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NURC
Neuroscience Undergraduate Research Conference

NURCxCJUR | Special Edition

Neuroscience Undergraduate Research Conference 2026

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NURC Neuroscience Undergraduate Research Conference

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Special Edition

May 2026

This issue is published on the traditional, ancestral, and unceded territory of the Coast Salish Nations, including xʷməθkʷəy̓əm (Musqueam), Sḵwx̱wú7mesh (Squamish), and səlilwətaɬ (Tsleil-Waututh).

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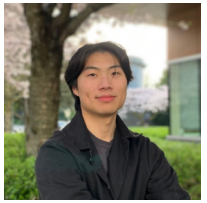

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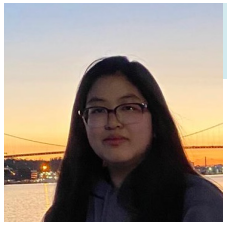

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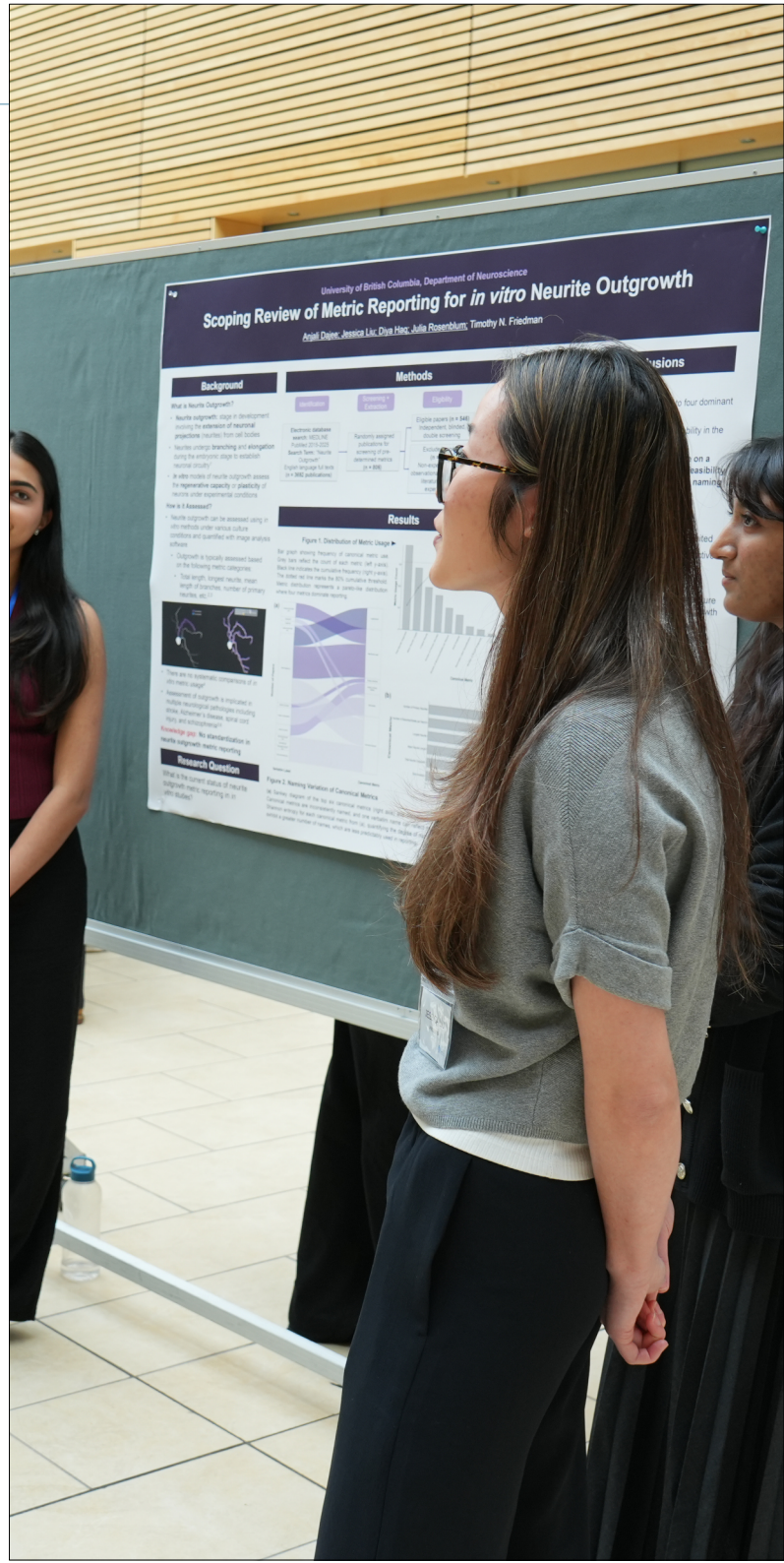
Neuroscience Undergraduate Research Conference 2026

The Neuroscience Undergraduate Research Conference (NURC) is an annual event at the University of British Columbia that brings together over 500 attendees, judges, panellists, and presenters to celebrate undergraduate research in our community. Organized by a dedicated team of students from the UBC Neuroscience Association, the conference highlights the curiosity, effort, and commitment behind each project.

This year marks the 10th Annual NURC, which took place on March 28th, 2026, hosted in the Life Sciences Institute at UBC. The conference featured a keynote from the UBC President, Dr. Benoit-Antoine Bacon, who was interviewed by esteemed neuropsychologist Dr. Hagar Goldberg. The conference featured a range of seminar panels highlighting student pathways into research, discussions on research ethics and academic opportunities, and insights into graduate school, medicine, and careers after graduation.

NURC would not be possible without the unwavering support of UBC's Undergraduate Program in Neuroscience, as well as our generous sponsors: Parkinson Society BC, Alzheimer Society of B.C, the Djavad Mowafaghian Centre for Brain Health (DMCBH), the Centre for Blood Research, Pacific Parkinson's Research Institute, Huntington Society of Canada, and the BC Epilepsy Society.



Plasma biomarkers and neuropathology as predictors of cognitive decline in aging populations

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Alzheimer's disease (AD) is characterized by progressive cognitive decline, where individuals differ in the rate of deterioration. Understanding how brain pathology and blood-based biomarkers relate to cognitive decline is important for improving prognosis. This study investigated individuals with dementia from the UBC Clinic for Alzheimer's and Related Dementias. Participants clinically diagnosed with dementia underwent a blood draw and repeated cognitive assessments, followed by autopsy-confirmed neuropathology evaluation. Plasma biomarkers were measured using the Quanterix single-molecule array (Simoa) platform. Cognitive performance was assessed using the Mini-Mental State Examination (MMSE), and participants were classified as rapid decliners (≥ 3 points/year) or non-rapid decliners (< 3 points/year). Neuropathological groups were defined based on the presence of Alzheimer's disease-related and other brain pathologies. Group comparisons and regression models were used to evaluate predictors of cognitive decline. Of the 42 participants, 20 were rapid decliners and 22 were non-rapid decliners. Plasma glial fibrillary acidic protein (GFAP) was significantly higher in non-rapid (*Mdn* = 255 pg/mL) than in rapid decliners (*Mdn* = 167 pg/mL; Mann-Whitney $p = .018$). Cognitive decline accelerated in later stages of the disease and differed based on neuropathological profile. Participants without AD pathology declined 7.53 MMSE points/year faster than those with AD and additional co-pathologies ($\beta = -7.53$, $p = .008$). Neuropathology burden, rather than biomarkers, was the stronger predictor of cognitive decline ($R^2 = .31$). Overall, these findings outline the role of neuropathology in the rate of cognitive decline and suggest that GFAP may be useful for identifying individuals at risk for rapid deterioration in late-stage dementia.

