Kaela  Mechanisms of disease  137
Selvan, Kavin  Detection, screening and diagnosis of health and disease  63
Shannon, Blake  Mechanisms of disease  138
Sheldrake, Elena  Prevention of diseases and health conditions and promotion of well-being  174
Sherazi, Ali  Mechanisms of disease  139
Sivagurunathan, Marudan  Detection, screening and diagnosis of health and disease  64
Smith, Corey  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  23
Snyder, Jessica  Mechanisms of disease  140
Song, Zheng  Mechanisms of disease  141
Sové, Richard  Determinants of health  79
Stewart, Chloe  Population, public health and education  160
Surmanski, Anette  Mechanisms of disease  142
Szabla, Robert  Prevention of diseases and health conditions and promotion of well-being  175
Szpalko, Alex  Early life programming and development  89
Tanashi, Ahmed  Detection, screening and diagnosis of health and disease  65
Tauro, Michael  Mechanisms of disease  143
Tellios, Vasiliki  Mechanisms of disease  144
Thavam, Sinthiya  Population, public health and education  161
Thiyagarajah, Mathura  Mechanisms of disease  145
Tong, Olivia  Detection, screening and diagnosis of health and disease  66
Tonial, Nicholas  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  24
Tran, An  Mechanisms of disease  146
Trelford, Charles  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  25
Turk, Matthew  Detection, screening and diagnosis of health and disease  67
Vickress, Jason  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  26
Vijeyakumaran, Meera  Mechanisms of disease  147
Wang, Hui  Detection, screening and diagnosis of health and disease  68
Wang, TianDuo  Detection, screening and diagnosis of health and disease  69
Wang, Yuning  Mechanisms of disease  148
Wong, Annette  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  27
Wright, David  Mechanisms of disease  149
Yang, Dae-Myoung (Danny)  Detection, screening and diagnosis of health and disease  70
Yeung, Spencer  Mechanisms of disease  150
Zhu, Cuilin  Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)  28
Zmavc, Katrina  Mechanisms of disease  151
## APPENDIX B: PLATFORM PRESENTATIONS OVERVIEW

### MORNING SESSION

<table>
<thead>
<tr>
<th>Name</th>
<th>Research Area</th>
<th>Salon</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baines, Kelly</td>
<td>Early life programming and development</td>
<td>D</td>
</tr>
<tr>
<td>Batista, Carolina</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
<tr>
<td>Deveau, Victoria</td>
<td>Early life programming and development</td>
<td>B</td>
</tr>
<tr>
<td>Fanous, Jacob</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
<tr>
<td>Faria, Frederico</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>E</td>
</tr>
<tr>
<td>Han, Tim Tian</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>E</td>
</tr>
<tr>
<td>Hawley, Zachary</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
<tr>
<td>Hunt, Nina</td>
<td>Mechanisms of disease</td>
<td>D</td>
</tr>
<tr>
<td>Irwin, Bridget</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>E</td>
</tr>
<tr>
<td>Jayawardena, Devika</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
<tr>
<td>Kuljanin, Miljan</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>E</td>
</tr>
<tr>
<td>Kum, Jina</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
<tr>
<td>Li, Zhe</td>
<td>Advancing health services provision and health policy</td>
<td>D</td>
</tr>
<tr>
<td>Maitland, Matthew</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
<tr>
<td>Naidu, Anish</td>
<td>Prevention of diseases and health conditions and promotion of well-being</td>
<td>B</td>
</tr>
<tr>
<td>Parham, Kate</td>
<td>Mechanisms of disease</td>
<td>D</td>
</tr>
<tr>
<td>Ruicci, Kara</td>
<td>Mechanisms of disease</td>
<td>E</td>
</tr>
<tr>
<td>Shin, Alice</td>
<td>Mechanisms of disease</td>
<td>D</td>
</tr>
<tr>
<td>Thorburn, Victoria</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
</tbody>
</table>

### AFTERNOON SESSION

<table>
<thead>
<tr>
<th>Name</th>
<th>Research Area</th>
<th>Salon</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benoit, Simon</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
<tr>
<td>Chen, Jennifer</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>D</td>
</tr>
<tr>
<td>Dammak, Salma</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>E</td>
</tr>
<tr>
<td>Dilliott, Allison</td>
<td>Mechanisms of disease</td>
<td>D</td>
</tr>
<tr>
<td>Dron, Jacqueline</td>
<td>Mechanisms of disease</td>
<td>E</td>
</tr>
<tr>
<td>Gugusteoa, Radu</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
<tr>
<td>Haddara, Mohammad</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>D</td>
</tr>
<tr>
<td>Jain, Rajiv</td>
<td>Mechanisms of disease</td>
<td>E</td>
</tr>
<tr>
<td>Johnson, Patricia</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>B1</td>
</tr>
<tr>
<td>McIntyre, Amanda</td>
<td>Advancing health services provision and health policy</td>
<td>B1</td>
</tr>
<tr>
<td>McRae, Samantha</td>
<td>Advancing health services provision and health policy</td>
<td>B</td>
</tr>
<tr>
<td>Milej, Daniel</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>E</td>
</tr>
<tr>
<td>Mirshafiei, Fatemeh</td>
<td>Mechanisms of disease</td>
<td>E</td>
</tr>
<tr>
<td>Nicholson, Andrew</td>
<td>Detection, screening and diagnosis of health and disease</td>
<td>D</td>
</tr>
<tr>
<td>Rodgers, Jessica</td>
<td>Advances in structural and physiological treatment of disease and therapeutic intervention (includes surgery and drugs)</td>
<td>D</td>
</tr>
<tr>
<td>Rudak, Patrick</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
<tr>
<td>Shahid, Marwan</td>
<td>Determinants of health</td>
<td>B</td>
</tr>
<tr>
<td>Shum, Michelle</td>
<td>Mechanisms of disease</td>
<td>B1</td>
</tr>
<tr>
<td>Somé, Nibene</td>
<td>Advancing health services provision and health policy</td>
<td>B1</td>
</tr>
<tr>
<td>Yin, Charles</td>
<td>Mechanisms of disease</td>
<td>B</td>
</tr>
</tbody>
</table>
London Health Research Day is presented in partnership by Lawson Health Research Institute and the Schulich School of Medicine & Dentistry. This unique research event showcases the outstanding research by top graduate trainees and postdoctoral scholars from across the city of London.

OUR PARTNERS INCLUDE

LUNCH AND RECEPTION SPONSOR

Trudell Medical International

TABLE SPONSORS