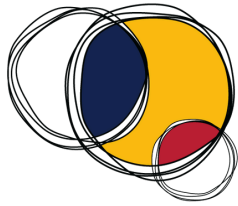


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Neural and Machine Systems
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Juniper
CAFE

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Schedule at a Glance

9:30am-10:00am	Attendee Check-In <i>New Medical Building</i>
10:00am-10:45am	Opening Remarks & Keynote Speaker (Dr. Caroline Pukall) <i>New Medical Building</i>
10:45am-11:00am	Morning Break <i>New Medical Building</i>
11:00am-12:00pm	Student Oral Presentation Session 1 <i>New Medical Building</i>
12:00pm-1:00pm	Lunch <i>New Medical Building</i>
1:00pm-2:00pm	Poster Session 1 (odd number posters) <i>Biosciences Hall Atrium</i>
2:15pm-3:15pm	Invited Speakers (Dr. Susan Boehnke & Dr. Linda Booij) <i>New Medical Building</i>
3:15pm-3:30pm	Afternoon Break <i>New Medical Building</i>
3:30pm-4:30pm	Poster Session 2 (even number posters) <i>Biosciences Hall Atrium</i>
4:45pm-5:45pm	Student Oral Presentations Session 2 <i>New Medical Building</i>
5:45pm-6:00pm	Awards and Closing Remarks <i>New Medical Building</i>

Student Oral Presentation Schedule

11:00am-12:00pm	STUDENT TALK SESSION 1
11:00am-11:11am	Altered social and depression-like behaviours support a two-hit model of stress in Long-Evans rats <i>Megan Babcock, MSc Candidate, Queen's University</i>
11:12am-11:23am	Investigating a novel treatment for spinal cord injury <i>Laura Camejo Espitia, MSc Candidate, University of Western Ontario</i>
11:24am-11:35am	Neurodevelopmental trajectory of anorexia nervosa: A longitudinal pan-Canadian multimodal imaging study <i>Rachel Dufour, PhD Candidate, Concordia University</i>
11:36am-11:47am	Identification of oculomotor abnormalities in behaviour variant frontotemporal dementia <i>Daria Hinton, 4th year Undergraduate, Queen's University</i>
11:48am-11:59am	Delivery of scAAV9.coSLC6A8 for restoration of creatine transporter function in a creatine transporter deficient mouse model: A dosage study <i>Chiara Sawilla, MSc Candidate, Queen's University</i>
4:45pm-5:45pm	STUDENT TALK SESSION 2
4:45pm-4:56pm	Reconsidering the relationship between negative symptoms, cognitive impairments, and depression in persons with schizophrenia <i>Supriya Bains, MSc Candidate, McMaster University</i>
4:57pm-5:08pm	The impact of a murine coronavirus (MHVJHM) upon alpha-synuclein and inflammatory factors in primary wild-type and LRRK2 G2019S mutant microglia and midbrain neuronal cultures <i>Stephanie Hobbs, MSc Candidate, Carleton University</i>
5:09pm-5:20pm	Ultraweak photon emission (UPE) neuroimaging device to read brain states: A proof-of-concept study <i>Dr. Victoria Hossack, Post Doc, Wilfrid Laurier University</i>
5:21pm-5:32pm	Improving the neuroD1-AAV-based gene therapy intracerebral injection protocol for optimal neuronal recovery <i>Golnar Taheri, PhD Candidate, Queen's University</i>
5:33pm-5:44pm	Exploring low-intensity pulsed ultrasound as a non-invasive strategy for medulloblastoma treatment <i>Tiffany Yu, 3rd year Undergraduate, Queen's University</i>

Speakers

Keynote Speaker: Dr. Caroline Pukall

Dr. Pukall joined the Queen's University psychology department in 2004, and is the Director of the Sexual Health Research Laboratory (SexLab) and the Director of the Sex and Relationship Therapy Service at the Queen's Psychology Clinic. She is also a Tier 1 Canada Research Chair in Sexual Health, and teaches courses related to sexuality at the undergraduate and graduate levels, having won several awards related to her teaching and research. She uses many methodologies to examine different aspects of human sexuality, including self-report measures, imaging methods, quantitative testing and sexual psychophysiology to allow for mixed-method and multimethod study designs. She looks at various clinical and non-clinical populations, with a focus on ensuring that sexual and gender/sex minorities are represented in her work.

Invited Speaker: Dr. Susan Boehnke

Susan Boehnke completed a PhD in Neuroscience (2002) at Dalhousie University as a Killam Scholar using psychophysical and neurophysiological approaches to examine spatial processing in the auditory system. She then completed a postdoctoral fellowship in primate visual and oculomotor neurophysiology with Dr. Doug Munoz at Queen's University. In collaboration with Drs. Munoz and De Felice (Brazil), she took a lead role in the creation and validation of one of the first non-human primate (NHP) models of Alzheimer's disease using behavioural, neurophysiological, neuroimaging, and molecular approaches. She led development of the first NHP biobank in Canada, and the establishment of normative values of fluid biomarkers of neurodegeneration in macaques. During COVID, her team used the NHP facility at Queen's to explore the effect of social enrichment and isolation on the brain.

In response to recent explosion of interest in neurotechnology, such as brain computer interfaces, she has now turned her attention to creating a micro-credential program in neurotechnology (<https://neurotechmicrocreds.com/>) and exploring ethical issues related to neurotech. She is also the lead of the Training Committee for Connected Minds: Neural and Machine Systems for a Healthy, Just Society, a large Canada First Research Excellence Fund (CFREF) program between York and Queen's Universities.

Invited Speaker: Dr. Linda Booij

Dr Booij is a Full Professor in the Department of Psychiatry at McGill University, clinician-scientist, clinical psychologist, and Head of Research and Academic Development of the Eating Disorders Continuum of the Douglas Mental Health University Institute in Montreal. At the Eating Disorders Continuum, she directs a research program that focuses on the biopsychosocial pathways of eating disorders. This includes studies on the brain and epigenetic mechanisms of anorexia nervosa, predictors and trajectories of eating-disorder treatment response, development of virtual interventions, and studies on the effectiveness of knowledge-transfer initiatives. Dr. Booij is also active in the broader eating-disorder community. She serves as an Associate Editor for the Journal of Eating Disorders, is past president of the Eating Disorders Association of Canada, and is co-chair of the Educational Programming Committee of the International Academy for Eating Disorders.

Eating Disorders are thought to result from complex biopsychosocial interactions. Increasing evidence suggests that epigenetic processes play a central role in how environmental factors can trigger genetic susceptibilities in the development of eating disorders.

In this presentation, Dr. Booij will present completed and ongoing research conducted at the Douglas' Eating Disorders Continuum on the biopsychosocial pathways of anorexia nervosa, including studies on epigenetic and brain mechanisms.

Student Poster Abstracts

1. Comparing human and non-human primate saccade, pupil, and blink behaviour during free viewing of dynamic video presented by Tyonna G. Ashby, undergraduate student at Queen's University

The visuo-oculomotor system is a highly interconnected circuit where external visual stimuli from the environment, cortical, and subcortical areas provide dynamic input into brain circuitry, shaping oculomotor behaviours such as saccade, blink, and pupil responses. Due to its interconnectedness, dysfunction or deterioration of cortical or subcortical networks can result in abnormal oculomotor behaviours, as seen in psychiatric disorders and neurodegenerative diseases. Non-human primates (NHPs) research is critical to understanding the neural circuitry involved in altered oculomotor behaviour due to the functional and anatomical homologies of their visual and oculomotor systems. Previous research has established the patterns of saccade, blink and pupil responses during critical epochs in controlled tasks. NHP oculomotor behaviour in unstructured free viewing of dynamic video clips provides the opportunity to explore the neural circuitry in more naturalistic tasks. This study aims to establish free-viewing behaviour in NHPs and quantitatively contrast it with human behaviour under identical conditions. Free-viewing videos were presented to NHPs and oculomotor behaviours were observed during video-based eye tracking (Eyelink 1000). Data collected from NHPs was compared to previously collected human controls. The task involved watching 3-5-second video clips featuring naturalistic scenes, such as animals, buildings, and humans. Each clip change created a large visual perturbation influencing ongoing saccade, pupil, and blink behaviour in NHPs and humans. These perturbations are altered in clinical groups. This study will be among the first to quantitatively contrast the free-viewing behaviour in humans and NHPs. Establishing these parallels will provide critical insights into the neural basis of oculomotor behaviour in health and disease.

2. Altered social and depression-like behaviours support a two-hit model of stress in Long-Evans rats presented by Megan Babcock, graduate student at Queen's University

Early-life adversity (ELA) increases the risk of later-life psychopathology, however, not everyone who experiences ELA will develop psychopathology. Recent studies in our lab sought to examine a “two-hit” hypothesis of stress sensitivity. This hypothesis posits that a first hit of stress in early life sensitizes an individual to a later hit of stress, leading to adverse outcomes when combined. In these studies, rats were exposed to either the limited bedding and nesting (LBN) stress paradigm as a “first hit” of stress or served as a neonatal no-stress control (nCON) group. Half the offspring from each neonatal condition were exposed to adolescent subthreshold chronic mild stress (CMS) as the “second hit,” while the other half served as adolescent controls (aCON). In our first experiment, subjects (n=12 males/group) were tested in a forced-swim test (FST) and social interaction test (SIT). Here, offspring from the two-hit (LBN-CMS) group displayed greater immobility in the FST, as well as decreased social play and increased aggression in the SIT compared to the other three groups (nCON-aCON, nCON-CMS, LBN-aCON), which were indistinguishable from each other. These findings suggest that the two-hit animals are experiencing a depression-like response. Current analysis of the density of immunoreactive oxytocin-producing cells in the hypothalamus aims to examine potential stress-induced changes in the brain's oxytocin system. We have also initiated a second experiment using an expanded range of behavioural tests, as well as incorporating sex as a variable. Here, offspring (n=10 males/group and n=10 females/group) were tested in the FST, sucrose preference test, and splash test. Data analysis from the second study is ongoing but results indicate that offspring in the two-hit group show depression-like reductions in sucrose preference compared to the other three groups. The brains from this study will be assessed for oxytocin mRNA expression in the hypothalamus using qRT-PCR.

3. The association of MANF/CDNF in neurodegeneration for pre-clinical animal models: A Systematic Scoping Review presented by Almila Bahar, graduate student at Queen's University

Background: Neurotrophic factors, such as mesencephalic astrocyte-derived neurotrophic factor (MANF) and cerebral dopamine neurotrophic factor (CDNF), play critical roles in neuroprotection, neuronal survival, and mitigation of cellular stress. These evolutionarily conserved proteins have been implicated in maintaining cellular homeostasis by modulating endoplasmic reticulum (ER) stress, autophagy, and lysosomal function. Despite growing interest in their potential for therapeutic applications in neurodegenerative diseases, a comprehensive understanding of their mechanisms across diverse organisms remains limited.

Objective: This systematic review aims to synthesize existing research on MANF and CDFN, focusing on their neuroprotective roles across vertebrates and invertebrates, including *C. elegans*, flies, and mice. By examining their impact on stress-related cellular pathways and their contributions to neurodegeneration and aging in different model systems.

Methods: This review will follow PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. A comprehensive search of electronic databases (e.g., Web of Science, Embase, and Ovid Medline) will identify original research articles examining MANF and CDFN across various organisms. Inclusion criteria will focus studies reporting molecular, functional, or behavioral outcomes related to neuroprotection and cellular stress responses, including ER stress, autophagy, oxidative stress and neuroinflammation. Studies involving mice, *Caenorhabditis elegans* (*C. elegans*), and *Drosophila* will be included to explore conserved neuroprotective mechanisms. For this review, neuroprotection is defined as the preservation of neuronal structure and function, mitigation of cellular stress, prevention of apoptosis, and promotion of functional recovery under pathological conditions. Reviews, meta-analyses, and studies lacking sufficient data or relevance to MANF/CDNF will be excluded.

Future Directions: Ultimately, a clearer understanding of how MANF and CDFN confer neuroprotection and regulate stress pathways will help drive the development of targeted treatments for a range of neurological conditions.

4. Characterizing cortical responses during preformance of KINARM sensorimotor tasks using fNIRS in healthy young adults presented by Cassidy Bretney and Hannah Nicholson, graduate students at Queen's University

The KINARM robot, and its associated task battery, has become the gold-standard for assessing sensorimotor function. This tool is particularly useful in diagnosing neurological conditions, as well as assessing rehabilitation. However, the KINARM has not been used in combination with functional neuroimaging, to better understand the cortical responses to these tasks. Functional near-infrared spectroscopy (fNIRS) provides a non-invasive neuroimaging technique which allows investigators to observe brain activity through the hemodynamic response. This study aims to use the KINARM and fNIRS together, in order to better understand the cortical responses driving sensorimotor performance on these standard KINARM tasks. Participants will wear the fNIRS cap while completing specific KINARM tasks: Object Hit, Object Hit & Avoid, Visually Guided Reaching, and Reverse Visually Guided Reaching. The primary objective of this study is to characterize the general cortical response to each task in the prefrontal and motor cortex of healthy participants. The secondary objective is to compare cortical activity to various performance metrics (ie. accuracy, and speed) to uncover potential associations. Preliminary results show distinct patterns of hemodynamic activity in the prefrontal and motor cortices during task performance. Contralateral motor regions exhibit stronger activation during tasks requiring precise reaching movements, while prefrontal activation is linked to decision-making demands in tasks like Object Hit & Avoid. Preliminary analysis also suggests correlations between select task performance metrics and the magnitude of cortical activation. Further analyses are needed to investigate these findings further in a larger population. This novel combination of KINARM and fNIRS provides a window

into how cortical activity drives motor task performance in the healthy individual. Insights gained from this research have the potential to inform personalized rehabilitation strategies and to establish a ‘healthy baseline’ which can be leveraged to improve early detection of sensorimotor deficits in clinical populations.