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Keywords: Alzheimer's disease, dementia, cognitive decline, neuropathology, plasma biomarkers, Simoa, GFAP, MMSE

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Impacts of neuroscience on Canadian involuntary admissions policy

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In 2017, the government of Canada recognized the growing opioid epidemic as a public health crisis, leading to increased political action surrounding substance use. One option explored, known as "involuntary admissions", typically refers to involuntarily detaining individuals suffering from a mental health crisis to an inpatient care facility, as a last resort effort to prevent significant harm to themselves or others. In 2024, the BC Mental Health Act was amended to include Substance Use Disorders (SUDs) as an applicable mental health diagnosis, allowing for involuntary admissions based on an SUD alone. In 2025, Alberta proposed similar legislation with the Compassionate Intervention Act, furthering the allowance of involuntary substance use care in Canada. This project aims to analyze these policies alongside the current neuroscience literature in mental health and substance use disorders to assess how neuroscience influences these policies. This was done in the form of a systemic literature review, encompassing legal and neuroscience sources alike. Furthermore, expert interviews were performed to examine how these policies are developed and understood. Interview responses underwent thematic analysis, with results showing a preference for voluntary over involuntary measures, a desire for scientists to be included in the policy-making process, and a need to balance the social determinants of substance use and scientific understanding within policy creation. This study is the first of its kind to evaluate Canadian involuntary admissions policies through a neuroscience lens, addressing how best to leverage the science behind SUDs to create informed policy and enable better treatment outcomes.

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Keywords: involuntary admissions, substance use, mental health policy

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Quantifying imagery in aphantasia

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Aphantasia refers to the absence of visual mental imagery, most often seen as a developmental disorder. Most often it is diagnosed by use of the 16-item vividness of visual imagery questionnaire (VVIQ), which asks the subject to rate the quality of their mental imagery. However, this test does not evaluate the accuracy of their visual imagery. Our goal was to measure the performance of aphantasic subjects on a forced-choice battery and to evaluate the hypothesis that the accuracy of their visual imagery was impaired. We evaluated 11 subjects, nine of which were recruited from the r/aphantasia subreddit. All scored well below the cutoff for aphantasia on the VVIQ, with a history of lifelong lack of imagery and no other neurological history. Their results were compared to those of 17 control subjects of similar age and gender. Subjects did a battery of nine imagery tests for object shape and size, colour and hue, letter and words, and famous faces. There were no significant differences between the aphantasic and control groups for any type of imagery. Also, the aphantasic subjects performed better than three subjects with congenital blindness, who have never been able to form visual representations. Aphantasic subjects have accurate representations of various visual object properties and can access them to answer questions. The results indicate a discordance between their poor qualitative impression of their imagery and their good ability to access mental images.

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Keywords: aphantasia, visual processing, neuroscience, ophthalmology, neurology, developmental disorder, visual memory, perception

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Developing adaptive machine learning models and integrating biomusic in a pediatric palliative care setting: A protocol paper

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Children with profound and multiple disabilities in pediatric palliative care often have limited means of communicating affective states, creating challenges for caregivers seeking to interpret comfort, distress, or engagement. Biomusic is an emerging technology that translates physiological signals such as heart rate, skin conductance, and skin temperature into real-time musical output, offering an intuitive channel for interpreting internal states. While biomusic has shown promise in clinical and caregiving contexts, its integration into pediatric palliative care requires careful attention to clinical workflow, caregiver practices, and ethical constraints. This protocol describes a two-part study designed to inform the development and implementation of an adaptive machine learning pipeline for biomusic in a pediatric hospice environment. First, an ethnographically informed observational phase will be conducted through volunteer shifts at Canuck Place Children's Hospice in Vancouver, BC to identify practical use cases, contextual constraints, and interaction patterns relevant to biomusic deployment. These observations will guide protocol design grounded in real-world care practices. Second, physiological data will be collected from healthy adult participants to develop and validate a supervised machine learning pipeline capable of classifying affective states (positive, neutral, negative) from autonomic signals. This pilot methodology supports feasibility testing of model development using standard physiological signal processing techniques and adaptive learning strategies to enable future personalization. Together, these phases aim to establish an ethically grounded and clinically informed framework for integrating biomusic into a pediatric palliative care centre. This protocol provides a replicable model for developing affective computing systems in sensitive healthcare settings while prioritizing contextual validity and caregiver usability.

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Keywords: biomusic, affective computing, physiological signals, minimally communicative persons, assistive technology

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The role of BRG1 in sensory axon growth and regeneration

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Long-distance axons in the spinal cord enable essential autonomic, motor, and sensory communication. While developing axons elongate robustly, mature neurons lose the ability to reinitiate growth after nervous system injury. This decline is partially driven by epigenetic changes that restrict axon growth programs during neuronal maturation, which limits the regeneration of severed axons following spinal cord injury. However, the specific epigenetic mechanisms that underpin the loss of axon growth capacity remain poorly understood. Brahma-related gene 1 (BRG1) is a chromatin remodeling factor involved in neuronal development and differentiation. BRG1 modifies gene expression by reshaping nucleosomes and coordinating enzymes that trimethylate lysine 27 of histone H3 (H3K27me3). Here, we examined whether BRG1 knockout promotes axon outgrowth in cell culture. Further, we evaluated the capacity of BRG1 knockout to promote axon regeneration after a spinal cord injury. Preliminary findings indicate that BRG1 may be an epigenetic regulator of axon growth in sensory neurons and suggest targeting chromatin remodeling pathways may enhance regeneration after spinal cord injury.

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Keywords: axon regeneration, epigenetics, chromatin-remodeling, spinal cord injury

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Evaluating buprenorphine/naloxone microdosing vs. standard dosing in emergency departments (EMED)

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The opioid epidemic remains a major public health crisis. Despite available opioid agonist therapy (OAT), barriers to access persist. Standard buprenorphine/naloxone inductions require active withdrawal, whereas microdosing inductions allow initiation without withdrawal. The Evaluating Microdosing in the Emergency Department (EMED) study is a randomized controlled trial in which Emergency Department (ED) patients receive either standard or microdosing take-home buprenorphine/naloxone kits. We hypothesize that microdosing may improve induction completion, improve retention on OAT, and reduce subsequent ED visits, hospitalizations, overdoses, and deaths. We screen patients with five presenting complaints, provider referral, or self-referral and enroll patients (≥ 18 years) with opioid use disorder (OUD) who are not on OAT, not in active withdrawal, or hospitalized. We confirm OUD using the Rapid Opioid Dependence Screen (RODS). We consent and randomize patients to standard or microdosing inductions and provide a corresponding five-day take-home kit. We attempt to follow-up for five days post-ED discharge. The study is recruiting across six EDs in Vancouver, Calgary, and Edmonton. As of February 27, 2026, we screened 36,997 patients, and 3,391 patients reported non-medical opioid use within the last 30 days, 1,607 screened RODS-positive, and 408 enrolled. Among enrolments, 304 were male and 104 female, with mean age of 38. We successfully contacted 112 patients during the five-day follow-up. The EMED study demonstrates that provision of take-home buprenorphine/naloxone standard and microdosing packages is feasible in EDs. Our data will inform whether ED-initiated buprenorphine/naloxone microdosing improves successful inductions and subsequent health outcomes for patients with OUD treated in EDs.

