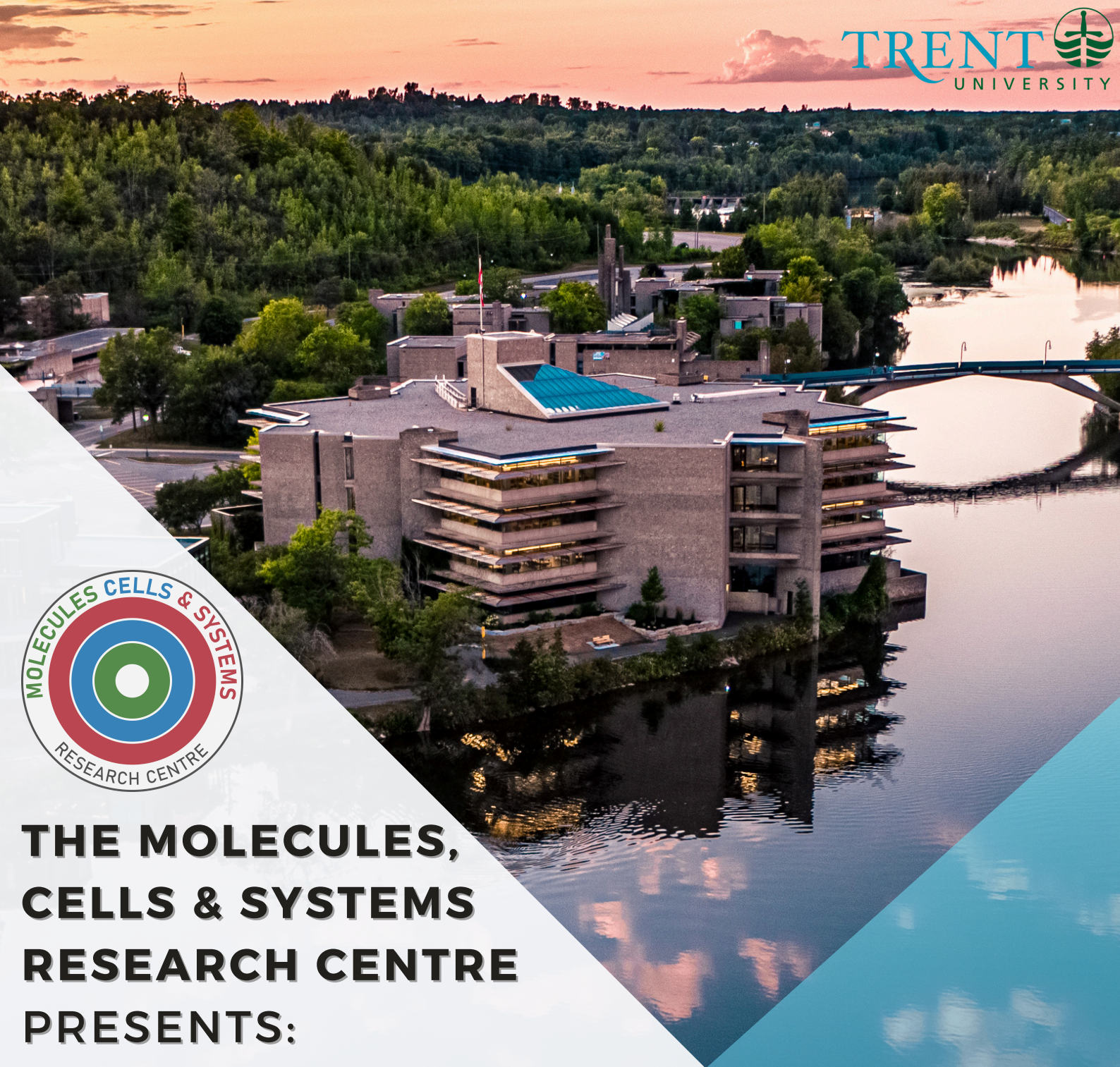

THE MOLECULES, CELLS & SYSTEMS RESEARCH CENTRE PRESENTS:

4th Annual Research Symposium

PROGRAM

APRIL • 24 • 2026

TRENT UNIVERSITY
THE STUDENT CENTRE: STOHN HALL (TSC 1.22)

PETERBOROUGH, ON

MCS OVERVIEW

THE MOLECULES TO SYSTEMS APPROACH

The life sciences are a broad grouping of disciplines that study organisms across the tree of life. All organisms, whether unicellular (e.g., bacteria) or multicellular (e.g., plants, animals; including humans), require fundamental molecular building blocks. These building blocks facilitate the formation of a cell, which associates with other cells to form systems within a multicellular organism. Through evolution, organisms have become more complex, facilitated by the formation of specialized interdependent molecular and cellular systems. The Molecules to Systems Approach seeks to understand the mechanisms underlying each stage of this biological hierarchy.

OUR VISION

Trent University has a wealth of expertise in various disciplines across the life sciences including molecular biology, cell biology, biochemistry, microbiology, developmental biology, physiology, kinesiology, psychology, and neuroscience. To exploit this competitive advantage, the Molecules, Cells & Systems Research Centre brings together researchers from the Departments of Biology, Chemistry, Forensic Science, Kinesiology, and Psychology to collaborate and apply the Molecules to Systems Approach in their research.