5. Responsible use of AI in quantitative neuroscience research presented by Jocelyn “Adelle” Barsky-Moore, undergraduate student at Queen’s University

Is there an ethically responsible way that AI can be used with subject data in quantitative research? In fields like neuroscience, AI proponents suggest that it has incredible capabilities to find complex patterns that could not be accomplished with traditional methods of data collection and analysis. However, AI comes with cons such as overfitting and spreading biases within the data (Badrulhisham et al, 2024). With limited official guidance on how to use AI in academic research, researchers need to consider how to uphold the principles of ethical and responsible research by maintaining data privacy, preserving intellectual property, avoiding plagiarism, and stating whether and how AI has been used (Government of Canada, 2023, Government of Canada 2024). Data safety is vital within the use of large language models (LLMs). LLMs are machine learning models trained through large amounts of text, which can understand and generate human language through processing immense amounts of text data. However, these models are not perfect. They rely on statistical predictions based on data to determine what is the next most likely word in a sequence. These models follow a gradient-based optimization during training to approximate these conditional probabilities. This presentation will demonstrate how localized LLMs can promote data safety. Localized LLMs get their name from running on a user’s device or local network. No data ever leaves the user’s computer. Some LLMs are better than others for different tasks. This presentation aims to help researchers determine whether using an LLM is right for them, how to optimize for accuracy, considerations between choosing localized (and open source) LLMs or closed-source options. This research builds upon previous papers discussing ways AI can be used in neuroscience and other forms of human subject research (Badrulhisham et al., 2024), focusing on the ethical and responsible use of AI in neuroscience research and data analysis.

6. Impact of repeated subconcussive hits on neurophysiological parameters in collegiate football athletes presented by Kim Huynh, graduate student at Queen’s University

Background: A subconcussive injury occurs from a direct or indirect impact to the head that does not result in overt symptoms. However, growing evidence suggests that the accumulation of subconcussive impacts can result in neurological symptoms, likely due to altered neural functioning. Previous magnetic resonance imaging (MRI) studies revealed that athletes who experienced a greater number of subconcussive impacts exhibited elevated cerebrovascular reactivity (CVR) and decreased cerebral blood flow (CBF) compared to athletes who experienced fewer impacts. CBF refers to the rate of blood supply to the brain, while CVR is the change in blood flow in response to a change in the demand for blood. The objective of this study is to determine the effects of repeated subconcussive exposure on neurophysiological parameters in football athletes. This study aims to contribute to the growing body of evidence regarding the impact of subconcussive injuries on brain health.

Methods: A total of 20 male collegiate football athletes were recruited to participate in this study. Participants underwent MRI scans to measure CVR and CBF at three time points: within two months prior to participation in full contact practices, following a 14-day training camp period, and within one month post-season. Statistical analysis was performed to compare CVR and CBF values at the three time points.

7. Attention modulates feedback and feedforward motor control systems for speech production within children presented by Rita Bishai, graduate student at Wilfrid Laurier University

Speech is critical for communication and as such, it is vital to understand the mechanisms involved in the control of speech production. Feedback and feedforward motor control systems work together to correct for errors in speech production and initiate speech, respectively (Guenther et al., 2006). Attention has been shown to impact the weighting of feedback and feedforward mechanisms under experimental manipulations of attention (Scheerer et al., 2016; Tumber et al., 2014), yet it is unclear if individual differences in attentional abilities may influence these mechanisms. This study measured attentional abilities to probe whether differences in attention might be related to the control of ongoing vocalizations in children ages 4-12. Children were instructed to produce vocalizations while their voice was suddenly shifted up or down 100 cents, 3 times per vocalization. Children's vocal pitch was recorded to assess the magnitude of compensatory responses to the 100 cent manipulations. Individual differences in attentional abilities were measured through performance on the sustained attention to response task (SART). Children who performed worse on the SART produced significantly smaller compensatory responses to the 100 cent manipulations. This relationship between attention and compensatory responses was still marginally significant when controlling for the effects of age. This suggests that attention modulates the weighting of feedback and feedforward control whereby reduced attentional abilities results in a decreased weighting of the feedback control system. Additionally, this finding is independent of age-related differences in attentional abilities and speech production suggesting a unique contribution of attention on speech motor control. Future research is required to help clarify the developmental implications of attentional differences on speech production.

8. Parkinson's Disease patient-centered study: Measuring the impact of adjusting deep brain stimulation parameters on gait and sensorimotor function presented by Abigail Ball, Alena Ionova, Sara Stephenson, & Khoi Tran, undergraduate students at Queen's University

Background: Parkinson's Disease (PD), the second most common neurodegenerative disorder, is characterized by motor symptoms like bradykinesia, tremors, rigidity, and gait disturbances. While levodopa offers initial relief, its long-term efficacy diminishes, leading to side effects like dyskinesia (Zafar and Yaddanapudi 2024). Deep brain stimulation (DBS), to structures like the subthalamic nucleus (STN), serves as a viable alternative, yet variability in patient symptoms poses challenges in optimizing DBS settings. This study aims to identify objective metrics and reliable biomarkers for evaluating DBS settings by integrating advanced neurotechnological and behavioural assessments.

Methods: In a single case-study, a DBS-treated (Vercise Gevia, Boston Scientific) PD patient participated. Motion was recorded using a markerless motion capture system (Theia3D, Theia Markerless, Inc.) during treadmill walking and the Timed Up and Go (TUG) test. Functional connectivity was assessed with functional near infrared spectroscopy (fNIRS) during rest and motion. Upper limb movements were evaluated with an exoskeleton (KINARM Robotic Lab).

Preliminary Results: Preliminary fNIRS results revealed higher connectivity between the dorsolateral prefrontal cortex and other regions on DBS settings less effective for gait. Theia3D identified variations in posture and right-arm swing across DBS settings, with 104 Hz programs showing increased torso lean and treadmill support use, while 149 Hz programs minimized support time but altered arm rigidity. KINARM tests revealed significantly worse spatial attention and visual-motor performance at 149 Hz and 104 Hz compared to baseline, with working memory unaffected.

Discussion: By identifying the biomarkers that objectively evaluate DBS parameters, we aim to develop a more personalized approach to DBS programing that alleviates motor dysfunction. Promoting patient engagement will set an example for future research guided by the lived experiences of individuals. Additionally, we will demonstrate the clinical applicability of multi-modal DBS assessment, which integrates diverse data sources to provide a comprehensive view of motor and cognitive symptoms.

9. Eye tracking to assess state anxiety during a mental task presented by Samantha Bjelis, graduate student at University of Waterloo

Anxiety, particularly state anxiety, is a transient emotional response affecting cognition and performance (Jouvent et al, 1999). Anxiety disrupts processing efficiency and sensory sensitivity, which manifests in tasks requiring precision and focus [Darvishzadeh et al, 2012; Eysenck et al, 2007]. Thus, anxiety disrupts cognitive performance, especially with high cognitive difficulty or load (Maloney et al, 2014). State anxiety has been also associated with poorer learning outcomes (Maloney et al, 2014). Given the ubiquity of state anxiety in everyday situations, it is important to develop indicators of state anxiety.

A key gap in research is the lack of objective measures of state anxiety. While there are many subjective measures, they do not fully capture the physiological effect. State anxiety involves neurophysiological and sympathetic responses to arousing stimuli that are indicative of a changing neurophysiology (Saviola et al 2020). Eye tracking metrics provide external measures to these internal state changes. Pupil dilation reflects an increase in cognitive load recruited to complete a given task (Krejtz et al, 2020). Micro-saccades (MS) are small eye movements associated with high-level attention modulation related to cognitive effort. Specifically, MS rate decreases while MS magnitude increases with greater task difficulty [Alnaes et al, 2014; Kretjz et al, 2020], resulting in poor fixational stability.

Maths Anxiety (MA) is a form of state anxiety induced by arithmetic tasks. Studies show that MA reduces neural activity in brain regions associated with arithmetic processing, leading to deficits in executive function. This reduces the ability to allocate cognitive resources effectively, diverting attentional resources and impairing task performance (Pizzie et al, 2020; Demedts et al, 2022).

To examine these changes, participants will engage in a time pressured math task. Participants will complete 45 basic arithmetic problems presented on screen, which must be verbally solved in 60 seconds. Eye movements and pupil size will be recorded using the Eyelink 2 tracker. Heart rate and subjective state anxiety will be tracked throughout the task. We hypothesize that the time pressure to complete the arithmetic task will induce state-MA, which will be associated with increased pupil dilation, decreased micro-saccadic rate, and larger MS magnitude.

10. Reducing the burden of post-intensive care syndrome: A pragmatic, mixed-methods, open-label randomized controlled trial of a post-ICU bundle of care presented by Natasha A. Jawa, graduate student at Queen's University

Background: Most ICU survivors experience post-intensive care syndrome (PICS), characterized by persistent cognitive/physical/psychiatric impairments. Caregivers similarly experience PICS-family. However, follow-up care for both groups is inconsistent and unstandardized. This study aimed to develop, implement, and evaluate the feasibility, perceptions, and effectiveness of a standardized post-ICU follow-up bundle of care for ICU survivors and caregivers on clinical and psychosocial outcomes.

Methods: This single-center, mixed-methods, open-label, pragmatic trial enrolled ICU survivor-caregiver dyads ≥ 18 years at high risk for PICS ($n=20$), randomized 1:1 to receive either the intervention (ICU diary and informational resources during admission, and outpatient follow-up care with an intensivist, social worker, and pharmacist at 1- and 3-months after ICU discharge) or control (no specific follow-up). Intervention materials and standard processes of care were developed and refined using constituent (physician, allied health provider, ICU survivor, and caregiver) feedback. Feasibility metrics included consent ($>80\%$), enrollment (4 dyads/month), follow-up ($>70\%$), and data capture ($>80\%$) rates. Implementation was assessed via process evaluation. Quantitative assessments evaluating cognitive, psychological, and functional status, as well as qualitative feedback were collected at 6-months via standardized clinical tools and cognitive interviews.

Results: From March-June 2024 we enrolled 20 dyads (87% consent rate). Data capture rates exceeded 90% at all time points. Follow-up visit completion rates were 33% at 1-month, 44% at 3-months, and 89% at 6-months. Clinic attendance varied due to illness severity and logistical barriers. Focus group/cognitive interview results demonstrated the benefits of the multifaceted intervention for emotional recovery and caregiver engagement: both groups found value in receiving these resources, but

desired individualized versions to support their own recovery journeys after discharge. Clinical outcome assessment is underway.

Conclusion: This study demonstrates the feasibility, acceptability, and perceived benefits of a structured post-ICU follow-up care bundle. Our findings inform scalable, standardized pathways for improving long-term outcomes of critical illness.

11. Catch-up saccade latencies in the presence of simulated central vision loss presented by Jenna Black, undergraduate student at Queen's University

Humans visually track moving objects by combining smooth pursuit and saccades, which coordinate to maintain the image of a moving object on the fovea. When smooth pursuit fails to keep up with the object's motion, a catch-up saccade is triggered to correct the resulting tracking error. In individuals with central vision loss, as seen in age-related macular degeneration, the loss of the fovea disrupts visual tracking and increases reliance on peripheral vision for tracking. Despite advances in research involving visual tracking with artificial central scotomas, a gap remains in understanding how catch-up saccades are triggered during pursuit under these conditions. Specifically, we are interested in how an artificial central scotoma will influence catch-up saccade latency. In our experiment, fully sighted participants completed a series of visual tracking tasks under simulated scotoma conditions, allowing us to assess the influence of visual occlusion on saccade-pursuit interactions in the absence of the fovea. We aim to investigate whether saccade latencies differ as a function of target visibility. We hypothesize that the increased uncertainty when a target is occluded by the scotoma will result in longer saccade latencies compared to when the target is visible in the participants peripheral vision. Our overall goal is to quantify the effects of artificial central scotomas on saccade latency to better understand saccade-pursuit interactions with central vision loss. Our findings could ultimately assist in the development of rehabilitation strategies for individuals with central vision loss, thus improving quality of life.

12. Exploring the link between impulsivity, using the UPPS-P Impulsive Behavior Scale, and decision-making regarding risky choice presented by Mariel Kandalaft, undergraduate student at McGill University

Acting without forethought is a characteristic some individuals exhibit in their everyday lives, particularly those prone to impulsive behaviour. Many personality models incorporate impulsivity as a fundamental psychological construct (Whiteside & Lynam, 2001). Consequently, understanding how impulsivity influences decision-making, especially in risky contexts, is essential for understanding the behavioural manifestation of personality traits. In this study, we aimed to determine the association between impulsivity, measured by the UPPS-P Impulsive Behavior Scale (Lynam, Smith, Whiteside, & Cyders, 2006), and risky choice decision-making measured by a risky choice paradigm (da Silva Castanheira, Fleming, & Otto, 2021; Rutledge, Skandali, Dayan, & Dolan, 2014). A total of one hundred and forty-two participants took part in the online study, where they completed the paradigm, as well as the UPPS-P questionnaire. The task involved presenting participants with a choice between risky and certain options. A regression analysis was conducted to examine the relationship between their probability of selecting the risky option. Moreover, higher scores on this scale predicted faster reaction times in the task, especially when participants rated high on both (Positive and Negative) Urgency subscales of the UPPS-P. Overall, this study provides a deeper understanding of how distinct facets of impulsivity contribute to risky decision-making behaviours, particularly by influencing both the likelihood and speed of risky choices.

