CONFERENCE ABSTRACT BOOK OPEN ACCESS

2025 National NeuGeneration Case Competition: Neurodegenerative Diseases

Eileen Danaee, BHSc (Honours) Student [1]*, Nnamdi Ndubuka, BSc (Honours) Student [2], Tyler Rotholz, BSc (Honours) Student [3], James Sunwoo, BSc (Honours) Student [2], Sophie Guerrico, BSc (Honours) Student [2]

[1] Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada K7L 3N6
[2] Department of Biomedical and Molecular Sciences, Queen's University, Kingston, Ontario, Canada K7L 2V7
[3] Department of Chemistry, Queen's University, Kingston, Ontario, Canada K7L 2S8

*Corresponding Author Details: neugeneration@clubs.queensu.ca

Abstract
The NeuGeneration National Case Competition is an annual research case competition organized by students in the NeuGeneration Club at Queen's University, open to students across Canada. Held virtually on April 25th, 2025, the competition aims to provide undergraduate students with valuable research experience and networking opportunities in the field of neuroscience. Undergraduate students in teams of 2-5 were given a month to prepare an abstract and an oral presentation on a novel research question within the theme of Neurodegenerative Diseases. This booklet showcases the abstracts of the top 5 teams. We hope you enjoy exploring the proposals and we encourage you to be on the lookout for next year's case competition through our Instagram (@q_neugeneration).

Keywords: neugeneration case competition; neuroscience; neurodegenerative diseases; alzheimer's disease; huntington's disease; chronic traumatic encephalopathy; neural stem cells; fNIRS-neurofeedback; psychedelics; cognitive rehabilitation

Table of Contents

Conference Abstracts
Note: These abstracts have been reproduced directly from the material supplied by the authors, without editorial alteration by the staff of the URNCST Journal. Insufficiencies of preparation, grammar, spelling, style, syntax, and usage are the authors.

NeuGeneration Abstracts

Dual-Targeting RI-AG03 Inhibitor: Evaluation in rTg4510/PS19 Mouse Models of Alzheimer's Disease

Tiana Dobaj, BSc Student [1], Niya Zhao, BSc Student [1]
[1] Faculty of Arts and Science, Queen's University, Kingston, Ontario, Canada K7L 3N6

Alzheimer's disease (AD), the most common form of dementia, affects nearly 50 million people and is characterized by neurofibrillary tangles of hyperphosphorylated tau, prompting neuronal death. RI-AG03, a peptide-based tau aggregation inhibitor, shows promise in Drosophila models targeting 306VQIVYK311 and 275VQIINK280 hot spots. Although initial studies demonstrate RI-AG03's efficacy in reducing tau aggregation in Drosophila, its therapeutic potential in mammals remains unexplored. We hypothesize that RI-AG03 reduces tau aggregation and associated neurotoxicity in rTg4510 and PS19 mice, improving motor and cognitive performance. Transgenic mice inbred with rTg4510 and PS19 will be divided into treatment and control groups of 10 mice, following preclinical research standards. They will receive low (6 mg/kg) and high (60 mg/kg) doses of RI-AG03 via oral gavage for 12 months, with monthly assessments. Cognitive performance will be assessed using the Barnes maze and novel object recognition test, while motor performance is measured using rotarod and beam walking. Tau pathology will be quantified in hippocampal homogenates using ELISA (AT8/T46 and PHF1/T46). Statistical analysis will use ANOVA with Tukey-Kramer post-hoc testing across experimental and control groups (including scrambled peptide and vehicle-only controls) for biochemical markers and behavioural outcomes. We expect ANOVA and t-tests to reveal neuroinflammation and tau aggregation, along with improved motor and cognitive function in RI-AG03 treated mice. These findings are suggested as RI-AG03 inhibits tau aggregation at both hot spots. Validating its efficacy in mammalian models will support its development as a tauopathy treatment, potentially aiding many with Alzheimer's disease.

Enhancing TRIM21-Mediated Clearance of mHTT with Heat Shock Proteins in Huntington's Disease in Mouse Models

Susie Choi, BHSc Student [1], Yunseo Choi, BHSc Student [1], Kiera Hui, BHSc Student [1],
Myungjin (Angela) Kang, BHSc Student [1], Jenna (Bo Fei) Yu, BHSc Student [1]
[1] Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada K7L 3N6