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Keywords: opioid addiction, substance use, buprenorphine/naloxone, drug overdose, micro-induction, emergency service hospital

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Effects of end-tidal carbon dioxide concentration on depth of anesthesia in children undergoing total intravenous anesthesia

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Carbon dioxide (CO_2) is a major end-product of metabolism and can be manipulated by anesthesiologists using controlled ventilation during general anesthesia. Elevated CO_2 levels (hypercapnia) are associated with increased sedation and selective pain suppression. Total intravenous anesthesia (TIVA) is a general anesthesia technique involving administration of propofol, which reduces brain activity by positively modulating $GABA_A$ receptors. However, the impact of varying CO_2 concentration on depth of anesthesia during TIVA has yet to be well-studied in a pediatric population. Propofol administration during TIVA can be guided by an electroencephalography-based monitor called the Bispectral Index (BIS), which indicates depth of anesthesia. This study aims to determine the effect of end-tidal CO_2 concentration ($etCO_2$) on the depth of anesthesia in children, measured by BIS. Following REB approval, patients aged 3–11 undergoing minimally stimulating procedures were recruited. Participants received a standardized TIVA anesthetic with a consistent blood concentration of propofol. BIS was recorded for three 15-minute $etCO_2$ levels: 30, 40, and 50 mmHg, assigned in random order. The relationship between BIS and $etCO_2$ will be examined using time series plots and generalized estimating equations. Data has been collected from 58 participants [34 male, median age 7.4 years] so far. The recruitment period is ongoing. A correlation between $etCO_2$ and BIS may inform anesthetic practice by optimizing $etCO_2$ levels during TIVA. This would benefit patients by potentially exposing them to less medication and reducing the chance of side effects, all the while reducing environmental impacts of drug wastage and disposables.

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Keywords: bispectral index, end-tidal CO_2, total intravenous anesthesia

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Optimizing estradiol measurement in rat serum for neuroendocrine studies: Assessing derivatization method efficiency

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17β-estradiol (E2) is the main circulating estrogen hormone that is also produced in the brain to modulate neural function and behaviour. Low circulating levels of E2 are associated with an increased risk of neurodegenerative and psychiatric disorders. In rodents, circulating E2 levels are low, making accurate detection challenging for neuroendocrine studies that require small, repeated blood samples. To enhance detection, E2 can be derivatized with 1,2-dimethylimidazole-4-sulfonyl chloride (DMIS) prior to quantification by mass spectrometry. However, current methods using 20 µL of serum often still yield undetectable E2 levels and preliminary data suggest that DMIS derivatization efficiency decreases at higher volumes. The objective of the current study was to determine whether increasing serum volume improves E2 detection or instead reduces DMIS derivatization efficiency. To address this, both derivatized and underivatized E2 was measured in increasing volumes of rat serum (20, 40, 60, 80, 100 µL), using liquid chromatography tandem mass spectrometry. Preliminary results reveal a decrease in derivatized E2 and an increase in underivatized E2 at higher volumes of serum, suggesting reduced DMIS derivatization efficiency. Further analysis may suggest that while modest increases in serum volume could improve E2 detection, excessive volumes may introduce additional matrix effects that compromise derivatization and quantification. Improved sensitivity in serum E2 measurements is important for understanding how circulating E2 levels may reflect brain E2 levels in specific regions, strengthening the relationship between endocrine states and neural outcomes. This study will inform strategies for detecting other low abundance analytes that influence brain function.

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Keywords: 17β-estradiol, DMIS derivatization, serum, matrix effects

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Impact of maternal and offspring sucrose consumption on exploratory and risk assessment behaviours in mothers and adult offspring in rats

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Sucrose (table sugar) is the most commonly added sugar and is frequently consumed at levels exceeding global health recommendations. In rats, maternal sucrose intake is not sufficient to alter anxiety-like behaviour in mothers. No studies have investigated the combination of maternal and offspring sucrose intakes in adult offspring. This study examined the effects of sucrose consumption on maternal and offspring exploratory and risk-taking behaviours, which are associated with anxiety in rats. Mother rats were fed either a control diet (CON) or a high-sucrose diet (HSD) for 16 weeks, including pregnancy and lactation. After weaning, offspring from each maternal diet group were subsequently fed CON or HSD, creating four offspring experimental groups (n = 10–12/diet group/sex). Mothers and adult offspring underwent Open Field (OF) and Elevated Plus Maze (EPM). Mothers and offspring underwent behaviour 5–8 days after offspring weaning and at postnatal day 56, respectively. Behavioural Observation Research Interactive Software (BORIS) was used to quantify exploratory and risk assessment behaviours (e.g., rearing, head dipping, ledge exploration). In mothers, there were no significant differences in exploratory and risk assessment behaviours between the diet groups, suggesting in no changes in anxiety. We are currently analyzing offspring behaviours. This study highlights the importance of considering both prenatal and postnatal dietary environments when examining the behavioural consequences of excessive sucrose consumption and will provide insight into vulnerable developmental period during which sucrose exposure shapes behavioural outcomes. These findings may inform future nutritional recommendations for sugar intake.

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Keywords: sucrose, anxiety, maternal diet, exploratory behaviour, risk-taking

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A genetic screen in Drosophila to identify immunoglobulin domain protein integrin ligands in peripheral glia

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Glial sheath formation around axons is a fundamental process required for the physiological function of neurons. In *Drosophila melanogaster*, integrins—receptors that link the extracellular matrix to the cytoskeleton—are essential for this process. Previous research established that the loss of integrins in the wrapping glia sublayer of peripheral nervous system (PNS) nerves leads to the collapse or total absence of the glial sheath; however, the specific ligands that activate these integrins remain unidentified. Based on evidence that Immunglobulin (Ig) domain proteins interact with integrins in other tissues, we hypothesized that specific Ig domain proteins mediate glial sheath formation in conjunction with integrins. To identify potential ligands, we cross-referenced proteins localized to glial sublayers with known Ig domain-containing proteins. We characterized the distribution of a subset of these candidate proteins within peripheral nerves using endogenous GFP-tagged protein lines. Initial imaging results indicated that Fascillin2 (Fas2) and Sidekick (Sdk) are promising candidates for integrin interactions, as they exhibit consistent and distinct expression patterns within the nerve. Current experiments are focused on determining the precise localization of Fas2 and Sdk relative to wrapping glia makers and utilizing RNAi to knockdown their expression. By assessing structural integrity of the glial sheath following knockdown, we aim to define its necessity in glial development. This study clarifies the molecular components required for nerve insulation and, given the high degree of conservation of these two proteins from insects to mammals, provides insight into the complex glial-axonal interactions that sustain nervous system function in all animals.

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Keywords: Drosophila, peripheral nervous system, glia, Ig-domain proteins, integrins

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Scoping review of metric reporting in in vitro neurite outgrowth assays

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Neurite outgrowth assays are widely used to assess the responses of neurons to experimental treatments like drugs or genetic perturbations. The impact of these treatments on the measurable growth response has direct application in development, plasticity, and regeneration studies. However, despite advances in imaging and analytical tools, there is no standardized framework for defining or reporting neurite outgrowth metrics. Ambiguous terminology and inconsistent definitions hinder comparability across studies, skewing our interpretation of progress in research. To address this issue, we conducted a scoping review to characterize metric usage and naming practices of *in vitro* neurite outgrowth studies published between 2015–2025. Using the search term "neurite outgrowth" in PubMed, we screened 806 publications and extracted data from 545 eligible papers using independent, blinded, and consensus screening. For each study, we recorded the verbatim metric name and mapped it to a canonical metric reflecting our assumed intended classification based on a predefined glossary. Approximately 80% of all metric reportings were confined to five dominant canonical metrics. At the same time, substantial semantic variability was observed: single canonical metrics were represented by many distinct names, and widely used metrics often lacked a dominant naming convention. Methods used for neurite outgrowth quantification often lacked transparency and left out key details of what they were measuring. Together, these findings reveal a field characterized by a shared set of common metrics but presented with inconsistent terminology. Standardized definitions and explicit reporting frameworks are needed to improve reproducibility, comparability, and cross-laboratory integration in neurite outgrowth research.