13. Investigating patterns of cerebral oxygenation in ICU patients with delirium using fNIRS: A feasibility study presented by Cassidy Bretney, graduate student at Queen's University

Delirium is an acute state of altered consciousness, affecting one's perception, cognition, and memory. This condition is a particularly large issue in the Intensive Care Unit (ICU) occurring in 80% of ICU patients. The presence of delirium in these cases is an independent factor for worse patient outcomes including increased length of hospital stays and ventilation, increased rate of readmission to the hospital, and increased mortality. As such, it is important to identify and effectively manage delirium. However, targeted, evidence-based therapies and management strategies are limited by poor understanding of the pathophysiology of delirium. Previous studies have found that cerebral oxygenation, as measured by Near-Infrared Spectroscopy (NIRS), is lower in critically ill patients with delirium as compared to non-delirious patients. A separate study using functional NIRS (fNIRS) observed significant depression throughout the frontal lobe of children experiencing delirium after anesthesia. However, fNIRS has not been used as a tool to investigate the pathophysiology of delirium in critically ill patients. This study aims to address this by using fNIRS alongside the standard CAM-ICU delirium screening in a population of critically ill patients. The overall objective of this program of research is to better understand the pathophysiological processes underlying delirium. Specifically, patterns of activation using fNIRS will be explored in critically ill patients to determine whether there is an association between fNIRS patterns of activation and delirium. There are three specific aims of this study. The first is to determine the feasibility of using fNIRS in critically ill patients. The second is to explore the changes in patterns of cerebral oxygenation and their association to delirium in critically ill patients. The third is to determine if fNIRS can serve as a predictor to the duration and severity of delirium in critically ill patients.

14. Investigating the contributions of the right temporoparietal junction in cybersickness and sensory reweighting during virtual reality exposure presented by Alyssa Lynn, graduate student at University of Waterloo

When using virtual reality (VR), individuals may experience cybersickness, characterized by nausea, dizziness and disorientation. Sensory conflict theory is the leading theory in cybersickness research, and states that incongruent signals from the sensory systems detecting motion cause cybersickness. In VR, visual motion cues often conflict with signals from the vestibular and proprioceptive systems, leading to sickness in some, but not all, users. Individual differences in cybersickness susceptibility may be attributable to users' ability to reweigh the contributions of each sensory system – a process known as sensory reweighting. Sensory reweighting allows users to prioritize reliable sensory information while minimizing the perceptual toll of noisy, unreliable or incongruent signals.

This study investigates the contributions of the right temporoparietal junction (rTPJ) in sensory reweighting and the outcomes for cybersickness. We hypothesize that the rTPJ is involved in sensory reweighting and its contributions can be manipulated through transcranial direct current stimulation (tDCS). Under this hypothesis, tDCS targeting the rTPJ can modulate sensory reweighting and decrease or increase cybersickness, depending on the tDCS polarity. Sensory modality reliance for upright judgements is assessed using the Oriented CHaracter Recognition Task (OCHART), which provides a measure of individual sensory weighting. The study findings will shed light on the contributions of the rTPJ in sensory reweighting and may help inform cybersickness mitigation strategies to improve VR experiences.

15. Who's a Good Dyad? Owners' pre-existing beliefs about their dog's impulsivity shape interactions in novel settings presented by Hannah Burrows, graduate student at Queen's University

Guardians' responses to their dogs' impulsive behaviours can act as real-time feedback, shaping the frequency and nature of these behaviours. This study examines these dynamics by (1) creating the Perception of Undesirable Pet Behaviors Survey (PUPS) to capture guardians' beliefs regarding their dogs' impulsive behaviours and (2) examining how these beliefs relate to the expression and management of dogs' impulsivity during interactions in novel settings.

PUPS overcomes some limitations of existing impulsivity scales by focusing on observable behaviours within specific social contexts (e.g., "I feel comfortable walking with my dog in places where there are bicycles, squirrels, or other moving objects") rather than inferred mental states (e.g., "My dog is often anxious"). An EFA identified core contextual behavioural dimensions of beliefs regarding impulsivity.

These prior beliefs about impulsivity are examined in relation to behaviour during a free play session and a surprise inhibitory control task (when desirable items are off-limits). State Space Grids (SSGs) are used to determine dynamic behavioural patterns between the dog and guardian over time, highlighting, for example, attractor states—persistent behavioural patterns, such as mutual engagement or avoidance—that indicate how each participant's actions influence the other's behaviour.

16. Exploring the role of sensory processing on intolerance of uncertainty presented by Adrianna Molenaar, undergraduate student at Wilfrid Laurier University

The environments we interact with contain varying amounts of sensory information with this information often being difficult to anticipate. People vary in how well they tolerate the unpredictable nature of sensory information, with some showing an intolerance of uncertainty (IU). IU commonly occurs in those with high levels of trait anxiety, autistic traits, depressive symptoms and panic disorders. Those high in IU often experience negative feelings and discomfort when exposed to an unexpected stimulus, for example, a louder-than-usual toilet flushing. This can lead to higher levels of anxiety, impacting overall well-being as unexpected stimuli are common in the sensory environments we navigate in our everyday lives.

When attending to unexpected stimuli, we often demonstrate threat-related attentional bias, the tendency to allocate more attention to unexpected stimuli, a bias that is exaggerated in those who are high in IU. Further, increasing the attentional load during an attentional bias task has a greater impact on those high in IU, making it more difficult for those high in IU to disengage from unexpected stimuli. To investigate threat-related attentional bias, a dot-probe task will be employed. Those high in IU are found to have faster response times on trials displaying negative emotion, suggesting more attention is allocated to negative stimuli.

This proposed research will explore how low and high sensory environments impact attentional bias in those with varying levels of IU. We expect that those higher in IU show faster reaction times to threatening stimuli in higher sensory environments, compared to lower sensory environments. This project will highlight the role of sensory processing in our ability to process and disengage from the unknown. Overall, this research will help to lay the groundwork for understanding how IU influences the well-being and attention of those interacting in a sensory-rich world.

17. Characterizing subthalamic and pallidal evoked recurrent neural activity in Parkinson's disease and dystonia presented by Reese Clinton, undergraduate student at Queen's University

Background: Deep brain stimulation (DBS) of the subthalamic nucleus (STN) or globus pallidus internus (GPi) is an effective treatment for movement disorders like Parkinson's disease (PD) and dystonia. Recent studies have identified evoked recurrent neural activity (ERNA) as an oscillatory feature of the subthalamo-pallidal network associated with optimal electrode placement. However, direct comparisons

of ERNA across brain regions, indications, and its clinical significance remain unexplored.

Methods: We performed ERNA “hotspot” mapping in 52 people with PD (n=28, 40 STN trajectories; n=24, 33 GPi trajectories) and 11 with dystonia (16 GPi trajectories) during awake, microelectrode-guided DBS surgeries. High-frequency stimulation (100 Hz for 2 seconds) was applied at each millimeter along the microelectrode recording trajectory, and ERNA features, including amplitudes, latencies, and growth rates of the first (P1) and second (P2) peaks, as well as the peak ratio (P1:P2), were extracted at the ERNA hotspot. Features were compared across structures and diseases and correlated with pre-operative off-medication Unified Parkinson's Disease Rating Scale Part III (UPDRSIII) scores.

Results: In PD, P1 and P2 latencies, amplitudes, and the P1:P2 ratio were significantly greater in STN than GPi; however, there were no differences in growth rates. In the GPi, P1 latency was longer in PD than in dystonia, while P2 latency and amplitude showed no differences. The P1:P2 ratio was higher in dystonia compared to PD. Growth rates differed between PD and dystonia for P2 but not P1, with little to no P2 growth in dystonia. The ERNA peak ratio negatively correlated with the UPDRSIII score.

Conclusions: This study identified disease- and site-specific differences in ERNA characteristics. The P1:P2 ratio, which negatively correlated with the UPDRSIII score, is of clinical relevance as it may serve as an indicator of GPe neuron recruitment efficiency. This ratio may guide real-time DBS parameter adjustments in a closed-loop manner.

18. Understanding developmental coordination disorder: A comprehensive review presented by Fatin Mustafa, undergraduate student at Queen's University

Developmental coordination disorder (DCD) is a neurodevelopmental condition that impairs the execution of motor movements and motor learning. The disorder progresses throughout adulthood and negatively affects the performance of daily activities and quality of life. Whilst the disorder is prevalent in 5-6% of children in Canada, it remains underdiagnosed and poorly understood. Thus, this literature review provides a comprehensive understanding of current research findings on DCD, through synthesizing information on the disorder's history, diagnosis, theories of etiology, as well as current and novel treatments. DCD has been identified throughout history with evidence of its prevalence dating back to the Victorian period. Studies conducted using imaging techniques such as magnetic resonance imaging (MRI) and functional MRI (fMRI), suggest that alterations in the frontal lobe, cerebellum, basal ganglia, thalamus, and white matter pathways may underly the condition. Modifications to functional connectivity and neuroplasticity are also thought to play a role in DCD development, with conflicting evidence on whether the mirror neuron system (MNS) participates in the etiology of the disorder. Current treatments are geared towards disease management and include physiotherapy, occupational therapy, as well as task based interventions, with pharmaceutical interventions administered in cases of comorbid DCD and attention deficit hyperactivity disorder (ADHD). Novel interventions, currently being investigated in clinical trials include the use of virtual reality (VR), to enhance motor performance, and transcranial direct current stimulation (tDCS), a non-invasive brain stimulation technique. Through highlighting the gaps in research about DCD, this review underscores the necessity for further research about the disorder's etiology and treatment interventions to improve health outcomes and the quality of life of impacted individuals.

19. Using LIPv and LIPd to divulge the microcircuitry of persistent activity in visuospatial working memory presented by Holly M. Crowson, graduate student at Queen's University

Working memory (WM), the cognitive ability to briefly store a small amount of information for goal-directed behaviour, is essential for daily functioning but its neural substrate is poorly understood. One proposed mechanism for retaining information with WM is persistent neuronal activation. Neurons in the lateral intraparietal (LIP) area of rhesus macaques have shown these patterns of persistent activity during visuospatial WM (VWM); differentiating neural firing patterns of the ventral and dorsal subdivisions (LIPv

and LIPd, respectively) may compliment existing structural and behavioural data to divulge some of the neuronal microcircuitry necessary for generating and maintaining persistent activity in WM. The present study aims to characterize the involvement of LIPv and LIPd in VWM, by analyzing neurophysiological data from the LIP area of four macaques while they perform a memory-guided saccade task (in which a visual stimulus is remembered across a delay). The spiking behaviour of LIP neurons is used to calculate visuomotor association, signal strength, variance, fidelity, and intrinsic dynamics including timescales and bursting activity. These metrics were analyzed with respect to cortical depth to distinguish the neurophysiological contributions of dorsal and ventral neurons to the LIP area. Signal strength, fidelity, and bursting activity were significantly higher for neurons located towards the dorsal side of the cortex, While visuomotor association and intrinsic timescale did not differ between the populations. These firing activity parameters help to characterize the structure-function relationships of LIP neurons in VWM, providing constraints for the parameters of further computational modelling to better understand the mechanism of persistent activity.

20. How verbal coaching impacts learning an electromyography-based body-machine interface which moves a cursor presented by Prisha Adya, Kristen Banks, Shayne Belchos, & Riya Patel, undergraduate students at Queen's University

Background: Body-machine interfaces (BMIs) are promising tools for improving human-computer interaction, with applications in rehabilitation, assistive technologies and performance enhancement in sports and physical training (Casadio et al., 2012). Electromyography (EMG)-based BMIs, which decode motor intentions from muscle signals, provides a non-invasive interface technology with high sensitivity, but face challenges such as variability in muscle activation, electrode placement and difficulty in mastering unintuitive motor tasks. Research demonstrates the importance of feedback in motor learning (Barradas et al., 2023; Cotellessa et al., 2024; Qiu et al., 2018), with verbal coaching shown to effectively enhance motor unit recruitment, reduce task variability and improve learning outcomes in various settings. However, the role of verbal coaching in supporting unintuitive EMG-based BMI learning remains largely unexplored.

Methods: This study examines the impact of verbal coaching on motor learning during unintuitive EMG-based BMI tasks. Participants aged 18–23 were tasked with controlling a computer cursor using wrist and forearm muscle activation. Divided into Coaching and No Coaching groups, the former received procedural verbal instructions (e.g., "Contract your wrist to the left"), while the latter received no guidance. Learning was assessed through cursor accuracy, task completion times and neural adaptation measured via electroencephalogram (EEG) gamma band activity over seven consecutive days.

Anticipated Results: It is hypothesized that participants receiving verbal procedural coaching will demonstrate improved learning outcomes compared to controls. Specifically, the coaching group is expected to achieve faster task completion times, higher cursor accuracy and enhanced neural adaptation as reflected in EEG gamma band activity (Hamada et al., 2023). These findings would highlight the value of verbal coaching in facilitating the learning of unintuitive BMI tasks, providing insights into its role in motor skill acquisition and neural plasticity.