ORGANIZING COMMITTEE

Dr. Robert Huber, Dr. Stephanie Tobin, Josephine Esposto, Sean Condie, Jordan Webb

ACKNOWLEDGEMENTS

Session Moderators:

Dr. Cayleih Robertson, Dr. Neil Emery, Dr. Jenifer Hendel, Dr. Neil Fournier

Presentation Evaluators:

Mark Seegobin, Katie Horlock-Roberts, Samantha Logan

Volunteers:

Blythe Ferguson, Alexandria Northey, Samer Owiar, Galair Prevost

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GSA

REGIONAL SCIENTIFIC OUTREACH & COMMUNITY ENGAGEMENT

PETERBOROUGH HIGH SCHOOL STUDENT REPRESENTATIVES

This year, the MCS Management Committee is thrilled to announce the inclusion of local Peterborough Student Representatives from each of the major secondary schools in the Peterborough community. This initiative highlights a commitment to scientific outreach and mentorship within the Kawarthas, where the students have been invited to observe and participate in the Symposium's sessions to gain firsthand experience in the academic exchange of ideas. We encourage all attendees to welcome these representatives and share insights into the diverse career paths and research opportunities available within the field. We hope that by inviting these bright minds, we can help inspire a lifelong passion for discovery and support the next generation of scientific leaders in our region.

PETERBOROUGH REGIONAL SCIENCE FAIR (PRSF) AWARDEES

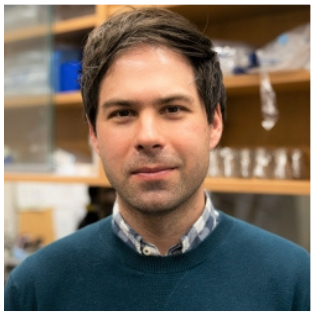
This year marks a significant milestone in our community outreach as the MCS Symposium proudly introduces **two inaugural awards** for the **Peterborough Regional Science Fair (PRSF)**. These awards, which will be presented annually, represent our long-term commitment to recognizing and supporting exceptional scientific talent at the secondary school level. By sponsoring these honours, we aim to provide local students with both the resources and the encouragement to pursue advanced studies in the molecular and cellular sciences.

Molecules, Cells & Systems Research Centre Certificate of Recognition for Best Project in the Life & Health Sciences

We are proud to recognize the award-winning students of the Peterborough Regional Science Fair in attendance today. Their projects represent the pinnacle of junior scientific inquiry in our region, demonstrating a level of technical skill and creative problem-solving that is truly commendable. By showcasing these awardees alongside our symposium presenters, we celebrate their early contributions to the scientific community and their potential to shape the future of the field!

Peterborough Regional Science Fair
PRSF
Established 1970
Peterborough Regional Science Fair
Ontario, Canada

MCS MANAGEMENT COMMITTEE



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M.Sc. Student (Psychology)
Trent University

SCHEDULE

TIME	EVENT
8:30 AM – 9:00 AM	**Registration -** *The Student Centre (TSC) Corridor (outside of TSC 1.07)*
9:00 AM - 9:05 AM	**Opening Remarks (Stohn Hall, TSC 1.22)** ***Dr. Robert Huber and Dr. Stephanie Tobin***
9:05 AM – 10:15 AM	**Session 1: Trainee Presentations (Stohn Hall, TSC 1.22)** ***Moderator: Dr. Cayleih Robertson*** 1. **Sarah Hill -** *Nitric oxide dioxygenase activity of* Giardia intestinalis *flavohemoglobins* 2. **Hannah Smith -** *Characterizing the role of tRNA isopentenyltransferase I in skeletal muscle* 3. **Brenden O'Brien -** *A single ethologically relevant stressor produces enduring sensitivity in a post-injury state* 4. **Jordan Webb -** *Evidence of allocentric spatial learning in male rats with large lesions of the hippocampus* 5. **Gillian Ekins -** *Mechanisms of weight gain and the effects of semaglutide in a female rodent model of epilepsy*
10:15 AM – 10:45 AM	*Coffee/Tea Break - TSC Atrium* ***PRSF Awardee Exhibits***
10:45 AM – 12:00 PM	**Session 2: Trainee Presentations (Stohn Hall, TSC 1.22)** ***Moderator: Dr. Neil Emery*** 1. **Lorna Phan -** *Stress, rest, and repeat until you die: The reality of skeletal muscle function* 2. **Adrian Guaman Vargas -** *The fungal eye: Interactions between photoreceptors and cytokinins that shape fungal morphology and physiology* 3. **Shelby Bryan -** *The interaction of the TATA-binding protein with DNA G-quadruplexes in a protozoan parasite* 4. **Nathan Kritzer -** *Cold-stressed-induced inflammation as a mechanism in brown adipose tissue to combat obesity development in UCP-1 KO mice* 5. **Zaryna Leo -** *ComBATing the cold: Understanding the promotion and inhibition of brown adipose tissue*
12:00 PM – 1:00 PM	*Lunch Break - TSC 1.07*