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Keywords: neurite outgrowth, in vitro, analytical methods, scoping review

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Development and optimization of an LC-MS/MS assay for quantification of DHEA conjugates in song sparrows

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Seasonal territorial aggression in male song sparrows is closely associated with elevated circulating testosterone during the breeding season. However, males remain highly aggressive during the nonbreeding season despite non-detectable testosterone levels. Dehydroepiandrosterone (DHEA), an adrenal steroid precursor that can be converted to testosterone in the brain, has been proposed as an alternative regulator of aggression. Traditional immunoassay studies have consistently reported high circulating DHEA concentrations during the nonbreeding season that correlate with aggressive behaviours. However, recent analyses using liquid chromatography-tandem mass spectrometry (LC-MS/MS) reveal dramatically lower concentrations of unconjugated DHEA. This discrepancy suggests that DHEA conjugates, such as DHEA-sulfate (DHEA-S) and DHEA-glucuronide (DHEA-G), may have contributed to previous immunoassay reports. To date, DHEA conjugates have not been reliably quantified in song sparrows, leaving the endocrine mechanisms of nonbreeding aggression unresolved. The present study aimed to develop and validate a sensitive and specific LC-MS/MS assay capable of quantifying DHEA-S and DHEA-G in song sparrow plasma. Steroid extraction was optimised to preserve conjugate integrity while maximising analytical sensitivity. The assay achieved a lower limit of quantification of 0.05 ng/ml. Method development was performed using pure standards of known concentration and validated in human serum, where DHEA-S was quantified but DHEA-G was not detectable. Together, these findings establish a methodological framework for reliable detection of DHEA conjugates. Future work will measure DHEA conjugates in plasma from breeding and nonbreeding male song sparrows under control conditions or following aggressive encounters to determine the role of DHEA conjugates in nonbreeding aggression.

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Keywords: steroid hormone, aggression, animal behaviour, songbird, liquid chromatography tandem-mass spectrometry, dehydroepiandrosterone, dehydroepiandrosterone-sulfate, dehydroepiandrosterone-glucuronide

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Bayesian analysis of the association between prolonged noise exposure and acoustic reflex threshold differences: Implications for cochlear synaptopathy

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Hearing loss currently affects 18% of the population worldwide. Hidden hearing loss (HHL) is less common, but managing it early may reduce the risk of dementia. HHL may be caused by permanent synapse damage; however, this condition fails to be detected by audiograms. This study proposed a diagnostic method—the Acoustic Reflex Threshold (ART) differences. We hypothesize that for participants exposed to prolonged loud noise, their ART threshold difference would be diminished. Bayesian analyses were employed to investigate the association. The primary outcome was the ART difference. The primary exposures were three groups: normal UBC students (NH), UBC Music students (students exposed to prolonged noise), and symptomatic participants (participants who complain of difficulty hearing; SYMP). The sample size contained 130 ears. A consistent pattern was observed in all models: the mean ART differences were highest for the normal UBC student group, followed by the UBC Music student group, and lowest for the symptomatic group, suggesting a diminished ART difference. NH was positively associated with ART differences (β = 1.42, 90% CrI [0.09, 2.73]), and SYMP was negatively associated with ART differences (β = −1.53, 90% CrI [−2.77, −0.30]). After adjusting for age, the effect became uncertain. Sensitivity analyses support these results. Age has an effect on the 2000 Hz ART difference; a similar pattern is displayed across groups, but the effect for noise is uncertain. These results suggest that the ART difference has the potential to be a candidate indicator for HHL upon future studies.

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Keywords: Bayesian analysis, hidden hearing Loss, acoustic reflex threshold, noise exposure, sensory neuroscience

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Comparing cognitive modes underlying pain experience and pain empathy

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Introduction: Pain empathy involves witnessing another individual experiencing pain and involves theory-of-mind processes. This study aimed to use functional magnetic resonance imaging (fMRI) to study the cognitive modes associated with experiencing pain and employing pain empathy.

Methods: An open-source empathic pain task was analyzed, whereby 54 participants were administered pressurized pain on their fingernail and then watched another individual experience the pain. This was crossed with auditory cues prompting future pain or not. Constrained principal component analysis for fMRI (fMRI-CPCA) was used to extract components from the fMRI data and task-induced blood-oxygen-level-dependent (BOLD) signal were analyzed.

Results: Auditory attention for response (AAR) mode activation was present only when the participant experienced pain stimulation following a pain cue. Response (RESP) mode activation was strong in both experiencing and observing pain stimulation conditions. In contrast, the default network (DM) showed a dissociation, activating when observing pain, but deactivating when experiencing pain, strongest when a pain stimulus was actually delivered.

Discussion: The findings show a dissociation of cognitive modes, whereby AAR activates when experiencing pain, RESP activates when experiencing and observing pain, and DM activates while observing others experiencing pain, but deactivates when experiencing pain. This confirms AAR as the pain network and DM involvement in engagement of theory-of-mind/mental projection. Further, this reflects RESP involvement specifically when responding to or imagining pain, even in a non-responding task.

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Keywords: pain, pain empathy, fMRI, fMRI-CPCA, cognitive modes

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Neural and learner-dependent evidence for word segmentation from 'rhythmic' perception and statistical learning

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Early word learning depends on extracting words from continuous speech, a process often attributed to statistical learning via tracking transitional probabilities (TPs) between syllables. Past literature has used electroencephalography (EEG) phase entrainment which shows enhanced neural responses at word-level frequencies after exposure to structured speech. Recent evidence suggests that rhythmic acoustic cues co-occurring with statistical regularities may be driving the segmentation effect and observed entrainment. This study aims to investigate this hypothesis by exposing adults to three different speech streams: (1) a statistically structured stream (high TPs between syllables within trisyllabic pseudo-words, and low TPs between word boundaries); (2) a non-statistical, but position-structured, rhythmic stream (even TPs across all syllabic transitions, but positional-constraints on each syllable); and, (3) an entirely random, baseline stream. Data will be collected via a Magstim EGI EEG, and entrainment will be quantified via inter-trial coherence for each stream. Participants will be asked to complete post-exposure segmentation tests to probe explicit segmentation performance. A working memory task (Forward Digit Span) and a rhythm-perception task will also be conducted to correlate entrainment values to verbal working memory and rhythmic perception. We predict neural entrainment to the syllable rate in all speech streams, and crucially, expect to see strong correlation between entrainment measures in both the rhythmic and statistically structured streams. Data collection is currently ongoing. This study will provide greater understanding of how the brain develops abilities to process structured speech, which has significant implications for clinical applications supporting developmental populations.