Conclusion: This study investigates the role of verbal coaching in unintuitive motor learning within EMG-based BMI research, aiming to advance BMI system designs for broader accessibility and impact in applications like rehabilitation and assistive technologies.

21. Unreinforced spatial learning and memory in juvenile and adult rats presented by Klara Doelle, graduate student at Carleton University

Introduction: The hippocampus and the anterior cingulate cortex (ACC) play integral roles in the formation and retention of spatial memory. It is hypothesized that early encoding occurs in the hippocampus and as time passes, information is integrated into cortical regions (e.g. the ACC) to create lasting remote memories. In the rodent brain, hippocampal maturation occurs after fundamental motor

and sensory systems have developed. This provides a window for studying aspects of spatial memory in the developing hippocampus. Most spatial memory tasks involve some form of reinforcement or reward to encourage learning; however, this may create additional confounding variables. Using spatial tasks, like the Morris Water Maze, it has been shown that spaced training is more beneficial for long term memory formation than massed training; however, it is unknown whether this has an influence on spatial learning in unreinforced tasks. The present study examined massed and spaced training trials using an Open Field (OF) task in juvenile and adult rats to elucidate developmental differences in unreinforced spatial learning and remote memory.

Methods: In this study, male and female Long Evans rats were trained on an OF task for two consecutive days. Rats were divided into two training groups: massed consisting of one 30-minute session and spaced consisting of three 10-minute sessions separated by 20-30 minutes. Training occurred at either PND21 or PND45, with testing performed three weeks later (PND43 or PND67). Comparisons were made between sexes, ages, and experimental groups. The number of c-Fos positive cells in the ACC and the CA1 region of the hippocampus is used as a measure of activity in these regions. Analysis of behaviour focused on the total distance travelled and time spent in the center of the OF.

Results: Preliminary results of behavioural data analysis suggest differences between juvenile and adult rats. More to come!

22. Assessing the ability of an 8-channel EEG to evaluate cue-reactivity in nicotine users presented by Anika Agarwal, Danielle Noronha, Angela Liu, Chuchao Peng, undergraduate students at Queen's University

Background: With the increasing popularity of cigarettes, e-cigarettes, and nicotine pouches among youth, novel cessation approaches are needed. Cue-induced cravings occur in response to stimuli that encourage smoking behaviour, like nicotine product promotional material or in media. Such cravings can promote addiction and lead to relapse but responses to such cues are often neglected in nicotine cessation approaches. Cue reactivity models provide insight into addiction by examining neural responses to stimuli related drugs like nicotine. P300, an event-related potential, can be used as a measure of cue-reactivity, as it correlates with cognitive engagement and craving intensity. Bu et al.'s (2021) study demonstrated the feasibility of a P300-based neurofeedback system to regulate cue-reactivity using a 64-channel EEG. However, these setups are costly and impractical for widespread clinical and commercial applications. Thus, this study aims to assess the ability of an 8-channel portable EEG to measure cue-reactivity and explore its applicability in nicotine cessation.

Methods: This study consists of two phases. Phase 1 validated the P300 signal-extraction capability of the gTec Unicorn Hybrid Black 8-channel EEG. Participants viewed 60 pictures from an emotion-evoking standardized image set, presented 5 times randomly. Participants rated each image on a 5-item Likert scale while wearing the EEG. P300 signals were extracted from EEGs using Python and MATLAB. Phase 2 will assess nicotine-related cue-reactivity in Queen's University undergraduate participants. EEG methods from Phase 1 will be used with nicotine-related cues (e.g., pictures of cigarette, e-cigarette, nicotine pouch). P300 intensity comparisons will be made between smoking and non-smoking groups.

Results: Phase 1 results show P300 can be measured using an 8-channel EEG. Phase 2 results will focus on assessing and validating the P300 signal as a measure of nicotine-related cue-reactivity. Results will inform if an 8-channel EEG can be widely used for nicotine cessation programs.

23. Emerging GPCR-targeted therapies for Parkinson's Disease: A systematic review and meta-analysis of novel approaches to motor function, neuroprotection, and disease progression presented by Amrit Dosanjh, Manal Rana, and Niki Zadafar, undergraduate students at Queen's University

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the degeneration of dopamine-producing neurons in the nigrostriatal pathway, resulting in motor and non-motor symptoms.

Current treatment options provide symptomatic relief but fail to address the underlying pathophysiology and are often associated with long-term complications. This systematic review and meta-analysis assesses novel G-protein coupled receptor (GPCR) targeted agents to modulate dopamine receptor pathways, reduce microglial activation, and mitigate mitochondrial dysfunction with the aim of addressing motor function, neuroprotection, and disease progression in PD patients. This review systematically searched databases including PubMed, ClinicalTrials.gov, and MEDLINE and identified 12 clinical trials from 2021 onward, investigating emerging therapies such as CVN424, a GPR6 inverse agonist; ND0612, a continuous subcutaneous infusion of levodopa-carbidopa; GLP-1 receptor agonists (lixisenatide and NLY01); and mitochondrial-targeted agents (ursodeoxycholic acid, UDCA). CVN424 and ND0612 demonstrated significant motor function improvements with favourable safety profiles. Lixisenatide and NLY01 exhibited neuroprotective and anti-inflammatory properties, though their efficacy varied based on patient age and treatment duration. Despite small sample sizes and demographic variability, UDCA showed promise in stabilizing motor function and mitigating mitochondrial dysfunction. Emerging therapies like mesdopetam and levodopa-carbidopa further reflect advances in dopamine receptor pathway modulation. Preliminary findings suggest that GPCR-targeted therapies have potential in addressing PD's complex pathophysiology. Despite promising results, limitations such as small cohort sizes, short trial durations, and population variability highlight the necessity for larger, long-term studies. Future research should focus on optimizing GPCR-targeted therapies by exploring their long-term effects across diverse patient populations. This review emphasizes the critical need for innovative, disease-modifying treatments to improve outcomes for individuals with PD.

24. Correction of arginine:glycine amidinotransferase (AGAT) creatine deficiency through a novel AAV9 construct presented by Tesla Peretti, graduate student at Queen's University

Creatine is a small molecule that plays an important role in energy metabolism and is required for overall health and neurodevelopment as seen from creatine deficiency syndromes (CDS). CDS are inborn errors of metabolism which result in neurological manifestations including intellectual disability, developmental delay, and seizures emphasising the importance of creatine in brain function. Endogenous production of creatine begins through the enzymatic function of arginine:glycine amidinotransferase (AGAT) to form guanidinoacetate (GAA) which is then converted to creatine by guanidinoacetate methyltransferase (GAMT) enzyme. AGAT-deficiency (AGAT-D) is an autosomal recessive disorder caused by loss of function mutations in the GATM gene encoding the AGAT protein preventing endogenous creatine production. Currently, the only treatment for AGAT-D relies on daily creatine supplementation, however this is not curative and does not restore AGAT function or normal creatine levels in the brain, leaving many symptoms untreated. We have conducted an intravenous proof-of-concept study with a novel AAV9 construct encoding the AGAT gene. AGAT deficient mice that received this gene therapy showed restoration of creatine concentration levels in brain, muscle, and serum as compared to heterozygous controls. Further studies will aid in determining optimal efficacy and safety of the vectors for translation into clinical use for AGAT-D and potentially other diseases.

25. Examining changes in event-related potentials across a season of college football presented by Cate Downie, undergraduate student at Queen's University

Background: A non-injurious head impact occurs when there is acceleration of the head that does not result in neurological symptoms. In contact sports, these impacts may occur repetitively over the season. Repetitive head impacts (RHIs) in football have been associated with changes in cognitive function, white matter structure, and cerebral blood flow. Traditional clinical imaging techniques like CT and MRI often miss subtle neurophysiological alterations from RHIs and there is growing interest in the validation of tools that can detect neurological changes associated with RHI that are portable and can be performed quickly. One such device is the NeuroCatch which measures event-related potentials (ERPs). ERPs, derived from

electroencephalography, measure cognitive response to stimuli and offer a robust and objective insight into cognitive function. NeuroCatch performs an auditory task and measures N100, P300, and N400 ERPs, reflecting auditory sensation, basic attention, and cognitive processing, respectively. Changes in ERP latency and amplitude, such as delayed N400 responses, are linked to concussion and repetitive non-concussive impacts. This technology can monitor head impact exposure during a season and can identify players with altered ERPs who may benefit from rest.

Methods: 21 Queen's University male football players completed a pre-season NeuroCatch scan. Twelve returned for a post-season scan after 2.5 months. Controls aged 18-22 were scanned twice, 2.5 months apart, and met inclusion criteria of no history of concussion, no contact sport participation, and no medications. NeuroCatch measures ERP amplitude and latency during a 6-minute auditory task through electrodes placed on the ear, forehead, and scalp. A two-way mixed-design ANOVA will be conducted to examine the effects of group (football vs. control) and time (pre-season vs. post-season) on ERP components (N100, P300, and N400) recorded using NeuroCatch.

26. Investigating sex differences in motor learning using robotic evaluation presented by Holly Quinn, undergraduate student at Queen's University

Sex differences have consistently been observed in motor performance, where males tend to perform gross-motor tasks with greater speed and accuracy than females (Moreno-Briseño et al., 2010). Alternatively, during fine-motor tasks, females tend to perform with greater precision and accuracy than males (Litsuko et al., 2020) and additionally display sex-specific neural activation patterns in regions associated with motor learning (Lissek et al., 2007).

The Kinarm is a robotic device that precisely measures upper-limb sensorimotor function, facilitating an assessment of motor learning and associated neurological strategies (Simmatis et al., 2020). When using the Kinarm, participants interact with a virtual system through the handles of the robot to perform interactive tasks, like visually guided reaching (VGR), where the participant reaches from a central to a peripheral target, and reverse visually guided reaching (RVGR), where the same task is performed with the visual feedback reversed compared to hand position. By tracking numerous parameters across trials, including reaction time and angular deviation from the target, the Kinarm provides insight into the learning processes associated with VGR and RVGR. Sex differences have yet to be investigated in a neurologically healthy population using the Kinarm. It is possible that the precise recordings made by this device may reveal subtle differences in motor function not observed by other apparatuses.

This study will investigate sex differences on the VGR and RVGR tasks by investigating across trial performance using the Kinarm robot. During VGR and RVGR, it is hypothesized that females will show lower angular deviation than males, but males will have faster reaction time than females.

This investigation will allow us to understand sex differences in upper limb motor learning and these findings could be used to develop more effective strategies to be used in rehabilitation where the goal is to optimize or restore motor function following neurological injury.

27. Examining a two-hit hypothesis of anxiety using the novelty-induced suppression of feeding presented by Georgia Elliott, undergraduate student at Queen's University

Later life onset of psychopathologies, such as depression and anxiety, have been correlated with incidences of early life adversity (ELA). However, it has yet to be determined how ELA influences trajectories of later life psychopathology onset. The two-hit hypothesis provides a potential explanation. This hypothesis proposes that the first hit (ELA) sensitizes individuals, such that a mild second hit stressor in later life is sufficient to evoke depression/anxiety in individuals with a history of ELA but not in individuals that have not experienced ELA. To understand the causal role in the two-hit hypothesis, I will use a rat model to test it. Neonatal conditions will include both a limited bedding paradigm (LBN; n = 20) and a control (nCON; n = 20) group. Once in adolescence, subjects from both neonatal conditions will be

randomized to receive either a subthreshold course of chronic mild stress (CMS) or will serve as adolescent controls (aCON). This results in four distinct conditions, each with ten rats; nCON-aCON, LBN-aCON, nCON-CMS and LBN-CMS. Anxiety-like behaviours will then be quantified using a novelty-induced suppression of feeding test (NSFT), where latencies to initiate snack consumption will be measured over five days. In that test, longer latencies indicate higher levels of anxiety expression. I hypothesize that double-hit subjects (LBN-CMS) will have longer latency periods compared to controls (nCON-aCON) and single-hit groups (LBN-aCON and nCON-CMS), whereas the latter three groups will not differ.

28. AAV9-mediated gene replacement therapy shows promise in the rescue of sex specific behavioural and electrophysiological phenotypes in a mouse model of X-Linked Intellectual Developmental Disorder-98 presented by Eve Racette, graduate student at Queen's University

Many cases of intellectual disability (ID) are linked to mutations of genes located on the X chromosome, one of which is the Neurite Extension and Migration Factor gene (NEXMIF). The loss of NEXMIF causes X-Linked Intellectual Developmental Disorder 98 (XLID98), a syndrome characterised by intellectual disability, autism spectrum disorders and drug-resistant epilepsy, along with other neurological and non-neurological symptoms. Interestingly, while both men and women are affected by XLID98 and typically present with the abovementioned cardinal features, some symptoms are sex specific, both in terms of prevalence and severity. While the role of NEXMIF is poorly understood, studies have suggested its involvement in neurite outgrowth and orientation, in neuronal migration and in gene expression. Our previous studies on the role of Nexmif in rodents have suggested that, like in humans, XLID98 is associated with a variety of sex-specific phenotypes, such as alterations in their behavioural and electrophysiological parameters. Given the lack of effective symptomatic treatments and the absence of curative therapeutics, our lab has focused its efforts on developing novel therapeutics to cure XLID98. Our hypothesis is that the symptoms associated with the loss of Nexmif in rodents can be rescued in a dose dependant fashion, using an AAV9-mediated gene replacement therapy. To verify this hypothesis, we designed a study where our proposed therapeutic is administered at various doses at birth and the following are assessed : a) the rescue of cognitive impairments via behavioural assays, b) the rescue of electrophysiological parameters using whole cell patch clamping, and c) the biodistribution of the Nexmif gene in treated mice. Considering the lack of appropriate therapeutics for XLID98, these results may pave the way to future clinical trials using our proposed gene therapy. These results will also shed light on the role that Nexmif plays in cellular and molecular mechanisms in the brain, as well as in peripheral organs.