SCHEDULE

TIME	EVENT
1:00 PM – 1:45 PM	**KEYNOTE SPEAKER** *Location: Stohn Hall, TSC 1.22* **Dr. Maithe Arruda-Carvalho**, Associate Professor (Psychology) University of Toronto Scarborough *Title: Neural Signatures of Fear Memory Processing Change Across the Lifespan*
1:45 PM – 2:30 PM	**Session 3: Trainee Presentations (Stohn Hall, TSC 1.22)** ***Moderator: Dr. Jenifer Hendel*** 1. **Stephanie Sissons -** *Reactivating memory engrams to reverse impaired fear memory recall after chronic seizures* 2. **Melanie Marlow -** *The unusual properties of the TBP transcription factor in the protozoan parasite,* Giardia Intestinalis 3. **Aditi Midha -** *Detection of mitochondrial tRNAs in skeletal muscle using modified Northern Blotting* **Concurrent Session:** **Peterborough High School Student Representatives + Peterborough Regional Science Fair Winners tour the MCS Faculty labs on campus:** **1:45 PM - 2:30 PM**
2:30 PM – 2:45 PM	*Coffee/Tea Break - TSC Atrium* ***PRSF Awardee Exhibits***
2:45 PM – 3:30 PM	**KEYNOTE SPEAKER** *Location: Stohn Hall, TSC 1.22* **Dr. Mark Bayfield**, Professor (Biology) York University *Title: A LARPing we will go! Recent insights from RNA's most versatile role players*
3:30 PM – 4:15 PM	**Session 4: Trainee Presentations (Stohn Hall, TSC 1.22)** ***Moderator: Dr. Neil Fournier*** 1. **Maxwell McPherson -** *Cell profiling in an energy deficit: The immune response to physiological and pathological stress* 2. **Linh Tran -** *Leaf me alone: Exploring how a plant hormone may prevent the transmission of* Giardiasis 3. **Sean Condie -** *Loss of sortilin slows proliferation and development in* Dictyostelium discoideum
4:30 PM	**Presentation Awards & Closing Remarks (Stohn Hall, TSC 1.22)**

Dr. Maithe Arruda-Carvalho
Associate Professor (Psychology), University of Toronto Scarborough

Neural Signatures of Fear Memory Processing Change Across the Lifespan

The medial prefrontal cortex (mPFC) is critical to cognitive and emotional function and underlies many neuropsychiatric disorders, including fear and anxiety disorders. Childhood and adolescence are the predominant age of onset for the majority of mental disorders, and coincide with anatomical and morphological changes within mPFC pathways. Yet, how such changes influence mPFC function and consequently affect behaviour and the onset of mental illness is currently unknown. Using a combination of viral tracing, optogenetic-assisted patch clamping and chemo and optogenetic manipulations during behaviour, we examined how mPFC subregions are differentially engaged in fear processing across ages, with a focus on the timing of mPFC recruitment and contribution to fear encoding, extinction and remote retrieval from infancy to adulthood in mice. Our data show that mPFC subprojections to the amygdala become necessary for fear encoding and extinction during adolescence and juvenility, respectively, with important consequences for fear expression. We also show important contributions of mPFC input projections to the regulation of remote memory retrieval in adult and infant mice. Our data highlight age-, sex- and pathway-specific contributions within mPFC pathways to fear processing, with crucial implications for early life influences on adult cognitive function.

ABSTRACTS

Dr. Mark Bayfield
Professor (Biology), York University

Originally from Barrie, I am a Professor in the Department of Biology at York University where I study RNA binding proteins and teach biochemistry and molecular biology. Previously I was a post-doctoral fellow at the National Institutes of Health (NIH), and I performed my doctorate at Brown University and my B.Sc. at McGill University. Our lab trains young scientists with the support of operating grants from CIHR and NSERC. I serve on the executive of the La and La-related protein society, the scientific advisory board of Riboclub and as Scientific Officer and panel review member for CIHR. Previously Dean's Special Advisor for development of Faculty of Science curriculum and initiatives at York University's Markham Campus. I'm also the Chair of Outreach, RNA Canada ARN & Co-lead of the Banting Research Foundation Mentorship initiative.

A LARPing we will go! Recent insights from RNA's most versatile role players

The La and La-related proteins (LARPs) are a superfamily of eukaryotic RNA binding proteins with a wide diversity of functions and broad links to human health. Some LARPs are nuclear have been linked to pre-tRNA processing, RNA chaperone function and the formation of stable ribonucleoprotein complexes, while other are cytoplasmic and have been linked to mRNA translation. Our lab studies a number of La and La-related proteins in fission yeast, ciliates and human cells and have recently expanded the repertoire of functions related to these factors, including telomerase function, mRNA splicing, snRNA & tRNA modification and mRNA stability. I will provide a survey of the functions linked to this essential class of factors as well as a summary of recent work performed in our group.