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Keywords: word segmentation, statistical learning, rhythm, neural entertainment, EEG

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Rewiring the bovine neuroenteric system: Transcriptomic divergence of the ruminant gut-brain axis

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The gut-brain axis (GBA) is a bidirectional communication system essential for coordinating physiological, immune, and neurobehavioral responses between the gut and the brain. However, its evolutionary divergence and physiological specializations in ruminants remain under-researched. This study explored the divergence of the ruminant GBA through a systematic literature review and bioinformatics approach to compare human and cattle genomes. Transcriptomics datasets were compared using *Bgee* expression data to verify genomic identifications and contrast the literature, exploring the tissue-specific expression patterns. While expression of central nervous system genes is highly conserved across mammals, significant divergence exists in digestive tissues between humans and ruminants, specifically in the ruminant forstomach (rumen, reticulum and omasum). The forestomach exhibits unique genetic "gains" for sensing microbial metabolites, notably indoles and short-chain fatty acids, within a specialized squamous epithelium that is optimized for absorption and fermentation rather than enzymatic digestion. In contrast, the abomasum ("true stomach") and intestines retain conserved immune, motility, and neurotransmitter pathways mirroring monogastric mammals. Understanding these specialized pathways is critical for addressing animal health and welfare. Specifically, microbial dysbiosis in the gut can trigger inflammatory cascades, such as NLRP3 inflammasomes, leading to neuroinflammation and systemic stress. Furthermore, identifying conserved pain-signalling mechanisms in the GBA, such as the PAR2-nociceptor axis, offers new avenues for managing pain during and after routine procedures like dehorning and castration. By integrating transcriptomic data with a physiological focus on the divergence of GBA genes in ruminants, this research provides a framework for identifying GBA tissue-specific molecular targets to improve livestock welfare.

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Keywords: gut-brain axis, ruminant physiology, comparative genomics, transcriptomics, neuroinflammation, microbial metabolites, dysbiosis, livestock welfare

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ASCME clinical trial: High-dose lisdexamfetamine and contingency management for methamphetamine use disorder

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Methamphetamine use disorder (MUD) has emerged as a major public health concern in Canada, yet no pharmacotherapies are currently approved for its treatment. Behavioural interventions, particularly contingency management (CM), demonstrate benefit, but treatment outcomes remain suboptimal. The Addition of high-dose Stimulant and Contingency Management Evaluation (ASCME) trial addresses this gap by evaluating a combined pharmacological and psychosocial approach to MUD. ASCME is a multi-centre, randomized, placebo-controlled, four-arm clinical trial enrolling 440 adults with moderate to severe MUD across Canadian sites. Participants are randomized to 12 weeks of treatment as usual (TAU) with: (1) placebo, (2) placebo plus engagement-focused CM, (3) high-dose lisdexamfetamine (LDX: up to 250 mg/day), or (4) LDX plus CM. LDX administration is double-blinded, while CM is delivered openly and incentivizes engagement in TAU. The primary outcome is reduction in days of self-reported methamphetamine use during the maintenance phase. Secondary outcomes include treatment retention, sustained abstinence, safety and tolerability, medication adherence, quality of life, and participant satisfaction. The ASCME trial is the largest study to date examining high-dose psychostimulant therapy for MUD and the first to assess its additive and synergistic effects with contingency management using a four-arm design. Findings from this study will provide critical evidence to inform clinical practice, service delivery and policy decisions regarding integrated treatment strategies for methamphetamine use disorder.

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Keywords: methamphetamine, contingency management, lisdexamfetamine, addiction treatment, CNS psychostimulants, behavioural intervention, randomized study, substance use

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Using high-resolution eye-tracking to compare fixation stability during naturalistic movie-watching vs. motion psychophysics

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Amblyopia is a common visual developmental disorder characterized by poor visual acuity in a healthy eye that lenses cannot correct. Other aspects of vision, such as motion perception and eye movements, are also affected. Fixation stability—the ability to maintain steady gaze on a target—is often impaired in amblyopia and may cause problems with motion perception. Until now, this has been assessed using psychophysical methods that are tedious and difficult for children to complete. This study assesses the feasibility of using naturalistic movie-watching to characterize visual deficits in amblyopia. We collected binocular eye gaze data from typically developing controls using an EyeLink1000+ eye-tracker while participants viewed a 15-minute "Despicable Me" movie clip. Participants also completed a motion-defined form (MDF) task, in which they identified shapes defined by relative motion, to obtain motion-discrimination thresholds. Fixation stability was computed for each participant as the spread of eye movements (bivariate contour ellipse area [BCEA]) around a central fixation cross in the MDF task. For the movie, BCEAs were calculated for segments categorized as fixations by a naturalistic eye gaze analysis script within identified areas of interest. A positive correlation was found between the MDF and movie-watching BCEAs, as well as between the MDF BCEAs and motion-discrimination thresholds. This study establishes a pipeline for high-resolution eye-tracking that will next be used to compare psychophysical measures of face/emotion perception and attention to naturalistic movie-watching. Future studies aim to include people with amblyopia and compare them to those with typical vision.

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Keywords: amblyopia, eye-tracking, motion perception, fixation stability, movie watching

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Audio-motor adaptation refines auditory-spatial representations in sighted individuals

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In dynamic environments, once-familiar actions can produce unexpected outcomes. Sensorimotor adaptation addresses this challenge in everyday motor control by recalibrating motor output in response to sensory feedback. Despite considerable research on visuomotor adaptation, little is known about auditory adaptation. This study examined whether auditory feedback alone is sufficient for sighted individuals to adjust motor commands by updating auditory-spatial representations. Sixteen participants aged 19–60 with healthy vision and hearing were exposed to sounds corresponding to each cell of a 9x9 grid, differentiated by amplitude and frequency. After familiarization with the auditory-spatial mappings, they were asked to identify the source cells of target sounds to assess learning. In the subsequent reaching task, they were blindfolded and used a computer mouse to move the cursor to one of four target locations, relying solely on auditory cues. Continuous auditory feedback was presented to guide cursor movements based on the learned auditory-spatial mapping. Following a baseline period, a 30° cursor rotation was introduced, creating a mismatch between anticipated and actual auditory feedback. After the reaching task, grid-sound mappings were reassessed without blindfolds. During the reaching task, participants exhibited reduced movement times, improved reaching accuracy, and more linear cursor trajectories, consistent with adaptation to auditory feedback in the absence of visual input. The magnitude of audiomotor adaptation correlated with improved auditory-spatial localization at post-test, suggesting that the adaptation process refined the auditory-spatial representations. These findings demonstrate that auditory feedback alone is sufficient to drive sensorimotor adaptation and to update auditory-spatial mappings.

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Keywords: sensorimotor adaptation, auditory feedback, spatial representation, motor learning

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From preprocessing to prediction: Reproducible network analysis of evidence gathering in schizophrenia

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Delusions are persistent false beliefs that significantly impair functioning in schizophrenia. One cognitive process linked to delusional reasoning is the jumping-to-conclusions bias, in which decisions are made based on limited evidence. Functional magnetic resonance imaging (fMRI) studies using uncertainty-based tasks have identified altered neural responses during evidence gathering in individuals experiencing delusions. However, variability in preprocessing approaches can substantially influence observed activation patterns, reducing reproducibility across studies. Recent advances in standardized preprocessing pipelines and data organization frameworks provide an opportunity to reanalyze existing datasets using more transparent and consistent workflows. This project aims to implement a standardized preprocessing pipeline to reanalyze an existing task-based fMRI dataset and examine the neural mechanisms underlying evidence gathering. Archival fMRI data collected at UBC (2014–2019) included 109 participants with complete anatomical and task data, comprising healthy controls and individuals with schizophrenia with and without delusions. The dataset was converted to Brain Imaging Data Structure (BIDS) format, and preprocessing was conducted using fMRIPrep on the Alliance Canada computing infrastructure. Planned analyses will apply fMRI-CPCA with finite-impulse-response modelling to characterize functional brain networks and hemodynamic responses during evidence gathering. Statistical analyses will be conducted in SPSS and compared to prior findings derived from SPM-preprocessed data. Standardized preprocessing reduced manual intervention but required troubleshooting related to cluster node variability. Preliminary CPCA analyses in the control group replicated previously identified functional networks. Establishing a reproducible framework may strengthen confidence in identifying neural systems associated with delusional reasoning and inform future targeted interventions.