29. Assessing the long-term effects of microbial ecosystem therapeutic (MET-2) on changes in inflammation and clinical response in major depressive disorder: A exploratory pilot study presented by Maria Farid, undergraduate student at Queen's University

The staggering prevalence of Major Depressive Disorder (MDD) and its impact on health-related quality of life necessitates innovative and effective therapeutic interventions. Emerging research suggests the link between the gut microbiome and MDD may be mediated through the gut-brain axis (GBA), a bi-directional signaling pathway between the gastrointestinal tract and the brain. Recent advancements have begun to investigate new avenues for the pathophysiology of MDD, highlighting the potential for microbial therapeutics as an alternative intervention. Previous research studying the role of gut microbiota on psychiatric disorders suggests treatments that aim to repopulate the gut microbiome, such as fecal microbiota transplantation, may contribute to addressing depressive symptomatology. This study explores the application of an alternative to fecal microbiota transplantation, known as Microbial Ecosystem Therapeutic (MET-2). MET-2 is a novel microbial-based intervention composed of 40 strains of

lyophilized bacteria purified from a healthy donor. This study aims to identify if changes in inflammatory markers (C-reactive protein, transforming growth factor- β , interleukin-6, and interleukin-10) levels following microbial treatment, can predict long-term clinical response status post-treatment. In this study, MET-2 was orally administered for 6 weeks to participants aged 18-45 years diagnosed with MDD. Clinical response was measured using the Montgomery-Asberg Depression Rating Scale and molecular assessments were conducted through blood sampling. Data will be analyzed to determine if changes in inflammation, if any, following microbial treatment can predict long-term depressive remission and responder status. Given the connection between the gut microbiome and MDD, it is hypothesized that participants who have a beneficial change in inflammation are more likely to achieve and/or maintain clinical response status post-treatment. This study highlights the importance of innovative treatments that target the underlying biological pathways of MDD, suggesting that further research into microbial therapeutics holds the potential for developing more effective and personalized interventions for depression.

30. Sex in chronic stress models: How sex differences impact the effect of ketamine in animal models of depression presented by Veronica Sinclair, undergraduate student at Carleton University

Major depressive disorder (MDD) is a debilitating illness with high prevalence worldwide. It is known that sexual dimorphisms are present in this disorder, but the mechanisms underlying these differences are currently unclear. Ketamine, an N-methyl-D-aspartate receptor antagonist being investigated as treatment for MDD, has been previously shown to mediate antidepressant effects through inhibition of translational regulators, including eukaryotic initiation factor 4E-binding proteins (eIF4E-BPs) in male mice. This study used mice lacking 4E-BP1 and 4E-BP2 (Eif4ebp1/2 double knock out [DKO]) and their wildtype littermates in a 5-week chronic variable stress (CVS) mouse model to determine the effects of sex on eIF4E-BP activity and ketamine response. Stressors included cage tilting, 15-minute restraint, wet bedding, no bedding, 10-minute forced swim, exposure to a predator odour (rat cage bedding), and altered light/dark cycle. The last stressor for both sexes was an overnight stressor and all mice received a ketamine (IP, 0.3 mg/kg) injection immediately after being returned to their normal cages that morning. They were then observed two hours later during a splash test, as well as 24, 25, and 48 hours later in open field, elevated plus, and Y maze tests respectively. Wildtype males consistently displayed a significant response to ketamine, however females showed little to no response to either stress or ketamine in most measures. These results suggest that differing biological mechanisms such as the role of sex hormones may play a role in the pathogenesis of depression and response to ketamine, particularly in mediating activity of 4E-BPs.

31. Validation of a transgenic mouse model expressing GFP in motor neurons for the study of peripheral nerve injury presented by Michela Fortner, graduate student at Queen's University

Peripheral nerve injury (PNI) is a significant clinical problem that often leads to functional deficits, despite advances in surgical repair¹⁻³. While peripheral nerves possess the capacity to regenerate, successful reinnervation is dependent upon a growth-supportive environment and is limited by factors such as gap length and prolonged axotomy^{2,3}. In order to develop clinical strategies for optimizing recovery, the molecular factors which antagonize regeneration must first be thoroughly characterized. Unfortunately, the reliability and reproducibility of data generated using conventional tissue visualization techniques is affected by many interacting factors and thus is of variable quality, in addition to being time- and resource intensive⁴⁻⁶. Recently, transgenic fluorescent reporter models have generated interest regarding their utility for visualizing regenerating nerves⁷⁻¹⁰. However, no models which specifically label motor fibres have been validated for the study of PNI.

Here, we aim to validate the use of a transgenic mouse model expressing a GFP under the control of the motoneuron-specific *Mnx1* promoter. We hypothesize that motor nerves will uniquely and robustly express the GFP. Further, we anticipate that the transgene will have no off-target effects on any of the assessed metrics of regeneration – both morphological and functional – nor on the myelination of peripheral axons.

Male C57Bl/6 and *Mnx1*-GFP mice will undergo transection and immediate microsurgical repair of the right median nerve. Every 3d post-surgery, volitional grip strength will be assessed. On D28, the nerve will be transected 4mm distal to the original repair site. Fluorogold neurotracer will be applied to the proximal stump for retrograde labelling, and allowed to incubate for 1 week. Following this, mice will be euthanized and spinal cord and DRG (C5-T1) tissue collected. Histomorphometry of the median nerve, confocal imaging of the spinal cord, and immunofluorescence imaging of DRGs will be performed for morphological analysis.

32. Predicting fertility treatment outcomes using EEG and sLORETA to measure activations of the insula and anterior cingulate cortex presented by Joselle Solarino, graduate student at Wilfrid Laurier University

Background: Infertility is a devastating condition affecting one in six individuals, leading many women to pursue fertility treatments such as in vitro fertilization (IVF), a process where an egg is fertilized in a controlled environment outside the body. Research has shown that the anterior cingulate cortex (ACC) and insula are key brain regions linked to uterine contractility, with functional MRI studies on labour and orgasms indicating their role in regulating uterine contractions, potentially influencing embryo implantation and IVF success.

Objectives: To address failed embryo implantation in IVF, I propose to: (1) investigate the relationship between ACC and insula activity and uterine contractility using electroencephalography (EEG) and standardized low-resolution electromagnetic tomography analysis (sLORETA), (2) analyze how differences in brain and uterine activity influence embryo implantation, and (3) develop a predictive model for successful implantation based on observed neural and uterine patterns.

Methods: Patients from Markham Fertility Centre undergoing a frozen embryo transfer (FET) with genetically screened euploid embryos will be recruited. EEG will measure insula and ACC activity, while sLORETA will localize brain activity sources. Transvaginal ultrasounds will record uterine contractility, and participants will complete stress and anxiety questionnaires pre and post-transfer. Data from pre and post-FET EEGs will assess changes in brain activity.

Data Collection: Preliminary data has been collected from four (n=4) patients, and recruitment is ongoing to ensure a sufficient sample size.

Significance: This research could advance reproductive medicine by identifying non-invasive, brain-based markers of implantation success, enabling early interventions to optimize outcomes. Predicting outcomes using brain activity could reduce unsuccessful IVF cycles and improve patients' overall experience.

33. Minority Stress, Resilience, Mental Health, and Well-Being of Racial and Ethnic Minority Canadian Medical Students presented by Aksaya Ghetheeswaran, graduate student at McMaster University

The Canadian Federation of Medical Students recently raised awareness that Canadian medical students (MSs) may experience poor mental health over the course of their education, where psychological distress may contribute to higher drop-out rates, reduced empathy, increased medical errors, and suicidal ideation (Maser et al., 2019; King et al., 2021). Racialized MSs often report higher levels of adverse mental health compared to their White counterparts (Morgan & Fortier, 2019). Despite Canada's multicultural environment, however, little is known about differences in adverse mental health experiences among MSs from varying racial and ethnic backgrounds. Here, minority stress, defined as

chronic psychological stress associated with belonging to a stigmatized social group (Rostosky & Riggle, 2017; Meyer, 1995; Meyer, 2003), may contribute to adverse mental health outcomes among equity-denied MSs.

The present study aims to identify whether higher levels of minority stress are associated with more severe adverse mental health outcomes and alterations in neuropsychological functioning (e.g., selective and sustained attention) among Canadian MSs of various racial and ethnic backgrounds. It is predicted that regression analyses will reveal that elevated scores of minority stress will be associated with an increased severity of experienced mental health outcomes such as anxiety, depression, posttraumatic stress disorder, and moral injury. Mediation moderation analyses will also be conducted to better identify risk and resiliency factors for the present associations between minority stress, mental health adversity and neuropsychological functioning. Addressing such differences in mental health experiences, as well as risk and resiliency factors, will encourage medical institutions to implement better support systems to improve the well-being of at-risk MS populations. Supporting MS well-being in medical school can help prevent burn-out and drop-out rates later in their careers. Thus, the current project has the potential to help alleviate the ongoing staffing shortages within the Canadian healthcare workforce in the long term through the upstream approach of targeting MSs.

34. Evaluating the efficacy of an application-based cognitive training intervention for ADHD and problematic internet use in adults: A randomized, placebo-controlled trial presented by Alyssa Swiderski, graduate student at McMaster University

Background: Attention deficit hyperactivity disorder (ADHD) is a highly prevalent neurodevelopmental condition characterized by persistent patterns of inattention and/or hyperactivity-impulsivity, often accompanied by an increased need for immediate gratification.¹ Engagement in online activities addresses this need creating a feedback loop that reinforces both problematic internet use (PIU) and ADHD symptoms, leading to psychological distress and reduced quality of life.^{1,2} Gamified therapeutic interventions hold promise to improve cognitive performance and ADHD symptoms via strengthening neural pathways related to executive functioning, and instant feedback mechanisms that sustain engagement.³ ReadON is a novel digital cognitive training program employing gamified tasks and leveraging principles of neuroplasticity and machine learning algorithms, to target individual cognitive skill deficits.⁴ Preliminary evidence supports its efficacy in improving working memory, and executive functioning, common deficits in ADHD.⁴ Digital therapeutics show potential as adjuncts to pharmacotherapy, but their effects on broader ADHD symptomology and comorbid PIU remain unclear.

Objective/Hypothesis: This study aims to evaluate the safety, tolerability, and efficacy of 10 weeks of ReadON digital cognitive training, as an adjunct to pharmacotherapy, in reducing ADHD symptomology and PIU in adults with ADHD of moderate-severe severity. Methods: In an 11-week randomized placebo-controlled clinical trial, adults with ADHD receiving pharmacotherapy will be randomized to receive either ReadON (n = 21) or placebo (n = 21). The active treatment group will use the ReadON software 3 times weekly for 10 weeks, while the control group will engage in a gamified application lacking cognitive training components. Primary outcomes include safety and tolerability (incidence of adverse effects and dropout rates); Secondary outcomes include changes in ADHD severity (Adult ADHD Self Report Scale) and PIU (Compulsive Internet Use Scale).

Implications: Findings may inform larger trials and influence tailored interventions to enhance cognitive and behavioural functioning in adults with ADHD.

35. The impact of somatosensory cues on cybersickness and postural stability during intense virtual reality gameplay presented by Megan Goar, graduate student at University of Waterloo

Virtual reality (VR) offers immersive experiences through electronic visual and auditory displays. While VR has applications in entertainment, training, and rehabilitation, its widespread adoption is limited by

cybersickness, a condition characterized by symptoms such as nausea, headaches, and disorientation. Cybersickness is thought to result from conflicting sensory information about balance and orientation relative to gravity, with highly sickening games presenting complex visual motion cues that conflict with vestibular and proprioceptive inputs. This sensory conflict not only induces cybersickness but also affects postural control. The somatosensory system is critical for maintaining standing posture, particularly through proprioceptive inputs from the legs, which are highly sensitive to postural sway occurring at the ankles. This study investigates the relationship between somatosensory feedback, cybersickness, and postural stability. Participants (n=20) completed four randomized trials, all playing an intense VR game (Adr1ft), but with different stance conditions that altered proprioceptive input: sitting, standing on a hard surface, standing on high-density foam, and standing on low-density foam. Cybersickness was assessed during each trial using the Fast Motion Sickness Scale at one-minute intervals for up to 15 minutes, with slope and curve tendencies analyzed to characterize its progression. Postural stability was evaluated using center of pressure (CoP) metrics, including root mean square, sway path length, and mean power frequency, collected over 20 seconds pre- and post-trial. It is hypothesized that reducing somatosensory information about whole-body orientation relative to gravity will increase the rate of cybersickness intensity and lead to greater CoP displacements. These findings could improve understanding of the relationship between conflicting sensory cues, cybersickness progression and postural stability. This may inform interventions to improve cybersickness, especially those targeting somatosensory feedback in VR environments.

36. Cockayne Syndrome A in a 6-year-old girl born to consanguineous parents: A case report presented by Razia Afroj, undergraduate student at Dhaka Medical College and Hospital

Introduction: Cockayne Syndrome (CS) is a rare autosomal recessive disorder caused by mutations in genes involved in DNA repair, primarily ERCC8 (Cockayne Syndrome A) and ERCC6 (Cockayne Syndrome B). These mutations affect tissues with high metabolic activity, such as the brain and skin, leading to progressive neurodegeneration, growth failure, and multisystem involvement.