ABSTRACTS

Trainees

Sarah Hill, Dr. Steven Rafferty

Nitric oxide dioxygenase activity of Giardia intestinalis flavohemoglobins

The parasitic protist Giardia intestinalis possesses flavohemoglobin (gFlHb), an enzyme with nitric oxide dioxygenase (NOD) activity that oxidizes nitric oxide (NO) to nitrate (NO3-). Of the eight G. intestinalis assemblages (A-H), only those that infect humans (A, B, E) encode gFlHb. Research on gFlHb as a potential drug target has focused on gFlHb-A, despite sequence differences of 13-29% relative to gFlHb-B and E. To determine if there were enzymatic differences among the gFlHb variants, all three were recombinantly expressed and their NOD activities were measured using a free radical analyzer equipped with a NO-sensitive electrode. Rates of NOD activity were comparable among variants (kcat: 35-62 s-1), as were their sensitivities towards the substrates NO (Km: 0.62-0.89 μM) and NADH (Km: 15-19 μM). Inhibition of NOD activity by miconazole (Ki: 5-13 μM) and cyanide (Ki: 1-5 μM) were comparable among all variants. However, nitrite (NO2-) a moderate inhibitor of gFlHb-A and E (Ki: 27 and 61 μM respectively), was a weak inhibitor for gFlHb-B (Ki could not be determined accurately); interestingly optical titrations show that NO2- binding affinity to ferric gFlHb was similar for all three variants (KD: 275-343 μM). Structural comparisons revealed residue differences in substrate entry and product exit pathways connecting the active site. Therefore, the NOD rate parameters of gFlHbs are similar, but their different sensitivities towards NO2- signify differences in substrate access and product egress that need to be considered if this enzyme is to be examined as a drug target.

Lorna N. Phan, Dr. Stephanie Tobin

Stress, rest, and repeat until you die: The reality of skeletal muscle function

Skeletal muscle is one of the most abundant organs in the mammalian body that allows for voluntary control essential for movement, posture, breathing, and temperature regulation. They are also highly adaptable as they can regenerate, repair, and remodel in the face of injury, a process termed myogenesis. However, these abilities can decline with chronic inflammation and mitochondrial dysregulation, which is implicated in chronic disease, immobilization, and aging. Inflammation is a typical response to tissue injury which accompanies the destruction and reconstruction of muscle tissues. The immune system recognizes molecules from damaged tissues and initiates activity of inflammatory cytokines to stimulate and promote muscle repair. The mitochondria are responsible for regulating metabolic status by adjusting their volume, structure and function in response to stress in skeletal muscles. Additionally, mitochondria produce the primary form of energy in organisms, adenosine triphosphate (ATP), to drive and meet the demand requirements of skeletal muscle. The objective of this study is to expand the knowledge of inflammation and mitochondrial activity by investigating the mechanisms of interleukin-16 (IL-16), tetraspanin CD9, and tRNA isopentenyltransferase 1 (TRIT1) in skeletal muscle. Experimental outcomes have been obtained using an in vitro C2C12 myoblast cell line, and in vivo mouse cardiotoxin-induced injury model, both of which are commonly used to study myogenesis. Further knowledge of inflammation and mitochondrial activity can improve predictions of disease progression in patients. It can also support the development of therapeutics to enhance muscle repair and help slow or reduce the effects associated with disease, immobilization, and aging.

ABSTRACTS
Trainees

Sean Condie, Dr. Robert Huber

Loss of sortilin slows proliferation and development in Dictyostelium discoideum

Sortilin (SORT1) is a protein trafficking receptor that packages proteins in the Golgi apparatus and delivers those proteins to various destinations within the cell. Research suggests that SORT1 traffics proteins to endosomes and may facilitate the re-capture of secreted proteins at the cell surface. However, the full extent of sortilin-mediated trafficking in the cell is not known. Here, we used the model organism Dictyostelium discoideum to gain insight into the evolutionarily conserved functions of SORT1. Bioinformatic tools confirmed the homology between human SORT1 and Dictyostelium Sort1 and protein docking software identified potential binding partners of both proteins. Loss of sort1 in Dictyostelium slows proliferation and reduces macropinocytosis. During the early stages of multicellular development, sort1-deficiency delays aggregation and appears to affect cell-to-cell adhesion. To localize Sort1, we expressed Sort1 with a C-terminal GFP tag in sort1- cells (sort1-:Sort1-GFP). As expected, we observed that Sort1-GFP localizes to the Golgi apparatus in growth-phase cells. However, in starved cells, the fusion protein localizes to the cell surface suggesting that Sort1 traffics signaling molecules to the cell surface for secretion and/or participates in the re-capture of secreted proteins. To assess this, conditioned buffer was concentrated and analyzed for the density-sensing molecule, countin. Initial results show deferential levels between WT and sort1- cells. Our current work is examining the broader role of Sort1 on intracellular protein trafficking and the link between the aberrant secretion of countin and delayed aggregation.