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Keywords: schizophrenia, delusions, jumping-to-conclusions, evidence gathering, fMRI, preprocessing, fMRIPrep, functional brain networks

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Rethinking motion artifact correction in fNIRS connectivity analysis

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Functional near-infrared spectroscopy (fNIRS) is a neuroimaging method beneficial for infant research due to its non-invasiveness and portability. Although proposed to be less motion susceptible compared with fMRI and EEG, motion artifacts still remain a significant challenge in fNIRS analyses. While a variety of motion correction methods have been proposed, their effectiveness has only been examined on univariate data, and their influence on multivariate data remaining unknown. The current study examines the impact of motion artifact correction methods on functional connectivity using data from both task-free and task-based sessions in 8-month-olds. Three correction options were examined: Spline interpolation, Wavelet filtering and Spline + Wavelet, with trial removal method as the default. Given the scarcity of exploration in this field, the current study remains exploratory with no hypothesis assumed. Our results indicated that Wavelet correction significantly increased the estimated marginal means of connectivity compared to trial removal and Spline groups for task-free data. As Wavelet functions by extracting the identified motion component, all the data is subjected to changes, which raises concerns. Accordingly, infants with the lowest motion contamination (< 5%) were examined. Our results indicated a significant difference between the trial removal and the Wavelet group for both task-free and task-based sessions, signifying that even for subjects relatively unaffected by motion artifacts, Wavelet significantly inflates functional connectivity levels. This finding suggests that more caution needs to be taken when applying motion artifact correction methods on multivariate data (e.g., functional connectivity). Future studies might invest more efforts into developing multivariate data appropriate correction methods.

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Keywords: functional near-infrared spectroscopy, fNIRS, motion correction, infants, functional connectivity

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Quality assessment of task-based fMRI data obtained using single-echo and multi-echo sequences

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Multi-echo (ME) functional magnetic resonance imaging (fMRI) is an emerging technique that aims to address signal dropout in regions with high magnetic susceptibility, such as the orbitofrontal cortex and medical temporal cortex. As opposed to single-echo acquisition, ME acquisition involves acquiring multiple volumes within a single repetition time, which can allow for recovery of signal dropout in these regions using optimal echo combination. This study aims to determine the quality of task-based fMRI data acquired through single-echo and ME sequences using two image quality metrics (IQM): (1) temporal signal to noise ratio (tSNR), and (2) a novel Z-score technique that assesses quality by comparing against a set of hypothesized patterns. The Scanned Working Memory (SCANME) task was employed and included 20 healthy controls who underwent fMRI scanning. The fMRI data was preprocessed using Statistical Parametric Mapping 12. Following preprocessing, Constrained Principal Component Analysis for fMRI (fMRI-CPCA) was utilized to retrieve networks, and IQMs were calculated afterwards. An inverse relationship was observed between tSNR and the novel Z-score technique. While longer echo times were associated with lower tSNRs, the average Z-scores were higher, indicating better network quality. While tSNR allows for a quick and easy method of obtaining IQMs, the novel Z-score technique may allow for a more accurate and in-depth determination of task-based fMRI data quality. Our future work is now employing this novel IQM to determine the quality of task-based fMRI data obtained using single band sequences versus multiband acceleration.

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Keywords: neuroimaging, functional magnetic resonance imaging, cognitive neuroscience, biophysics, blood-oxygen-level-dependent signal, task-based fMRI, multi-echo fMRI

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Early exaggerated dopamine-dependent behaviour followed by age-related decline in a LRRK2 Caenorhabditis elegans model of Parkinson's disease

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Parkinson's disease (PD) is the second most common neurodegenerative disease in the world and is characterized by the loss of dopaminergic neurons in the substantia nigra, a brain structure critical for motor control. Mutations in the LRRK2 gene, which plays key roles in neuronal plasticity and vesicle trafficking, represent the most common known genetic contributors to both familial and idiopathic PD. Although LRRK2-associated pathology is often studied in the context of age-dependent neurodegeneration and dopamine loss, how LRRK2 influences dopamine-dependent behavioural responses during early adulthood and across aging remains unclear. Tracking behavioural changes across multiple adult ages is challenging in mammalian models due to long lifespans and cost. To address this, we studied a dopamine-dependent behaviour in the nematode *Caenorhabditis elegans*, which shares conserved dopamine signaling mechanisms with humans and has a short lifespan that enables efficient measurement of age-dependent functional changes *in vivo*. Using the lab's Multi Worm Tracker (MWT), we measured basal slowing—the reduction in locomotion in food-rich environments regulated by dopamine signaling—in worms expressing human LRRK2 wild-type or the pathogenic LRRK2 (G2019S) variant and compared them to wild-type (N2) controls at multiple adult stages. We observed an early increase in basal slowing in LRRK2-expressing adult-aged worms relative to controls, followed by a progressive decline with age. This pattern suggests that LRRK2-associated dysfunction may involve early functional alterations in dopamine signaling that precede later behavioural decline. This research reveals how LRRK2 influences dopamine-dependent behaviour across aging and provides insight into the progression of dopaminergic dysfunction in Parkinson's disease.

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Keywords: Parkinson's disease, dopamine signalling, neurodegeneration, LRRK2 gene, Caenorhabditis elegans, basal slowing

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Probing infants' neural and physiological sensitivity to fairness

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Fairness plays an important role in how individuals evaluate others and engage in cooperative behaviour. Behavioural research indicates that infants in the second year of life reliably detect unequal resource distributions and prefer fair over unfair distributors. However, findings in younger infants are less consistent and appear sensitive to task complexity. Together, this pattern suggests that expectations of equal distributions may emerge early but vary in robustness across development. Despite growing behavioural evidence, the neural and physiological mechanisms supporting infants' processing of fairness remain largely unknown. The present study examines six-month-old infants' neural activity, measured with electroencephalography (EEG), and pupillary responses to fair and unfair distributions. Infants viewed animated resource distribution events depicting equal or unequal allocations, followed by still images of the distributors. Drawing on prior EEG findings from helper and hinderer paradigms, we predict greater P400 amplitudes to unfair versus fair agents (indexing social processing) and no condition differences in the Nc component (indexing attentional processing). We additionally assess whether pupil dilation differs across fair and unfair events. By extending electrophysiological and pupillometry research to the domain of fairness, this study will contribute to the understanding of the early neural foundations of sociomoral cognition.