Huntington's disease (HD) is a neurodegenerative disorder affecting 2.7 per 100,000 worldwide, causing progressive motor-behavioural abnormalities. HD results from expanded polyglutamine (polyQ) tracts in huntingtin (HTT), with >39Q causing pathogenesis. Tripartite motif-containing protein 21 (TRIM21), which contains a cytosolic E3 ubiquitin ligase and an antibody receptor, initiates auto-ubiquitination. TRIM21 has also shown potential for treating Alzheimer's disease by targeting tau proteins. Previous research indicated that TRIM21 with the 3B5H10 antibody allowed more TRIM21 to bind longer polyQ tracts, selectively degrading mutant HTT (mHTT). Heat shock proteins (HSP) are molecular chaperones that help refold misfolded or degrade aggregate proteins. We will investigate whether HSP co-delivery enhances TRIM21-mediated clearance of mHTT aggregates in HD mouse models. This 12-week study will utilize a controlled *in vivo* experimental model using R6/2 transgenic mice. The study will consist of three groups (n=10 per group): (1) HSPs and TRIM21, (2) TRIM21 only, and (3) untreated controls, delivered via electroporation. Primary outcome will quantify mHTT aggregates in the striatum and cortex using immunohistochemistry and Western blot. Secondary outcome will involve motor-behavioural assessments. HSPs are expected to enhance mHTT clearance by stabilizing misfolded intermediates and preventing premature aggregation, increasing the pool of soluble mHTT available for TRIM21 recognition. TRIM21, in complex with the mHTT-selective 3B5H10 antibody, will ubiquitinate mHTT for proteasome-mediated degradation of the TRIM21-antibody-mHTT complex while preserving wild-type HTT. By enhancing TRIM21 action, HSP poses an advanced therapeutic approach for HD, delaying or preventing disease onset.

The Impact of fNIRS-Neurofeedback on Cognitive Rehabilitation Outcomes in Early Huntington's Disease

Orlin Chowdhury, BHSc Student [1], Shayna Sharma, BHSc Student [1], Shrika Vejandla, BHSc Student [1]
[1] Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada K7L 3N6

Huntington's Disease (HD) is an inherited neurodegenerative disorder that leads to a gradual decline in cognition and motor function. 40% of patients in the presymptomatic disease stage experience mild cognitive impairment that later progresses into dementia, highlighting the need for cognitive rehabilitation. Functional Near-Infrared Spectroscopy-Neurofeedback (fNIRS-NF) is a promising HD rehabilitation method that leverages brain-computer interface to measure blood oxygenation levels as a measure of brain activity that will map onto visual or auditory stimuli for HD patients to

manipulate their own underlying neural activity. fNIRS-NF is cost-effective, accessible, and poses strong spatial specificity compared to traditional neurofeedback imaging methods, but has yet to be explored in HD treatment. This 4-week study investigates the therapeutic potential of fNIRS-neurofeedback in improving cognitive rehabilitation outcomes in early HD. Forty adults with early HD (stage II on the Shoulson-Fahn scale) will be randomly allocated into experimental and control treatment groups. Participants will engage in cognitive exercises such as the N-back, memory games, and planning activities 2 times/week. The experimental group will receive fNIRS-NF during rehabilitation using the NIRScout XP system coupled with NIRStar software. Both groups will complete the Symbol Digit Modalities Test (SDMT) before beginning rehabilitation and upon completion. Data will be analyzed using an Independent Samples T-Test in SPSS. We anticipate a greater increase in SDMT scores in the experimental group post-treatment compared to baseline than in the control group. These findings may offer a novel, effective, and non-invasive method to enhance cognitive performance in early HD patients.

Potential Therapeutic Effects of Psychedelics on Cognitive Function and Brain Atrophy in Alzheimer's Disease

Amelie Hamlyn-Lovis Herrera, BAS Student [1], Emmanuel Verduzco Aldana, BAS Student [1],
Nima Ahadi-Sarkani, BSc Student [1]
[1] Faculty of Science, McMaster University, Hamilton, Ontario, Canada L8S 4L8

Alzheimer's Disease (AD) is a progressive neurodegenerative disease characterized by cognitive decline, amyloid-beta (Aβ) plaques, neurofibrillary tangles, and neuroinflammation. AD disrupts serotonin receptors, particularly 5-HT2A, impairing the synthesis of proteins vital for modulating gene expression of neuroplasticity-enhancing neurotrophins in the hippocampus and neocortex. Previous preclinical studies have linked psychedelics to AD pathophysiology by inducing functional and structural neural plasticity in brain circuits to delay or reduce brain atrophy. Psilocybin, a psychoactive alkaloid, has the potential to modulate neuroplasticity and inflammation via the 5-Ht2A receptor to improve cognitive function. Our study aims to address the potential clinical use of psilocybin for AD by delaying or reversing brain atrophy and enhancing cognitive function. We will conduct a preclinical randomized control trial to investigate the neurobiological and cognitive efficacy of psilocybin using an animal model of AD, APPswe/PSEN1ΔE9 mice, exhibiting Aβ plaques and impaired neurogenesis. A sample of AD-model mice (N=16) will be housed following regulations. Half (n=8) will receive a control treatment (e.g. saline solution), while half (n=8) will receive a 1 mg/kg dose of psilocybin three times a week, for four weeks through intraperitoneal injection, following published protocols. We will use MRI, PET, and the Y-maze cognitive ability test thrice: at baseline, at the 2-week midpoint, and post-treatment. We anticipate the psilocybin treatment group will demonstrate improved cognitive performance, reduced Aβ plaques and neuroinflammation levels, and increased neuroplasticity markers (synaptic density). Future research should investigate whether these effects could be replicated in human participants.