Maxwell McPherson, Dr. Cayleih Robertson, Dr. Stephanie Tobin

Cell Profiling in an Energy Deficit: The Immune Response to Physiological and Pathological Stress

Within metabolism there are two main pathways; anabolism and catabolism, both essential for maintaining an energy balance. An energy balance is when caloric intake is equivalent to the caloric output, however, an imbalance can occur due to various stressors. When energy expenditure is much higher than energy intake, an energy deficit occurs where two major tissues are affected, adipose tissue and skeletal muscle. Adipose tissue is highly dynamic and plays a major role in energy storage. Inversely, skeletal muscle is one of the largest energy consumers and is responsible for voluntary movements. Both these tissues play crucial roles in maintaining an energy balance and adapting to periods of energy imbalance. Physiological and pathological mouse models are used to measure the immune responses within adipose and muscle tissue. A cold exposure (physiological) model is used to examine the adaptive response since mice are able to comfortably increase lipolysis and direct fatty acids to brown fat and skeletal muscle for thermogenesis. A cachexia (pathological) model is simulated using monocrotaline (MCT) injections, where adipose lipolysis and skeletal muscle proteolysis heavily contribute to tissue wasting. Both tissues have a large population of immune cells within their microenvironment which regulate their activity and response to various stress factors. Using flow cytometry to analyze immune populations will provide a greater understanding as to which immune cells are responsible for the immune response. This will provide a strong foundation to how immune populations shift with stress.

ABSTRACTS
Trainees

Gillian Ekins, Dr. Neil Fournier

Mechanisms of Weight Gain and the Effects of Semaglutide in a Female Rodent Model of Epilepsy

Epilepsy is a neurological disorder characterized by recurrent seizures which is often accompanied by behavioural and physiological comorbidities, including obesity. Similar weight gain has been observed in rodent models of epilepsy, such as electrical kindling. Across two studies in female rats, kindling was associated with increased food intake, elevated blood glucose levels, and significant weight gain despite normal glucose tolerance, suggesting metabolic disruption independent of classical insulin resistance. A replication study (n = 34) confirmed these findings and further demonstrated that semaglutide, a GLP-1 receptor agonist, attenuated weight gain and improved metabolic outcomes in fully kindled animals. Semaglutide treatment also produced trends toward reduced anxiety-like behaviours in some behavioural assays. Together, these findings indicate that chronic seizure-related neural alterations may disrupt systemic metabolic regulation and that GLP-1–based therapies may offer a promising approach for managing epilepsy-associated metabolic comorbidities. The current study extends this work by examining whether these metabolic effects replicate in male Long–Evans rats (n = 35) to assess potential sex differences. Additionally, we investigate whether earlier semaglutide intervention, initiated after 30 stimulations, can modify the metabolic trajectory of kindled animals. Understanding the interaction between seizure activity and metabolic regulation may inform strategies to improve long-term health outcomes in epilepsy.

Brendan O'Brien, Dr. Neil Fournier

A Single Ethologically Relevant Stressor Produces Enduring Sensitivity in a Post-Injury State

Past research demonstrates that stress can modulate pain processing, producing either analgesia (i.e., stress-mediated reductions in pain sensitivity) or hyperalgesia (i.e., stress-mediated enhancements in pain sensitivity) depending on contextual and temporal factors. Clinically, stress-related pain sensitization has most often been attributed to prolonged dysregulation of stress-responsive systems. However, emerging evidence suggests that sensitization may also develop rapidly following acute threat exposure under conditions of prior peripheral injury. Using a rodent model, we examined whether a single exposure to the predator odor 2,5-dihydro-2,4,5-trimethylthiazoline (TMT) interacts with prior inflammatory injury induced by Complete Freund's Adjuvant (CFA) to produce persistent mechanical hypersensitivity. Mechanical sensitivity was assessed longitudinally using von Frey testing. TMT exposure alone did not alter nociceptive thresholds, and CFA-induced hypersensitivity resolved within 7–10 days. In contrast, animals that experienced both injury and subsequent predator odor stress developed enduring mechanical hypersensitivity that persisted for months after peripheral inflammation had resolved. These findings demonstrate that acute stress can rapidly and robustly sensitize pain pathways following injury, suggesting that a single threat-relevant event during a vulnerable post-injury state may promote the transition from adaptive, injury-related nociception to persistent pathological sensitization.

ABSTRACTS
Trainees

Stephanie Sissons, Dr. Neil Fournier

Reactivating Memory Engrams to Reverse Impaired Fear Memory Recall After Chronic Seizures

Epilepsy is a chronic noncommunicable neurological disease characterized by recurrent unprovoked seizures and cognitive impairments, including memory deficits. Up to 50% of individuals with epilepsy experience persistent memory deficits, underscoring the need for targeted therapies. Memory formation relies on patterns of neuronal ensembles (engrams) activated during learning, and successful recall requires reactivation of these same cells. Pathological activity can disrupt engram circuit connectivity, potentially impairing reactivation during retrieval. However, whether disrupted engram reactivation directly contributes to epilepsy-related cognitive deficits remains unclear. To investigate seizure-related memory impairment, we used an activity-dependent neuronal tagging and chemogenetic strategy to selectively label and manipulate CA1 hippocampal engrams formed during contextual fear conditioning in male rats. Following encoding, animals underwent repeated pentylenetetrazole (PTZ)-induced seizures or vehicle treatment. To determine the optimal time point for capturing learning-related engrams, we quantified c-fos (an immediate-early gene commonly used as a marker of recent neuronal activity) expression in the hippocampus on learning days 2 and 3 and at a 1-week recall. PTZ-treated animals exhibited significant impairments in contextual fear recall. Importantly, chemogenetic reactivation of the originally captured engram partially rescued memory performance. These findings indicate that memory engrams may remain partially intact despite seizure-induced circuit remodelling yet fail to undergo natural reactivation. Together, our results suggest that epilepsy-related memory deficits may reflect impaired engram reactivation rather than permanent memory loss, highlighting the potential reversibility of seizure-associated cognitive dysfunctions.