Case Presentation: A 6-year-old South Asian female, born to consanguineous young parents, presented with global developmental delay, growth failure, and neurological deficits. She first presented at 2 years of age with concerns of gross motor delay, poor growth, low appetite, and microcephaly. Developmental milestones were delayed, with a social smile at 8 months, standing at 25 months, and unsteady walking at 30 months. Physical examination revealed short stature, weight below the 3rd percentile, microcephaly, spasticity of the lower limbs, dental caries, tooth decay, and severe dry skin without photosensitivity. Speech delay was noted, with the patient able to form only two-word sentences. Brain MRI showed diffuse hypomyelination and cerebral atrophy. Genetic analysis identified a probable homozygous variant of uncertain significance (VUS) deletion on chromosome 5q12.1 involving exon 2 of the ERCC8 gene, consistent with CSA. The patient has been receiving physical and speech therapy, along with dental and skin care since the age of 2, which initially improved her condition. However, her ability to walk has deteriorated over time. Vision and hearing assessments remain normal.

Conclusion: This case underscores the progressive nature of Cockayne Syndrome A, emphasizing the importance of early diagnosis and multidisciplinary management to address developmental and neurological challenges. Early genetic counseling and therapeutic interventions are essential for improving outcomes, especially in consanguineous populations.

37. Understanding cybersickness in virtual reality: The role of sensory reweighting, age, and quantified movement presented by Fira Gulifeire, graduate student at University of Waterloo

Cybersickness (CS) in Virtual Reality (VR) is a form of motion sickness that causes dizziness, nausea, and discomfort, potentially hindering VR's adoption in various fields. Previous research has shown varied CS

levels across age groups, but the underlying mechanisms remain unclear. This study investigates the relationship between CS, sensory reweighting ability, age, and movement patterns, building upon recent work by Chung and Barnett-Cowan (2023) on sensory reweighting in VR and Dilanchian et al. (2021) on age-related differences in VR interactions. We hypothesize that: 1. Individuals who effectively reweight sensory inputs, including downweighting visual and bodily information relative to gravity, will experience fewer CS symptoms. 2. Older adults, due to differences in movement patterns and balance control, will exhibit greater sensory mismatch and more pronounced CS symptoms, with movement sway and Center of Mass (CoM) velocity displacement correlating positively with sickness severity across age groups. Participants across different age groups will undergo VR exposure while their movement is captured using a Qualisys markerless motion capture system. Sensory reweighting ability will be assessed by measuring perceptual upright before and after VR exposure. Kinematic data will be analyzed using Theia 3D and Visual 3D software to examine relationships between movement patterns, CS severity, and sensory reweighting ability. This study aims to contribute to our understanding of age-related differences in CS susceptibility and their underlying mechanisms. The findings may inform future research on VR design and usability across different age groups, potentially improving VR applications in fields such as healthcare and rehabilitation.

38. Uncovering the temporal dynamics of autism stigma: An EEG investigation

presented by Natalia Van Esch, undergraduate student at Wilfrid Laurier University

Social neuroscience research on stigma is limited, with most studies focusing on race or gender biases (Amodio & Cikara, 2021), and little work addressing autism stigma. While previous studies have investigated brain regions associated with stigma (Harris & Fiske, 2006; Amodio & Cikara, 2021; Forbes, 2024), there is a lack of research on the temporal dynamics of these processes. Event-related potentials (ERPs) obtained via electroencephalogram (EEG) offer a promising approach to address this gap. Studies on race and gender bias have revealed that early components such as the N100 (associated with early orienting and attentional processes) and P200 (associated with goal-directed attention and motivation) are sensitive to implicit bias (Amodio, 2010). These findings suggest that ERPs can reveal biases that occur before conscious awareness, making them an ideal tool for exploring the temporal aspects of autism stigma (Amodio & Cikara, 2021; Forbes, 2024).

This study aims to examine ERPs produced during the presentation of autistic and non-autistic individuals in still images. EEG data will be analyzed to assess the N100, P200, and other relevant components (such as P300 and N400; Amodio & Cikara, 2021; Mahieux et al., 2024) associated with social categorization, comparing ERPs between autistic and non-autistic stimuli. We expect to see larger ERP amplitudes for autistic stimuli compared to non-autistic, especially for N100 and P200 components indicating participants view autistic individuals as an outgroup (Amodio, 2010). We will also run exploratory analyses aiming to identify which late ERP components are related to stigma. These findings aim to uncover the temporal dynamics of stigma and propose ERP measures as a more objective method of quantifying autism stigma, reducing the influence of various confounds, such as social desirability bias, seen in current stigma research.

39. The glymphatic system and amyloid beta assessed simultaneously in Alzheimer's Disease using PET/MRI presented by Lisa Hebert, graduate student at Carleton University

Alzheimer's Disease (AD) is a progressive neurodegenerative disease with no known cure, comprising up to 70% of dementia cases. Over 733,000 Canadians are currently living with AD or other dementias. That number is estimated to reach 1 million by 2030, placing an even greater burden on caregivers, the healthcare system and long-term care facilities. One of the hallmarks of AD is a pathological accumulation of amyloid beta (A β) protein in the brain. The glymphatic system, discovered in 2012, is a brain clearing process which removes metabolic and toxic waste buildup, including A β . This process

occurs primarily during sleep. With sleep disturbances a risk factor for, and a symptom of AD, it follows that inadequate sleep would lead to insufficient clearance. Animal studies have provided evidence of this phenomenon, however, there are currently no accepted non-invasive methods to safely assess the glymphatic system in humans. Combining several Magnetic Resonance Imaging (MRI) techniques and correlating them to A β deposition using Positron Emission Tomography (PET) will show the unique dimensions and a more complete picture of this clearance pathway. Saliva samples are also collected to quantify A β concentration using a validated non-invasive biosensor, capable of indicating the earliest stages of AD or Parkinson's Disease. This study aims to determine which non-invasive imaging combinations will sufficiently assess the health of the glymphatic system in humans and whether its impairment correlates to A β burden, symptomatology and sleep disturbances in AD. To date, no research has simultaneously combined these various methods to study the glymphatic system in AD. Determining how sleep-related mechanisms clear the human brain may point to possible pharmaceutical targets or sleep therapies. Research on preventing or slowing the progression of dementia is essential to allow our expanding elder population the autonomy to age-in-place for longer, and curb caregiver and healthcare system burdens.

40. A two-hit hypothesis of anxiety expression in the rat, using the shock-probe burying test presented by Jessica Wee, undergraduate student at Queen's University

Early life adversities (ELA) are linked to a heightened risk of developing psychopathologies, like depression and anxiety. However, not all individuals exposed to ELA experience mental health issues, thus, indicating the importance of understanding the mechanisms behind such variability. The Two-Hit Hypothesis suggests that early life stress (first hit) sensitizes the response to later stressors (second hit), such that even a mild stressor could trigger psychopathology, and further, that the second hit of stress is necessary for psychopathology to manifest. This proposal aims to further explore the two-hit hypothesis in an animal model by exposing rats to the Limited Bedding and Nesting (LBN) paradigm, during the neonatal period, followed by subthreshold chronic mild stress (CMS) during adolescence. Thus, there will be 4 groups; neonatal control (nCON)/adolescent control (aCON), nCON/CMS, LBN/aCON, LBN/CMS (n =10/group). The rats will be tested in the Shock-Probe Burying test, to measure their anxiety-like behaviour. I hypothesize that the LBN/CMS rats will display significant increases in anxiety-like behaviour compared to the no-stress controls (nCON/aCON), and both of the single-hit groups (nCON/CMS, LBN/aCON). Furthermore, I also expect that the two single-hit groups will not differ from each other or the no stress-controls. This study hopes to further understand the two-hit hypothesis and potentially inform better prevention and treatment strategies for anxiety and depression related to ELA.

41. Comparative systemic analysis between high intellectual potential and high functioning autism: A research study presented by Arani Hiritharan, undergraduate student at Queen's University

Introduction: Neurodivergence encompasses a range of conditions, including High Intellectual Potential (HIP) and High-Functioning Autism (HFA). These conditions, often viewed as contrasting ends of a spectrum, present diagnostic challenges due to overlapping traits, particularly in clinical, cognitive, and social domains. This systematic analysis explores the extent of their similarities to improve diagnostic accuracy and treatment outcomes.

Methods: A systematic review of peer-reviewed articles was conducted, focusing on diagnostic criteria, cognitive & psychometric factors, clinical & neurophysiological features, sensory modulation and etiology of HIP and HFA. Data sources included studies utilizing diagnostic tools such as DSM-5 and WISC-IV. Due to a lack of articles directly comparing HIP and HFA cross-referencing and cross-analysis of studies was used.

Results: HIP individuals demonstrated superior cognitive and psychometric functioning compared to those with HFA. When examining clinical features, both groups exhibited autistic traits, with HIP showing a lower prevalence than HFA. Clinical and neurophysiological evaluations also highlighted nuanced differences, supported by varying international diagnostic criteria. Etiological findings revealed common developmental influences, including atypical brain hemisphere symmetry and fetal testosterone exposure. Both groups shared sensory modulation challenges.

Conclusion: HIP and HFA exhibit significant similarities across several domains, but nuanced differences and the scarcity of direct comparative studies prevent a definitive conclusion regarding their convergence. Misdiagnosis risks highlight the need for precise diagnostic criteria and standardized assessments. Future research must directly compare these populations with diverse demographics to enhance operationalization and construct validity. Standardized global diagnostic frameworks is a necessity to enhance accuracy and reduce diagnostic bias.

42. Exploring the relationship between decreased sound tolerance and social profiles presented by Ashleigh Wickie, undergraduate student at Wilfred Laurier University

Humans are inherently social creatures, yet considerable variability exists in our social behaviours. It is unclear what factors contribute to this variability. Given the complex and abundant sensory stimuli present in our daily environments, differences in sensory processing abilities may contribute to the variation observed in social behaviours. Individual differences in sensory processing may have significant effects on an individual's capacity to navigate social settings and may influence the development and expression of social competence. In this study, we investigated the relationship between one form of sensory processing difference, Decreased Sound Tolerance (DST), and social competence. Existing literature suggests that it is common for individuals with DST to engage in social avoidance behaviours to mitigate exposure to distressing sounds. However, limited research explores the potential relationship between DST severity and social competence. As such, a sample of 2095 undergraduate students completed an online survey designed to assess their DST severity and social competence. Initially, to parse the variability in social competence, scores on the multidimensional social competence scale (MSCS), underwent a k-means cluster analysis. This analysis yielded four unique social profiles based on seven social competence domains (e.g., social motivation, emotion regulation etc.). Misophonia and hyperacusis questionnaires were then used to evaluate differences in DST across the social profiles. The results indicated varying severity levels of both misophonia and hyperacusis across the four social profiles, with the individuals who reported the highest social competence exhibiting the lowest levels of DST. These findings highlight the potential relationship between sensory processing differences, such as DST, and social functioning.

43. The impact of a murine coronavirus (MHV-JHM) upon alpha-synuclein and inflammatory factors in primary wild-type and LRRK2 G2019S mutant microglia and midbrain neuronal cultures presented by Stephanie Hobbs, graduate student at Carleton University

Parkinson's Disease (PD) is characterized by a loss of midbrain dopamine neurons and the accumulation of aggregates of oligomeric and fibril forms of the alpha-synuclein protein. A multi-hit hypothesis points to an interaction between genetic and multiple environmental risk factors in the cause of the disease. Much evidence has indicated that mutations in the inflammatory gene, leucine rich repeat 2 (LRRK2), is critically linked to PD. Moreover, viral infection may play a role as an environmental trigger and may do so by augmenting the pro-inflammatory consequences of LRRK2. The present study utilized primary midbrain microglia and neurons from wildtype and LRRK2-G2019S mutant mice. Murine Hepatitis Virus (MHV) was utilized as a model for coronavirus infection and real-time live cell imaging and immunobiological assessments used to assess changes in microglial morphology, microglia-neuron

interactions and alpha-synuclein aggregation in response to MHV. Thus far, we have found that MHV robustly infects midbrain dopamine neurons and microglia, leading to time-dependent neurodegeneration. The virus also caused microglial activation, increased motility, and resulted in cell fusion with the formation of complex syncytia networks. These effects were generally increased in the LRRK2 G2019S derived cells and the mutation appeared to catalyze the spread of alpha-synuclein. Our preliminary data indicate an importance for microglia and LRRK2 in coronaviral neurotoxicity and alpha-synucleinopathy, which has tremendous clinical implications.