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Keywords: infant, fairness, electroencephalography, pupillometry, P400, Nc

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Trait shame-proneness and visuospatial attentional orienting

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Shame is a self-conscious emotion characterized by humiliation, self-devaluation, and distress related to the perception that the self is unworthy, flawed, or immoral. Although research has examined self-consciousness and its relationship to emotional experience, relatively little attention has been given to whether shame-proneness may be associated with processing external information. This study will examine whether high and low shame-proneness groups differ in attentional orienting and reorienting by comparing performance on cued (valid) and uncued (invalid) trials of a Posner Cueing Task. This study will be conducted as a cross-sectional, correlational study with a quasi-experimental group comparison. First, participants will complete the Test of Self-Conscious Affect (TOSCA), a self-report questionnaire assessing shame-proneness and related self-conscious affective tendencies. Based on these scores, participants will be categorized into high and low shame-proneness groups. Next, all participants will complete the Posner Cueing Task as a behavioural measure of attentional orienting. Reaction time differences across cued and uncued trials will assess attentional efficiency and reorienting cost. It is expected that individuals with higher shame-proneness may show differences in attentional performance, potentially reflecting greater internal self-focus or reduced efficiency in responding to external cues. If an association between shame-proneness and attentional orienting is observed, the findings would suggest that self-consciousness may not only influence emotional experience and self-evaluation, but also how individuals process and respond to external information. These findings may also have practical relevance in contexts where shame and evaluation are prominent, such as classrooms, public speaking, and social environments.

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Keywords: shame-proneness, attentional orienting, Posner Cueing Task, self-consciousness, reorienting cost, reaction time

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EEG dynamics across the dying process: Neural signatures of brain ischemia

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This ongoing study investigates how brain activity changes during the dying process, as oxygen and blood flow decline, leading to loss of neural function. Using electroencephalography (EEG) recordings from patients who underwent withdrawal of life-sustaining treatment at Vancouver General Hospital, the study examines how cortical activity evolves in real time as the brain transitions toward electrical silence. Previous studies have shown that brief bursts of organized EEG activity can persist after cardiac arrest, suggesting that coordinated neural dynamics may outlast circulation. However, most existing work captures only minutes surrounding death, and continuous EEG recordings spanning the full progression from intact brain activity to electrocerebral silence remain rare. This study extends the observation window to several hours, using recordings from an ongoing clinical research protocol. EEG data are being preprocessed to remove noise and are segmented around key physiological events including oxygen desaturation, cardiac arrest, and electrocerebral silence. Analyses will include oscillatory power across standard frequency bands (delta through gamma), spectral parameterization using the FOOOF algorithm to extract the aperiodic spectral exponent (a measure of the non-rhythmic EEG background reflecting cortical excitation-inhibition balance), and measures of signal complexity quantifying how information-rich the EEG signal is as neural function deteriorates. By tracking these EEG markers across the full temporal progression of dying, the aperiodic exponent is expected to progressively flatten as the brain approaches electrocerebral silence. The findings could refine brain-death assessment, enhance EEG interpretation after cardiac arrest, and advance understanding of how neural function fades as the brain shuts down.

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Keywords: EEG, spectral exponent, aperiodic activity, dying brain, withdrawal of life-sustaining treatment, brain ischemia, neural deterioration, FOOOF

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Visualizing chronic pain perception in autistic adolescents through digital health data

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Chronic pain is a prevalent and multifaceted experience in adolescence, closely linked to sleep quality, mental health, and daily functioning. Autistic adolescents experience disproportionately high rates of chronic pain yet remain significantly underrepresented in pain research. Digital health tools, including ecological momentary assessment (EMA) platforms, offer opportunities for real-time symptom tracking and personalized self-management; however, their accessibility and usability for neurodiverse populations remain largely unexplored. This study evaluates the feasibility and acceptability of *MyWeekInSight*, a data collection and visualization platform designed to support autistic adolescents with chronic pain. Eight participants aged 13–18 will complete EMA surveys three times daily over one week, while also accessing interactive visualizations that highlight relationships between pain, mood, sleep, and activity based on their entered EMA data. Participants will also take part in semi-structured interviews to explore experiences with usability, accessibility, comprehension, and engagement. Qualitative thematic analysis will identify key barriers, facilitators, and areas for platform refinement. Recruitment commenced in February 2026. Findings will offer novel insights into how autistic adolescents record, interpret, and engage with pain-related data through digital tools. By centering lived experiences, this study addresses a critical gap in representation and advances understanding of pain in neurodiverse youth. Results will inform user-centered adaptations to improve accessibility and engagement, supporting more meaningful self-monitoring and communication with healthcare providers. Ultimately, this work aims to guide the development of inclusive digital health platforms and contribute to more equitable pain assessment and management for autistic adolescents.

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Keywords: neurodiversity, autism spectrum disorder, chronic pain, youth, ecological momentary assessment, digital health

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Brain-targeted estradiol delivery in rats via intranasal administration: Time and dose dependent effects in the periphery

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Estrogens are steroid hormones produced by the gonads and brain that can regulate neuronal function and behaviour. Estradiol (E2), the primary estrogen, is often studied for its neuroprotective effects. However, clinical use of E2 has been restricted by harmful side effects in the periphery (e.g., estrogen-sensitive breast cancer, gynecomastia). Intranasal administration of E2 is a promising strategy to specifically target the brain while reducing exposure to the periphery. To assess any exposure outside of the brain after intranasal E2 administration, it is useful to examine the liver because it converts administered compounds (such as E2) into metabolites for excretion. Here, we investigated hepatic levels of common estrogens and their metabolites following intranasal E2 administration in rats. Male and female Long-Evans rats (n = 3/sex/dose/timepoint) were treated with E2 (0–4 mg/kg) intranasally and the liver was collected 6 or 24 hours later. Steroids were isolated from tissue by liquid-liquid extraction and analyzed using liquid chromatography-tandem mass spectrometry. We were able to quantify estradiol and another common estrogen, estrone, in the liver. The levels were elevated in a dose-dependent way and were higher after 6 hours compared to 24 hours. Some metabolites were detected but could not be quantified. Results show substantial exposure of E2 in the periphery at the doses tested, even 24 hours after administration. Future experiments will focus on lower doses to properly minimize peripheral exposure using the intranasal method, enabling more individuals to receive treatment for steroid-sensitive neurologic and psychiatric disorders.

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Aggregating DE trends across transcription factor perturbation studies

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Generating genetic perturbations of transcription regulators (TFs) has become increasingly important for characterizing neuropathologies. Differential expression (DE) analyses are widely used to identify genes with altered expression following a TF perturbation, with the broader goal of uncovering the regulatory mechanisms underlying biological conditions. If DE signatures reflect downstream consequences of regulatory activity, then in principle they should encode information about the upstream TRs driving those changes. This motivates my central question: Can we use DE results to assign candidate TRs to neuropsychiatric diseases? To address this, I utilized data from Gemma, a resource developed in the Pavlidis lab containing over 23,000 standardized human and mouse DE analyses. Of these, 3,611 TF perturbation studies spanning diverse experimental contexts were selected for downstream analysis. Previous work in the lab had conducted uniform processing and data aggregation across human and mouse perturbation datasets, ultimately ranking a set of eight TR-target relationships. Here, I build on the workflow, expanding to a wider spectrum for TR ranking. I perform aggregation of curated DE results to organize the data, and I provide a data overview comparing mouse and human datasets. I then use this data to interpret DE patterns of TF perturbation studies and identify trends through correlative analysis. Through this approach, we aim to better understand the underlying regulation of neuropathological conditions and begin systematically assigning transcription factors to specific neuropsychiatric diseases.