Linh Tran, Dr. Neil Emery, Dr. Janet Yee

Leaf Me Alone: Exploring How a Plant Hormone May Prevent the Transmission of Giardiasis

Giardia intestinalis is a parasitic protist that infects human causing giardiasis, a common diarrheal infection worldwide. The infection initiates when a host ingests infectious cysts in contaminated food or water. Once inside, the cyst hatches in the stomach to release the trophozoites, which swim down and proliferate within the intestinal tract. In the small lower intestine, some trophozoites undergo encystation and develop back into cysts, which are released in the feces where they become a source of new infections. Encystation is the key process that allows the parasite to continue the transmission of the disease which can be induced in vitro. N6-benzyladenosine (BAR) is a type of adenine-derived signalling molecule, also known as cytokinin, that is rarely found in nature. This research demonstrates how the spiking of BAR into the encystation medium inhibits the cyst formation and disrupts the intracellular metabolite dynamics. Furthermore, it suggests that BAR may be targeting the encystation process specifically. These findings expand the current knowledge of the relationship between cytokinin and Giardia, while revealing BAR's potential beyond its role of being a plant hormone.

ABSTRACTS
Trainees

Nathan Kritzer, Dr. Cayleih Robertson

Cold-Stressed-Induced Inflammation as a Mechanism in Brown Adipose Tissue to Combat Obesity Development in UCP-1 KO Mice

Brown Adipose Tissue (BAT) is an important tissue that regulates thermogenesis and energy metabolism, via UCP-1, in mammals during early development. Previous BAT studies have used UCP-1 knockout (KO) mice models to determine the effect of UCP-1 on obesity development by feeding these mice high fat diets (HFD). Although these KO mice should gain weight and develop obesity faster than control mice, this is not always observed as sometimes KO mice develop obesity slower than the control mice. Past research suggests that cold stress and the subsequently induced inflammatory response may be associated with this 'anti-obesity' effect observed in these KO mice. We hypothesize that inflammation is a factor causing the resistance to obesity in the cold-treated KO mice. We predict that higher levels of inflammation via collagen deposition will be seen in the cold-treated KO mice in comparison to warm-treated control and KO mice, as well as in comparison to cold control mice. The mice were raised at thermoneutral temperatures (30°C) or at cold temperatures (18°C) during puberty and fed a HFD after reacclimating to thermoneutral temperatures. Bat samples were obtained from these mice at different timepoints and stained using a Picrosirius Red stain for collagen detection. Each tissue sample was imaged under microscope and analyzed using ImageJ for relative abundance of collagen. The results of this experiment will confirm the presence of inflammation from cold stress and whether it is a possible factor causing the observed 'anti-obesity' effect observed.

Melanie Marlow, Dr. Janet Yee

The Unusual Properties of the TBP Transcription Factor in the Protozoan Parasite, Giardia Intestinalis

Giardia intestinalis infects humans and over 40 animal species. Giardia has a compact (~12 Mb) genome containing a reduced complement of genes encoding proteins for essential cellular processes including transcription. The TATA-box binding protein (TBP) is a universal eukaryotic transcription factor but Giardia TBP (gTBP) is highly divergent, featuring substitutions at residues critical for binding to double-stranded DNA and a markedly narrowed DNA-binding pocket. We aimed to characterize the DNA-binding properties of gTBP and the structural properties of the DNA it binds to. Recombinant gTBP was used in electrophoretic mobility shift assays (EMSAs) to study its interaction with DNA. G-quadruplex formation in DNA was analyzed by circular dichroism spectroscopy and thioflavin T fluorescence assays. We determined that gTBP binds single-stranded DNA in two distinct modes. In A mode, gTBP multimers bind to ssDNA sequences with four or more consecutive guanine bases that can form secondary structures called G-quadruplexes, which have roles in gene regulation, genome stability, and telomere function in other eukaryotes. In contrast, B mode involves monomeric gTBP binding to more flexible DNA regions, measured by low base stacking energies. A search of the Giardia genome showed that regions of low base stacking energies are prevalent in the promoters of genes encoding proteins, tRNAs, and rRNAs, suggesting that this may be an important feature recognized by gTBP for gene regulation. These findings offer a novel perspective on eukaryotic transcription regulation, highlighting an unconventional interaction between gTBP and ssDNA including between gTBP and G4s.