Dual Biomarker Tracking of Early CTE in Active Collision-Sport Athletes

Ucheoma Obiji-Nnorom, BHSc Student [1], Daniel McDonald, BHSc Student [1]
[1] Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada K7L 3N6

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disease that remains clinically silent for years after repetitive head impacts, limiting opportunities for intervention. Recent research in retired athletes links elevated plasma neurofilament-light chain (NfL)—a marker of axonal degeneration—to worse executive function and post-mortem CTE pathology. Furthermore, digital speech analytics can detect signs of frontotemporal degeneration. To date, no study has paired ultra-low-volume biochemical sampling with auditory phenotyping in active collision-sport athletes. This study will enroll 30 male professional hockey players and 30 age-matched non-contact-athlete controls. Finger-prick dried blood spots will be collected pre-season, 3 and 7 months later, quantified for NfL on a Simoa platform; recorded 60-second monologues will be analyzed with openSMILE to extract vowel-space area, articulation rate, and pause ratio. Athletes will complete a questionnaire logging check counts, sleep, and concussion symptoms, providing exposure and covariates for analysis. Mixed-effects regression adjusted for age, sleep, and mood will test group x time interactions; this logistic model combining Δ-NfL and Δ-speech metrics is expected to correctly identify at least 75 % of high-impact players. By asking whether a single hockey season elevates NfL and worsens speech-motor metrics, this study aims to create the first field-deployable screen for pre-clinical CTE and enable annual monitoring in contact sports. We hypothesize that players logging ≥300 body-checks will exhibit ≥20% NfL elevation and a ≥0.5 standard deviation contraction in vowel-space area, while non-contact controls remain unchanged. This study fills a critical knowledge gap by uniting biochemical and functional signatures of early CTE neurodegeneration.

Conflicts of Interest
The authors declare that they have no conflicts of interests.

Authors' Contributions
ED: Led organization of the 2025 NeuGeneration Case Competition, served on the planning committee for the 2025 NeuGeneration Conference, assisted authors with their abstract submissions, reviewed the abstract submissions and ensured that they adhered to correct formatting standards, drafted the conference abstract booklet, and gave final approval of the version to be published.
NN: Served on organization committee of the 2025 NeuGeneration Case Competition and gave final approval of the version to be published.
TR: Served on organization committee of the 2025 NeuGeneration Case Competition and gave final approval of the version to be published.
JS: Co-Chaired the planning committee for the 2025 NeuGeneration Conference, supervised the 2025 NeuGeneration Case Competition and gave final approval of the version to be published.
SG: Co-Chaired the planning committee for the 2025 NeuGeneration Conference, supervised the 2025 NeuGeneration Case Competition and gave final approval of the version to be published.

Acknowledgements
We would like to acknowledge the following members of the NeuGeneration team for working hard to ensure that this year's competition was a huge success: Isabelle Kosche as Delegates Executive, Alex Uriarte as Experience Pod Director, Izza Ahmed as Marketing Pod Director, and Kian Pelechaty as Finance Executive. We would also like to thank Adam Renato Carbonara and Parsa Balalaie for judging the case competition. Additionally, we would like to thank our campus representatives, Josh Conn (Wilfrid Laurier University), Eidan Erlich and Noa Boulakia (Waterloo University), Noah Sanders (Western University), Stephanie Davidson (University of British Columbia), Katrina Mina (McGill University) and Olamide Mabadeje (McMaster University) for increasing the reach of our case competition to students at their respective universities. Finally, we would like to thank our mentors Tetiana Poliakova, Brianna Bristow, Aryan Singh, Doriana Taccardi, Beatriz Rigo Moises, Noa Boulakia and Tyler Rotholz for sharing their invaluable knowledge and research expertise with our delegates, and for guiding them through the case competition.

Funding
We would like to thank the Residence Society of Queen's University for providing us with the First Year Experience Fund, allowing us to broaden the impact and reach of our case competition.

Article Information
Managing Editor: Jeremy Y. Ng
Article Dates: Received May 11 25; Published Jun 03 25

Citation
Please cite this article as follows:
Danaee E, Ndubuka N, Rotholz T, Sunwoo J, Guerrico S. 2025 National Neugeneration Case Competition: Neurodegenerative Diseases. URNCST Journal. 2025 Jun 03:9(5). https://urncst.com/index.php/urncst/article/view/917
DOI Link: https://doi.org/10.26685/urncst.917