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The impact of season and an aggressive encounter on the circulating levels of DHEA-like steroids in male song sparrows, Melospiza melodia

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Steroid hormones play a central role in regulating aggression across vertebrates. In male song sparrows (*Melospiza melodia*), breeding season aggression is driven by high levels of circulating testosterone. However, aggression persists in the non-breeding season despite low circulating testosterone, suggesting an alternative mechanism. Dehydroepiandrosterone (DHEA), a weak androgen convertible to testosterone in the brain, was initially identified as a regulator of non-breeding aggression through radioimmunoassays, as high circulating levels were found during the non-breeding season. However, recent studies using highly specific liquid chromatography-tandem mass spectrometry (LC-MS/MS) found DHEA levels 10 times lower, suggesting an overestimation by radioimmunoassay due to antibody cross-reactivity with structurally similar steroids. While these DHEA-like steroids may be critical regulators of non- breeding aggression, they remain unexamined in birds. Here, we measured DHEA, androstenedione, testosterone, pregnenolone, 17β-estradiol, and 11 DHEA-like steroids in the plasma of free-living adult male song sparrows using LC-MS/MS. Both breeding and non-breeding season subjects were exposed to a simulated territorial intrusion (STI) or a control treatment (n = 8/treatment/season). As expected, testosterone and androstenedione were detected in all breeding subjects, and 17β-estradiol in some breeding subjects. In contrast, DHEA, pregnenolone, and 11 DHEA-like steroids fell below the lower limits of quantification. STI did not significantly alter any steroid levels. To conclude, the 11 DHEA-like steroids are unlikely modulators of non-breeding aggression or sources for antibody cross-reactivity in male song sparrows. Future studies will investigate alternative sources of RIA cross-reactivity with the aim to identify modulators of male non-breeding aggression in this species.

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Sex-dependent effects of GABAergic modulation on behavioural inhibition

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Inhibitory control, the ability to suppress an action to achieve a goal, is a core component of adaptive decision-making. Disruptions in inhibitory control are often implicated in anxiety-related disorders. However, findings regarding the exact relationship between behavioural inhibition and anxiety are mixed. To investigate this interaction, we manipulated GABAergic signalling using the anxiogenic GABAA receptor inverse agonist FG 7142. In a prior study involving active and inhibitory avoidance tasks, FG significantly reduced active avoidance in male but not female rats, while inhibitory avoidance remained unaffected in either sex. Nonetheless, the effects of FG on behavioural inhibition under motivational conflict, where reward pursuit carries the risk of punishment, remain unclear. To address this, we used a novel behavioural task in which rats must inhibit reward-seeking behaviours to avoid foot shocks when an audiovisual cue was presented. Male and female rats were trained on the task and were each administered 1 mg and 10 mg/kg FG, then the proportion of punished reward-seeking trials was analyzed across sex. We found that FG altered behavioral inhibition in a sex-dependent manner. Males administered 10 mg/kg FG demonstrated increased punished reward-seeking behaviours, whereas females exhibited no comparable changes. No significant differences between 1 mg/kg FG and vehicle were observed in either sex. Along with the study on active and inhibitory avoidance, the results suggest that reduced GABAergic signalling does not broadly impair punishment avoidance, but selectively disrupts inhibitory control in a sex-dependent manner when reward pursuit is in conflict with the presence of threat.

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Proteomic analysis of injured dorsal root ganglion neurons identifies ATAD2 as a novel regulator of axon growth

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Adult mammalian central nervous system axons are unable to regenerate following spinal cord injury, leading to a loss of sensory and motor function. In contrast, adult dorsal root ganglion (DRG) neurons in the peripheral nervous system can re-acquire axon growth competence following nerve injury by changes in gene expression that enhance axon growth competence. While transcriptional networks underlying axon growth ability have been well-characterized using RNA sequencing, RNA transcription can poorly correlate with protein translation, and axon growth competence regulators may be missed. Thus, we performed proteomic analysis of injured DRG neurons to identify injury-induced protein-level changes. Interestingly, ATAD2 is significantly upregulated at the protein level in the DRG following peripheral nerve injury but remains unchanged at the transcriptomic level. ATAD2 contains a bromodomain which binds to acetylated histones, altering chromatin accessibility and downstream expression of transcriptional targets. To investigate its function, we pharmacologically inhibited the function of ATAD2 using the bromodomain drug GSK8814 and hypothesized that this would reduce axon regeneration because of ATAD2's initial upregulation. DRGs were isolated from 8- to 12-week-old mice and cultured for 24 hours in the presence of 10 µM GSK8814 or control. Cultures were fixed, stained to visualize axon outgrowth, and imaged using fluorescent microscopy. Axon outgrowth was quantified using ImageJ. DRG neurons cultured in the presence of 10 µm GSK8814 had a significant decrease in axon outgrowth compared to controls. These findings suggest that ATAD2 is a novel regulator of axon growth in DRG neurons and highlights the importance of multiomic approaches to regeneration.

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Neurodegenerative features in the retina associated with Alzheimer's disease: Dynamic amyloid, glial, and AQP4 changes in brain and retina of control and 3xTg-AD mice

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Alzheimer's disease (AD) involves progressive amyloid-β accumulation, astrocytic and microglial activation, and disruptions in soluble waste clearance. The retina may mirror central pathology, yet early retinal changes and their relationship to brain pathology remain unclear. Here, we examined amyloid precursor protein/amyloid-β (APP/Aβ1-16), aquaporin 4 (AQP4), and glial markers (GFAP and IBA1) across brain and retinal regions in control and 3xTg-AD mice. Male and female mice were analyzed at 3 and 12 months. Retinal and hippocampal tissues were processed for immunohistochemistry using antibodies 6E10, APP, AQP4, GFAP and IBA1. Confocal microscopy, quantitative image analysis in ImageJ, and statistical testing evaluated age- and model-dependent effects. Amyloid-β progression largely mirrored brain pathology but revealed unexpected early retinal patterns, highlighting retina-specific vulnerability. AQP4 increased with age yet correlated only partially with amyloid accumulation, suggesting complex glial-vascular remodeling. Microglial and astrocytic activity rose at early stages but declined with age, revealing a dynamic shift in neuroinflammatory roles across retina and brain. Brain-retina comparisons showed partial correlations, uncovering both parallel and region-specific pathology. These findings highlight age-dependent glial and amyloid dynamics, with early retinal amyloid patterns and AQP4 dissociation providing a sensitive window into central disease mechanisms and the evolving role of neuroinflammation in AD progression.

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Investigating MED15 as a therapeutic target in pediatric neuroblastoma

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Neuroblastoma (NB) arises from the abnormal growth of neuroblasts: neural crest-derived immature nerve cells that differentiate into peripheral neurons during fetal development. When the maturation of neuroblasts into neurons is disrupted, it can remain in an undifferentiated, highly proliferative state, leading to NB, the most common solid tumour in infants and a major cause of childhood cancer mortality. Molecularly, transcription factors and their various co-regulators, such as the Mediator, often drive the dysregulated gene expression that promotes tumour growth. MED15, a subunit of the Mediator complex, is highly expressed and associated with poorer survival in NB. Our lab previously generated MED15 knockout (KO) models in lung adenocarcinoma cells and observed the downregulation of oxidative stress response and pro-inflammatory immune genes, signifying a role for MED15 in these cancer cells; however, MED15's functions in NB are unknown. Therefore, this project aims to determine how MED15 influences oxidative stress and immune-response pathways in NB using CRISPR-Cas9 gene editing to generate stable MED15KO clones in two NB cell lines (IMR-32 and SK-N-AS). Changes in the oxidative stress and immune gene/protein expression will be assessed using qRT-PCR (RNA) and Western blotting (protein). Based on our prior findings, we expect MED15KOs to show reduced oxidative stress and immune response genes/proteins. Many childhood cancers, including NB, remain in need of new treatments. This project may identify MED15 as a new therapeutic target by demonstrating its role in allowing neural crest-derived neuroblasts to proliferate into cancer instead of developing into normal neurons.

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