ABSTRACTS
Trainees

Jordan Webb, Hugo Lehmann

Evidence of allocentric spatial learning in male rats with large lesions of the hippocampus

The hippocampus (HPC) is the neural substrate of allocentric spatial representation–viewpoint-invariant cognitive maps. HPC lesions disrupt performance on allocentric tasks such as the Morris Water Task (MWT), in which rodents learn the location of a platform submerged within a circular pool. Navigation from varied start points requires integrating multiple information sources, including allocentric representations of the environmental cue configuration. Rats with HPC lesions, however, may learn to navigate by using a less effective alternative strategy; a body-centered (egocentric) cue-based search strategy. Here, we investigated whether HPC lesion size predicts the shift in reliance from egocentric to allocentric strategy use in the MWT. Control and HPC groups showed evidence of learning, with the control rats adopting an allocentric strategy after a few trials. The HPC rats, in contrast, showed more heterogeneous strategy use with 19% adopting a consistent egocentric strategy and notably, 15% adopting a consistent allocentric strategy. Performance did not correlate with lesion size. These findings suggest that rats with large HPC lesions acquire and use allocentric spatial information in the MWT, highlighting the potential contributions of other brain regions to spatial learning and memory.

Adrian Guaman Vargas, Dr. Neil Emery

The Fungal Eye: Interactions Between Photoreceptors and Cytokinins That Shape Fungal Morphology and Physiology

Fungi perceive environmental light through conserved photoreceptor systems collectively described as the fungal “eye,” yet how light signalling integrates with endogenous hormonal regulation remains largely unresolved. While fungal photoreceptors have been extensively characterized at the molecular level, their downstream coordination with cytokinin metabolism has not been experimentally examined in a comparative, wavelength-specific framework. Concurrently, accumulating evidence demonstrates that fungi synthesize cytokinins—adenine-derived signalling molecules traditionally associated with plant development—suggesting a broader evolutionary role for hormone-mediated regulation across kingdoms. This study investigated whether wavelength-specific light exposure alters mycelial morphology and endogenous cytokinin abundance across phylogenetically diverse fungi, including Neurospora crassa, Chondrostereum purpureum, Tremella mesenterica, Monascus ruber, and Lepista nuda. Cultures were exposed to twelve discrete light treatments spanning ultraviolet, visible, infrared, and dark conditions. Colony development was quantified using high-resolution image-based morphometrics to measure radial expansion, perimeter complexity, circularity, branching architecture, and optical density. Mycelial fractions were harvested and analyzed using targeted UHPLC–HRMS/MS with isotope dilution to quantify free-base cytokinins and metabolites, including isopentenyladenine (iP), trans-zeatin (tZ), cis-zeatin (cZ), dihydrozeatin (DZ), and methylthiolated derivatives. Significant morphological responses were observed under red, green, pink, and ultraviolet light, with species-specific shifts in colony architecture and pigmentation patterns. Cytokinin profiles varied not only among species but also within species across light treatments, demonstrating wavelength-dependent reconfiguration of endogenous hormone pools. Notably, free-base cytokinin abundance shifted under specific spectral conditions independent of total biomass, supporting the hypothesis that cytokinins function as internal signaling intermediates linking photoreceptor activation to developmental regulation. By integrating quantitative morphometrics with targeted cytokinin profiling, this study provides experimental evidence that light quality restructures both fungal morphology and endogenous hormonal signaling, advancing a mechanistic framework for photoreceptor–cytokinin crosstalk in fungi with implications for ecological adaptation, cultivation optimization, and light-based disease management strategies.

ABSTRACTS
Trainees

Shelby Bryan, Melanie Marlow, Dr. Janet Yee

The interaction of the TATA-binding protein with DNA G-quadruplexes in a protozoan parasite

Giardia intestinalis is a waterborne protozoan parasite that causes a severe and infectious form of diarrhea in humans. Eukaryotic transcription initiation by RNA polymerase II involves an essential transcription factor called the TATA-binding protein (TBP) originally characterized by its ability to bind TATA-box sequences within promoter regions of double-stranded DNA (dsDNA). The Giardia homolog (gTBP) is highly divergent and lacks three of the four conserved phenylalanine residues key to the binding and unwinding of dsDNA. Previous work from our laboratory demonstrated that gTBP preferentially binds single-stranded DNA (ssDNA). This observation is supported by predictive structural modeling indicating that gTBP possesses a significantly narrower DNA-binding domain compared to other eukaryotic TBPs. Our lab has identified two distinct modes of gTBP binding to ssDNA, neither of which depends on AT-rich DNA. In the binding mode we named the “A mode,” gTBP recognizes ssDNA containing runs of four or more guanine nucleotides in row. Such guanine-rich sequences have the potential to form secondary DNA structures called G-quadruplexes (G4s), which have important roles in in gene regulation, DNA replication, telomere functions, and genome stability. My research aims to determine whether Giardia promoter sequences recognized by gTBP in the A-mode can adopt G4 conformations, using a fluorescence-based thioflavin T (ThT) assay. I will also assess whether gTBP can compete with ThT for binding to these G4 structures. Together, this work will clarify whether G4 formation contributes to promoter recognition in Giardia and other roles of gTBP in this parasitic protist.