44. The impact of traumatic brain injury on caudal fin regeneration in zebrafish presented by Makenna Wiebe, graduate student at Wilfrid Laurier University

Zebrafish are well known for their extraordinary regenerative capabilities, such as the ability to regenerate caudal fins within weeks and restoring neural tissue over several months, making them a valuable model for studying regeneration. While the local signaling pathways that drive regeneration at injury sites are well-characterized, the potential systemic influence of the brain on regenerative processes remains unexplored. In humans and other complex organisms, injuries often result in fibrosis (scarring) rather than regeneration, despite both processes being initiated by inflammation, a divergence attributed to differences in signalling regulation. In *Xenopus laevis* (African clawed frogs), which share developmental pathways with zebrafish, limb regeneration is limited but can be enhanced by drug treatments. Interestingly, drug-induced limb regeneration in *Xenopus* has been associated with increased expression of limb growth-related genes in the brain, suggesting a systemic role for the brain in regeneration. This study investigates how brain injuries influence caudal fin regeneration in zebrafish and explores changes in the expression and activity of key regenerative genes and their associated proteins in the brain. We hypothesize that zebrafish with concurrent mid-forebrain lesions and caudal fin amputations will exhibit reduced fin regrowth compared to controls and that brain expression of regeneration-associated genes will be higher in zebrafish with intact brains, indicating that the brain may play a supportive role in regeneration. Zebrafish will undergo caudal fin amputations and mid-forebrain lesions, with regrowth, gene expression (via qPCR), and protein activity (via immunohistochemistry) analyzed at 3 and 14 days post-injury. This study aims to elucidate the role of the brain in systemic regeneration mechanisms, providing a deeper understanding of the interactions between neural and peripheral systems during tissue repair, which could inform future research on enhancing regenerative outcomes in vertebrates

45. Investigating neural integrity in amyotrophic lateral sclerosis through the oculomotor system presented by Emmalee M. Hunter, undergraduate student at Queen's University

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease of both the upper and lower motor neurons, characterized by progressive weakness and muscle atrophy. Although historically considered a pure motor neuron disease, early and frequent impacts on cognitive and autonomic processes support the prevailing notion of ALS being a multisystem disorder. However, the neural basis of extra-motor involvement in ALS remains poorly understood. Pathological involvement in cortical and sub-cortical brain regions may precipitate as alterations in saccade, blink, and pupil behaviours, which are measurable via unstructured, video-based eye-tracking. In this study, 21 ALS patients recruited from the Ontario Neurodegenerative Disease Research Initiative and 34 age-matched healthy controls completed a free viewing task. Participants viewed a pseudo-randomized sequence of 10 one-minute movies featuring dynamic, complex scenes that change abruptly every 2-5 seconds. The participants viewed the clips with no specific task instructions while saccades, blinks, and pupil size were recorded. Data analysis focused on low-level oculomotor parameters throughout the task and responses to clip changes, which continuously introduce new visual stimuli and fluctuations in luminance levels. Preliminary analysis shows that ALS participants exhibited a higher blink rate and a stronger tendency to fixate on central

regions of the videos than controls, potentially impairing the acquisition of visual information from dynamic scenes. The ALS participants also exhibited a smaller baseline pupil size and weaker constriction and dilation responses than controls. These findings suggest the pathological involvement of extra-motor brain regions that modulate attention and autonomic control. Understanding the extra-motor features of ALS could enhance early diagnosis, improve patient stratification, and inform the development of targeted therapeutic strategies.

46. Correlating glioblastoma cell growth kinetics and stemness to patient outcomes presented by **Katelyn Wu, undergraduate student at Queen's University**

Glioblastoma is the most common and aggressive primary brain tumour in adults, with recurrence being virtually inevitable despite treatment with surgical resection and chemoradiotherapy. The timing of recurrence varies among individuals, and no standard of care exists for recurrent glioblastoma, which is associated with a poor prognosis. Current prognostic variables, including neurologic status, extent of surgical resection, biomarkers, and tumour characteristics such as location, involvement, and size, provide limited predictive accuracy. Furthermore, a significant quantitative relationship between the in vitro growth kinetics and stemness of glioblastoma with the timing of recurrence remains unexplored, highlighting the need for improved predictive measures. The purpose of this research project is to correlate the in vitro cell growth kinetics and stemness of patient-derived glioblastoma tumours with patient outcomes to improve prognostic accuracy by predicting the timing of recurrence, ultimately enhancing patient care. Patient-derived glioblastoma tumors will be analyzed over 14 days to investigate correlations between in vitro growth patterns, stemness markers, and patient outcomes. Tumors will be cultured in 12-well plates, and imaging will be conducted on days 2, 4, 6, 8, 10, 12, and 14 using 10X and 4X magnification to capture and analyze growing glioblastoma spheres. Red blood cell lysis will be performed on day 5. MATLAB image analysis will determine sphere volumes, specific growth rates, and doubling times. At each time point, spheres will also be collected, fixed, and stained for stemness markers, including CD133, Nestin, and SOX2. These findings will be compared to patient data, including the period of progression-free survival, treatment modalities, and tumor pathology, to identify correlations between in vitro glioblastoma sphere growth patterns and patient outcomes. Due to the significant variability in glioblastoma behaviour across patients, it is hypothesized that a correlation between in vitro glioblastoma cell growth kinetics, stemness, and patient outcomes can be identified.

47. Evaluating VR-integrated EEG neurofeedback to improve attention and focus on adults presented by **Adeel Haq, Allen Wu, Avery Fitzpatrick, & Jessica Sun,** undergraduate students at Queen's University

Background: Neurofeedback (NF), a brain-computer interface technology, allows individuals to regulate their brain activity using real-time auditory or visual feedback to improve cognitive functions such as attention. Traditional NF methodologies often utilize two-dimensional (2D) feedback mechanisms, which can lead to participant disengagement due to the repetitive and lengthy nature of the sessions. This study pioneers the use of a three-dimensional (3D) Virtual Reality (VR) integrated with EEG-based NF (VR-EEG-NF) to enhance participant engagement and their cognitive benefits. Specifically, the goal is to evaluate the efficacy of VR-EEG-NF in stabilizing the beta-theta ratio (BTR), a biomarker of attentional control, to improve attention and concentration in adults while addressing the engagement limitations of traditional 2D NF approaches.

Methods: Employing a single-blind, randomized, sham-controlled design, this study evaluates the efficacy of VR-EEG-NF in enhancing the beta-theta ratio (BTR). Participants are healthy adults who are randomized to receive either authentic NF or sham feedback. The intervention comprises five weekly sessions lasting 15 minutes each, wherein participants engage in a VR game tailored to train and assess their ability to modulate their BTR in response to cognitive demands. Real-time EEG data are captured using the Galea

biosensing headset integrated with Varjo VR goggles, which monitor EEG, EOG, EDA, and PPG signals. The primary outcome is the alteration in BTR from baseline to post-intervention, assessed during and immediately after each session. Secondary outcomes include changes in Stroop test scores to measure improvements in attention and executive functioning. The effectiveness of VR-EEG-NF and participant responsiveness are analyzed using a mixed-effects model, which accounts for intra-subject variability and the structured nature of the repeated measures. Ethical approval was secured from the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board, with all study participants providing informed consent prior to participation.

Results: To be completed.

48. The role of neural oscillations in fundamental cognitive processes: A literature review presented by Harini Saravanakumar, undergraduate student at Queen's University

Neural oscillations, synchronized rhythmic electrical activity within neuronal circuits, are increasingly recognized as fundamental to cognitive processes, including attention and memory formation. Existing literature demonstrates the significance of theta (4–8 Hz), alpha (8–12 Hz), beta (13–30 Hz), and gamma (30–100 Hz) oscillatory bands, which coordinate communication across brain regions (Ward, 2003). Theta oscillations, predominantly generated in the hippocampus and prefrontal cortex, facilitate memory encoding and retrieval. Gamma oscillations, linked to fast-spiking interneurons, enhance sensory integration. Alpha rhythms regulate inhibitory control over cortical processing, and beta oscillations mediate cognitive stability (Neural Oscillation - an Overview | ScienceDirect Topics, n.d.). Cross-frequency coupling, such as theta-gamma phase-amplitude coupling, illustrates how these bands interact to synchronize local and global neural networks during complex tasks (Belluscio et al., 2012). Despite its importance, the mechanisms by which these oscillations influence brain connectivity remain poorly understood as studies diverge in their conclusions regarding their functional implications, suggesting the need for more refined research to pinpoint their complexities. An in-depth evaluation reveals several limitations in current research, including inconsistent definitions of oscillatory states and variability in brain region specificity. Additionally, many studies focus predominantly on resting-state activity, neglecting task-driven oscillatory dynamics that may better reflect real-world cognitive demands (Uhlhaas & Singer, 2010). Emerging evidence points to novel avenues for research, a particularly promising one being the role of oscillations in neuroplasticity and their potential modulation by non-invasive brain stimulation techniques. Furthermore, there remains a significant gap in understanding how oscillatory disruptions contribute to neuropsychiatric disorders such as schizophrenia and Alzheimer's disease (Basar, 2013). Future research should prioritize longitudinal studies integrating electrophysiological data to better characterize oscillatory mechanisms across the human lifespan (Ruggeri et al., 2022). Approaches combining machine learning and multi-scale neuroimaging are also crucial for better mapping oscillatory dynamics and uncovering their overall roles in cognition (Singh et al., 2022). Advancing this understanding holds promise for novel interventions aimed at restoring cognitive function and treating neurological disorders.

Student Oral Presentation Abstracts

1. Altered social and depression-like behaviours support a two-hit model of stress in Long-Evans rats presented by Megan Babcock, graduate student at Queen's University

Early-life adversity (ELA) increases the risk of later-life psychopathology, however, not everyone who experiences ELA will develop psychopathology. Recent studies in our lab sought to examine a “two-hit” hypothesis of stress sensitivity. This hypothesis posits that a first hit of stress in early life sensitizes an individual to a later hit of stress, leading to adverse outcomes when combined. In these studies, rats were exposed to either the limited bedding and nesting (LBN) stress paradigm as a “first hit” of stress or served as a neonatal no-stress control (nCON) group. Half the offspring from each neonatal condition were exposed to adolescent subthreshold chronic mild stress (CMS) as the “second hit,” while the other half served as adolescent controls (aCON). In our first experiment, subjects (n=12 males/group) were tested in a forced-swim test (FST) and social interaction test (SIT). Here, offspring from the two-hit (LBN-CMS) group displayed greater immobility in the FST, as well as decreased social play and increased aggression in the SIT compared to the other three groups (nCON-aCON, nCON-CMS, LBN-aCON), which were indistinguishable from each other. These findings suggest that the two-hit animals are experiencing a depression-like response. Current analysis of the density of immunoreactive oxytocin-producing cells in the hypothalamus aims to examine potential stress-induced changes in the brain's oxytocin system. We have also initiated a second experiment using an expanded range of behavioural tests, as well as incorporating sex as a variable. Here, offspring (n=10 males/group and n=10 females/group) were tested in the FST, sucrose preference test, and splash test. Data analysis from the second study is ongoing but results indicate that offspring in the two-hit group show depression-like reductions in sucrose preference compared to the other three groups. The brains from this study will be assessed for oxytocin mRNA expression in the hypothalamus using qRT-PCR.

2. Investigating a novel treatment for spinal cord injury presented by Laura Camejo Espitia, graduate student at University of Western Ontario

Spinal cord injury (SCI) disrupts the connectivity between the brain and the spinal cord, leading to loss of motor, sensory, and autonomic function. Currently, there are no clinically proven therapies to improve spinal cord function after injury. Most SCIs are incomplete, leaving some axons undamaged. These spared axons occasionally sprout and synapse on targets that have lost innervation, leading to limited functional recovery. This reparative sprouting is restricted due to the presence of chondroitin sulphate glycosaminoglycans (CSPGs). Animal studies using Chondroitinase ABC, an enzyme that degrades CSPGs, have repeatedly shown improved functional recovery post-SCI. Unfortunately, the clinical translation of Chondroitinase is hampered by its instability and challenging route of administration. An alternative approach is to target Sox9, a transcription factor that upregulates CSPG levels in the injured spinal cord. Using computational and medicinal chemistry, we identified a lead candidate drug, TD874, that potently inhibits SOX9 activity and exhibits a superior pharmacokinetic profile. We will evaluate the effect of TD874 administration on Sox9 target gene expression and CSPG levels in the injured rat spinal cord. We will assess target gene expression at 5 days post-treatment and measure CSPG protein levels at 28 days following daily administration of TD874. We predict TD874 will have a dose-response effect on both Sox9 target gene expression and subsequent CSPG levels in injured spinal cords. These preclinical studies of TD874 set the stage for experiments to evaluate the therapeutic effectiveness of TD874 in small and large animal models of SCI. This project aims to develop a new clinically translatable therapeutic strategy to facilitate recovery following SCI.

3. Neurodevelopmental trajectory of anorexia nervosa: A longitudinal pan-Canadian multimodal imaging study presented by Rachel Dufour, graduate student at Concordia University

Introduction: Adolescence is a crucial developmental period for eating disorders, including anorexia nervosa. Recent research has sought to investigate biopsychosocial mechanisms at play in the development of EDs, such as brain processes. Previous research in people with anorexia nervosa suggests reduced gray and white matter volume and altered white matter connectivity, although results have been inconsistent. However, sample sizes are small, and few studies focus exclusively on young people. The current project aims to examine brain processes of anorexia nervosa in youth within a larger scope.

Methodology: As part of this pan-Canadian study, 235 young people with anorexia nervosa and 235 healthy controls (aged 12-25) are being recruited from nine specialized treatment centers, including in Kingston. Participants undergo magnetic resonance imaging (MRI) of different regions of interest at baseline and 12-month follow-up: dorsal prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex, insula, hippocampus, amygdala and caudate nucleus. Diffusion tensor imaging (DTI) is used to measure white matter connectivity. Eating-disorder symptoms are measured using an interview and questionnaires. General linear models will be used to analyse results.

Results: Recruitment began at the Douglas Institute (Montreal) and at the Kingston site. Harmonization (the process of matching the different MRIs) is underway at the other hospital sites. To date, 9 controls and 18 patients (6 Binge/purge type, 14 Restrictive type) have completed baseline data collection (mean age = 18 years, 93% female). Quality metrics indicated high contrast, high sensitivity, clarity, low noise and absence of artifacts. Preliminary results of structural anatomy and DTI will be presented at the conference.