Hannah Smith, Dr. Stephanie Tobin

Characterizing the role of tRNA isopentenyltransferase I in skeletal muscle

tRNA isopentenyltransferase I (TRIT1) catalyzes the isopentenylation of adenosine 37 in mitochondrial and cytosolic tRNA, stabilizing codon:anticodon basepairing and improving translation. Due to its important role in TRIT1 is extremely important for ATP production and deficiencies can result in mitochondrial dysfunction. Combined oxidative phosphorylation deficiency 35 is a pediatric illness resulting from TRIT1 deficiency that is characterized by global developmental delay, seizures, and musculoskeletal. Despite its critical role in the mitochondria, the function of TRIT1 in skeletal muscle remains understudied. To investigate this relationship, C2C12 myoblasts were used to assess the subcellular localization and protein levels of TRIT1 throughout myogenesis. Cells were used for immunofluorescence staining and collected for western blotting after 0 hours and 144 hours of differentiation. Immunofluorescence staining for TRIT1, mitochondria, and nuclei, demonstrated a predominantly nuclear localization at both timepoints, with an increase in cytoplasmic localization and nuclear puncta in differentiated cells. While the high level of nuclear localization was unexpected, this finding suggests an additional function of TRIT1 in the nucleus. Furthermore, western blotting indicated the presence of a TRIT1 isoform at ~43 kDa in addition to the expected band at 53 kDa, and differential enrichment of TRIT1 in nuclear and cytoplasmic fractions. The shift in localization and presence of isoforms indicates that TRIT1 may have multiple distinct functions throughout the cell and during myogenesis. Understanding the role of TRIT1 in skeletal muscle will help with the development of treatments for muscular illness and COXPD35, which ultimately improves the quality of life for those impacted.

ABSTRACTS
Trainees

Aditi Midha, Dr. Stephanie Tobin

Detection of Mitochondrial tRNAs in Skeletal Muscle Using Modified Northern Blotting

N6-isopentenyladenosine (i6A37) is a modified nucleotide in the anticodon loop of tRNA that facilitates the stability of codon-anticodon interaction after translation. The presence of this modified nucleotide in tRNAs reduces frame-shifting mutations, enhancing protein synthesis and accurate translation of the genetic code into protein. Mammalian TRIT1 catalyzes the formation of i6A37 on specific tRNAs in the cytoplasm and mitochondria, including mt-tRNATrp, mt-tRNATyr, mt-tRNASer (UCN), and mt-tRNAPhe. Cells that are TRIT1 deficient have a defined phenotype known as Combined Oxidative Phosphorylation Deficiency 35 (COXPD35) which is characterized by dysfunction of mitochondrial translation and respiratory chain function. As TRIT1 is necessary for normal mammalian translation, the complete loss of TRIT1 leads to embryonic death. In the Tobin lab, we tried to develop a hybridization-based Northern blotting method to detect mtRNAs directly in skeletal muscle from mice. We will primarily focus on the well-characterized mt-tRNATrp and mt-tRNAPhe (the two targets of mtTRIT1, an established mitochondrial tRNA isopentenyltransferase, to demonstrate transfer of isopentenylyl groups to tRNA). I extracted total RNA from skeletal muscle of mice and resolved it by denaturing urea PAGE, transferring the resolved material to nylon membranes, and probing the membranes with biotinylated oligonucleotides to identify and quantify mt-tRNATrp and mt-tRNAPhe levels in the isolated skeletal muscle. Building upon this detection method, plans are to develop and implement a PHAM (Positive Hybridization in the Absence of Modification) probe to directly assess i6A37 modification on the mt-tRNA.

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ComBATing the cold: Understanding the promotion and inhibition of brown adipose tissue

Brown adipose tissue (BAT) is a key component of non-shivering thermogenesis, the primary mechanism enabling small mammals to survive cold winters. When activated, BAT undergoes extensive remodeling, increasing mitochondrial density and vascularization to meet the energetic demands of heat production. Recent work has identified numerous genes upregulated during cold exposure, yet the transcriptomic processes governing BAT plasticity and the suppression of thermogenesis to conserve energy remain less understood. This study aims to clarify the gene expression patterns underlying this regulatory balance. Deer mice were housed at thermoneutrality for six weeks, with half subsequently transitioned to cold conditions for seven days. BAT gene expression was then quantified using RNA-seq and analyzed through bioinformatic pipelines. We predicted that cold-induced upregulated genes would promote BAT activation, whereas downregulated genes would function as inhibitors. Transcriptomic analysis revealed clear separation between cold-exposed and thermoneutral samples, with strong clustering by temperature in the PCA. Differential expression testing identified a distinct set of temperature-responsive genes, including both up- and down-regulated transcripts consistent with pathways known to influence BAT behavior. Volcano plots highlighted several genes with large fold-changes and strong statistical support. Together, these findings demonstrate that BAT remodeling is tightly associated with coordinated shifts in gene expression, offering new insight into the molecular mechanisms that balance thermogenic activation with metabolic conservation.

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