Conclusion: This is the first large-scale MRI study in young people with anorexia nervosa. Results will help clarify associations between brain structure, connectivity, and relevant clinical characteristics in anorexia nervosa. A better understanding of these associations will inform treatment for anorexia nervosa.

4. Identification of oculomotor abnormalities in behaviour variant frontotemporal dementia presented by Daria Hinton, undergraduate student at Queen's University

Behavioural variant frontotemporal dementia (bvFTD) is a neurodegenerative disease defined by impairments in personality, emotion, social behaviour, and/or language. Despite being one of the leading causes of early-onset dementia, affecting over 55 million individuals worldwide, standardized diagnostic methods and predictive biomarkers are lacking. The overlapping clinical presentation between bvFTD and other neurodegenerative and psychiatric disorders often results in misdiagnosis, delaying treatment onset. Thus, there is an urgent need for an accurate, accessible, and inexpensive tool to enhance the early detection and differential diagnosis of bvFTD. This study aims to identify the oculomotor phenotype of individuals with bvFTD to guide eye-tracking as a potential biomarker for bvFTD and assist in diagnosis. 19 individuals with bvFTD (mean age = 67.7) and 31 healthy age- and sex-matched controls (mean age = 67.1) were recruited through the Ontario Neurodegenerative Disease Research Initiative. Participants completed a structured interleaved pro- and anti-saccade task (IPAST) and an unstructured free-viewing (FV) task, evaluating cognitive control, attention and brainstem premotor pathways. In the IPAST, a coloured fixation point appeared on the centre of the screen in front of the participant to instruct a pro-saccade (green) or anti-saccade (red) when an eccentric visual stimulus appeared. The FV task involved watching sequences of video clips (3-4 seconds in duration) containing scenes of nature, people, buildings, etc. Eye movements during both tasks were collected using the Eyelink 1000 plus eye-tracker. Preliminary data demonstrates that bvFTD patients performed worse on the anti-saccade trial and had a higher frequency of errors than controls. On the FV task, bvFTD patients had reduced mean saccade amplitude, smaller baseline pupil size, reduced pupil constriction and dilation, faster blink rates, and reduced blink duration compared to healthy controls. These results demonstrate how bvFTD-affected oculomotor pathways can perpetuate changes in eye behaviour, enabling eye-tracking to aid detection and diagnosis.

5. Delivery of scAAV9.coSLC6A8 for restoration of creatine transporter function in a creatine transporter deficient mouse model: A dosage study presented by Chiara Sawilla, graduate student at Queen's University

Cerebral creatine deficiency syndromes are inborn errors of creatine metabolism, impairing the synthesis and transport of creatine. Under normal physiological conditions creatine is synthesized endogenously and released into the blood stream to provide cellular energy. Due to creatine's hydrophilic nature, it is unable to cross the lipid membrane of cells, thus uptake is reliant on creatine transporters. Creatine transporter deficiency (CTD), an X-linked disorder caused by mutations in the SLC6A8 gene, impairs the Na⁺/Cl⁻ dependent creatine transporters. Clinically, CTD presents as intellectual disability, developmental delays, behavioural abnormalities, and speech and motor impairments. There are currently no effective treatments; creatine supplementation has shown limited success in restoring creatine levels in the brain of affected individuals. The delivery of a codon optimized SLC6A8 transgene via self complementary adeno-associated virus 9 (scAAV9.coSLC6A8) has been proposed as a treatment for CTD. Previous delivery of SLC6A8 transgene in a SLC6A8Y⁻ murine model showed increased creatine levels in the brain, a decrease in hyperactivity, and an increase in muscle strength. This study investigates the dose-response of scAAV9.coSLC6A8 in a Slc6a8Y⁻ murine model. Five cohorts (n=6 mice/cohort) received intrathecal lumbar injections of either vehicle or varying vector doses (3.5e11, 7.5e11, or 1.1e12 vg/mouse) at 6 weeks of age. Short term (13 weeks) and long term (24 weeks) outcomes were assessed through weight and grip strength testing, as well as biochemical analysis of tissues collected at endpoint. Long term cohorts additionally underwent hyperactivity analysis in digitally ventilated cages. Results demonstrated a dose-dependent increase in weight and grip strength at both timepoints. Mass spectrometry confirmed elevated creatine levels in both peripheral and central organs of treated mice. Additionally, all treated groups exhibited reduced hyperactivity compared to untreated knockout controls. These findings provide critical insight for potential clinical applications in CTD patients.

6. Reconsidering the relationship between negative symptoms, cognitive impairments, and depression in persons with schizophrenia presented by Supriya Bains, graduate student at McMaster University

Schizophrenia is a severe mental health disorder characterized by cognitive impairments and psychotic symptoms. While antipsychotic medications are effective in treating positive symptoms of psychosis (e.g., hallucinations) the treatment of negative symptoms (e.g., blunted affect, avolition, asociality, and anhedonia) continues to be an area of schizophrenia research. Negative symptoms are correlated in severity to cognitive impairments on a cross sectional basis (Harvey et al., 2006). The nature of their relationship remains unclear, with several possible explanations: causality, bidirectionality, shared pathways, or distinct pathways. This complexity is further compounded by the challenges of global and domain-specific perspectives on negative symptoms and cognition (Melillo et al., 2023). Interestingly, a meta-analysis confirmed a significant association between negative symptoms and depression (Edwards et al., 2019). The present study further explores the relationships between negative symptoms, depression and cognition using cognitive domains and depression as predictors of the severity of negative symptoms in persons with schizophrenia. Forty participants (M = 45.98, SD = 13.82, 27M and 13F) diagnosed with schizophrenia or schizoaffective disorder were enrolled from outpatient clinics at St. Joseph's Healthcare Hamilton. Participants completed measures of psychosis (PANSS), depression (DASS-21), and cognition (NIH Toolbox Cognition Battery: executive function, attention, processing speed, working memory, language, and verbal learning and memory). A linear regression model will be used to predict the variance in negative symptoms explained by depression and cognitive impairments, provided that the assumptions for a linear model are valid. Following this, a hierarchical regression will evaluate the incremental explanatory value of cognitive domains while controlling for depression. Higher-order cognitive domains, such as executive functioning and working memory, are likely to explain the

most variance in the severity of negative symptoms due to their role in goal-directed behaviour, social interactions, and emotional expression. A closer inspection of the relationship between negative symptoms, depression, cognitive domains will facilitate the development of targeted interventions for persons with schizophrenia.

7. The impact of a murine coronavirus (MHVJHM) upon alpha-synuclein and inflammatory factors in primary wild-type and LRRK2 G2019S mutant microglia and midbrain neuronal cultures presented by Stephanie Hobbs, graduate student at Carleton University

Parkinson's Disease (PD) is characterized by a loss of midbrain dopamine neurons and the accumulation of aggregates of oligomeric and fibril forms of the alpha-synuclein protein. A multi-hit hypothesis points to an interaction between genetic and multiple environmental risk factors in the cause of the disease. Much evidence has indicated that mutations in the inflammatory gene, leucine rich repeat 2 (LRRK2), is critically linked to PD. Moreover, viral infection may play a role as an environmental trigger and may do so by augmenting the pro-inflammatory consequences of LRRK2. The present study utilized primary midbrain microglia and neurons from wildtype and LRRK2-G2019S mutant mice. Murine Hepatitis Virus (MHV) was utilized as a model for coronavirus infection and real-time live cell imaging and immunobiological assessments used to assess changes in microglial morphology, microglia-neuron interactions and alpha-synuclein aggregation in response to MHV. Thus far, we have found that MHV robustly infects midbrain dopamine neurons and microglia, leading to time-dependent neurodegeneration. The virus also caused microglial activation, increased motility, and resulted in cell fusion with the formation of complex syncytia networks. These effects were generally increased in the LRRK2 G2019S derived cells and the mutation appeared to catalyze the spread of alpha-synuclein. Our preliminary data indicate an importance for microglia and LRRK2 in coronaviral neurotoxicity and alpha-synucleinopathy, which has tremendous clinical implications.

8. Ultraweak photon emission (UPE) neuroimaging device to read brain states: A proof-of-concept study presented by Dr. Victoria Hossack, Post Doc at Wilfrid Laurier University

Quantum sensing is a rapidly advancing field that can provide neuroscientists insights into neural communication that were never before possible. Ultraweak photon emissions (UPEs) are small particles of light that are coupled to the formation of reactive oxygen species (ROS) in the mitochondrial. UPEs may have a role in cognition as they travel along nerve fibers² and are modulated by neurotransmitters³. Similar to quantitative electroencephalography (qEEG), UPEs have a high temporal resolution compared with other neuroimaging tools (e.g. fMRI, PET). Currently we are developing a standardized neuroimaging tool that can measure photonic signalling and read real-time brain state dynamics. Here we present preliminary data as a proof-of-concept demonstration of our platform. We measured qEEG simultaneously with UPEs to confirm brain activation changes during an auditory task relative to the resting state. Signal processing analyses determined how qEEG and UPE neural signalling fluctuated in the time and frequency domains. Significant changes in neural activation in the temporal lobes during the auditory task were found in both qEEG and UPE measurements. Our results demonstrate that UPE neuroimaging presents a novel low cost, non-invasive tool to read brain states. Built on principles from quantum biology, our device has the potential to drive a paradigm shift in neuroscience which will revolutionize the way we understand brain function.

9. Improving the neuroD1-AAV-based gene therapy intracerebral injection protocol for optimal neuronal recovery presented by Golnar Taheri, graduate student at Queen's University

Despite medical advancements reducing stroke mortality, stroke remains the second leading cause of death globally. The increase in glial cells and neuronal death disrupts the neuron-to-glia ratio. Additionally, glial cells release neuroinhibitory factors, contributing to neurodegeneration. Recent studies highlight a novel gene therapy using ectopic expression of the transcription factor NeuroD1, which effectively re-balances the glial-neuronal ratio and restores motor function post stroke in rodent and non-human primate (NHP) models. However, there is ongoing debate about the best delivery protocol for this therapy. This study aims to find the optimal injection protocol for motor function recovery and enhanced neuronal transdifferentiation. A total of twelve NHPs underwent middle cerebral occlusion surgery. Fourteen days following the surgical intervention, the animals had NeuroD1-adenoviral-associated virus (AAV) administered using three distinct protocols: control, Protocol1 (P1), and Protocol2 (P2). Nine months after the initial stroke, the NHPs were euthanized, and brain tissue was harvested for analysis. The tissues were stained for neuronal (NEUN and MAP2) and glial (GFAP, IBA1) markers using immunofluorescence techniques. Cell counts were performed automatically using FIJI, and the data were analyzed with a one-way ANOVA, a post hoc Tukey test, and Prism 9 software. Our preliminary data shows that control animals had a significant decrease in the NeuN/GFAP ratio in the ipsilateral hemisphere compared to the contralateral hemisphere, which was restored to normal levels in both P1 and P2. Moreover, the significant increase in GFAP+ cells in the ipsilateral hemisphere was restored to normal levels in P1 and P2. These findings support the effectiveness of both injection protocols in returning the histological balance. More investigations are in progress regarding these neurons' functionality and development levels.

10. Exploring low-intensity pulsed ultrasound as a non-invasive strategy for medulloblastoma treatment presented by Tiffany Yu, undergraduate student at Queen's University

Medulloblastoma is the most common malignant pediatric brain tumor, representing approximately 20% of all brain tumors in children. Among its molecularly distinct subgroups, the Sonic Hedgehog (Shh) subgroup features overactive Shh signaling—a pathway critically regulated by the primary cilium. Conventional treatments, including surgical resection, chemotherapy, and radiation, often yield high morbidity rates and long-term neurocognitive impairments, underscoring the urgent need for more precise, less invasive strategies. Low-intensity pulsed ultrasound (LIPUS), a focused ultrasound modality delivering low-energy, targeted sonication, has demonstrated the ability to modulate cellular structures such as the primary cilium. Recent data in neuronal models indicate that LIPUS reduces both cilia length and incidence, suggesting a possible route to inhibit aberrant signaling cascades. Drawing upon this premise, our project investigates whether LIPUS can disrupt the overactive Shh pathway in medulloblastoma cells by mechanically stimulating the primary cilium. To establish proof of concept, we first demonstrate medulloblastoma cell responsiveness to known Hedgehog pathway inhibitors, Vismodegib and Ciliobrevin A. Our findings show dose-dependent reductions in Gli1 expression as measured by qPCR, confirming efficient disruption of Shh signaling. Immunocytochemistry further reveals alterations in cilia morphology and incidence, particularly in Ciliobrevin A-treated cells. Having validated these positive controls, we have designed and are constructing a specialized rig to deliver LIPUS to cultured medulloblastoma cells. We will then evaluate LIPUS-mediated changes in Gli1 expression and cilia architecture using qPCR and immunocytochemistry. By exploring mechanical ciliary modulation as a means to curb tumor growth, our study aims to introduce LIPUS as a novel, non-invasive adjunct to existing medulloblastoma therapies.^{10,11} This approach potentially addresses key limitations of current treatments, reducing both tumor burden and associated neurotoxicity.^{1,5} If successful, LIPUS-mediated ciliary disruption could offer a safer, more targeted therapeutic avenue for pediatric medulloblastoma and other cilia-dependent malignancies, ultimately paving the way for its translation into clinical applications.