UNDERGRADUATE RESEARCH IN NATURAL AND CLINICAL SCIENCE AND TECHNOLOGY (URNCST) JOURNAL
Read more URNCST Journal articles and submit your own today at: https://www.urncst.com

CONFERENCE ABSTRACT BOOK OPEN ACCESS

SciNapse 2022-2023 Undergraduate Science Case Competition: Neuroscience and The Future

Rebecca Krnel, BSc Student [1]*, Andréanne Ménard, BSc Student [1]

[1] School of Psychology, Faculty of Social Sciences, University of Ottawa, Ottawa, ON, Canada K1N 6N5

*Corresponding Author: rkrne054@uottawa.ca

Abstract
The SciNapse Undergraduate Science Case Competition (USCC) provides an opportunity for undergraduate students to experience the development of a novel research proposal. A case is presented to all participants and, using in-depth literature search (publications, reports, studies, and published writings), students connect and pinpoint key elements allowing them to develop a hypothesis in support of the case in question. Participants also develop a methodology which will test the validity of their hypothesis. This year's case topic focused on the still emerging discipline of neuroscience and its far-reaching implications for the future of human and societal development. In teams of 1-4, undergraduate students tackled the case and provided novel research ideas that may lead to major advancements in neuroscience and its related fields. In total, the 2022-2023 USCC attracted 314 undergraduate students from 12 universities across North America. The top 25% of written submissions in each division are highlighted in this abstract booklet. You may find more information on the annual SciNapse USCC on our website at http://scinapsescience.com.

Keywords: SciNapse USCC; undergraduate research; science case competition; neuroscience

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.

Lower Division

A comparative analysis of the effect of substrate stiffness on the efficacy of pro-regenerative astrocyte-inducing factors FGF-2 and BMP-4

Rohan Krishna, BHSc Student [1], Noam Silverman, BSc Student [1], Ryan Phan, BHSc Student [1]
[1] McMaster University, Hamilton, Ontario, Canada L8S 4L8

Neurodegeneration is connected to the degradation of extracellular matrix components, causing cell loss and contributing to diseases such as Alzheimer's. Astrocytes are glial cells important for developing and repairing neurons. They are essential for brain homeostasis, so they are heavily studied for regenerative therapies for neurodegenerative disease. Astrocytes are induced into a pro-regenerative phenotype when grown on stiff substrates or exposed to specific growth factors, particularly fibroblast growth factor 2 (FGF-2) and bone morphogenetic protein 4 (BMP-4). In this in vitro study, rat primary cortical astrocytes will be cultured on soft and stiff substrates and exposed to varying amounts of FBF-2 and BMP-4. This study aims to determine the effect of substrate stiffness on the efficacy of FGF-2 and BMP-4 on promoting pro-regenerative phenotype. Cell proliferation and glial fibrillary acidic protein (GFAP) expression are key indicators of reactive astrocytes. 5-bromo-2-deoxyuridine staining will be used to analyse proliferation, while GFAP expression will be determined using anti-GFAP

antibody conjugated with Alexa Fluor 594. We hypothesise there will be greater positive correlation between the concentration of FGF-2 or BMP-4 and expression of markers of pro-regenerative phenotype under stiff substrate conditions, compared to softer substrate, thus indicating dependency or synergy between FGF-2 or BMP-4 and mechanotransduction pathways. Specifically, we expect more cell proliferation, GFAP expression, and expression of pro-regenerative genes in cultures on stiff substrates with higher levels of FGF-2 and BMP-4. We hope the results of this proposed study will provide useful information for the development of regenerative therapies for neurodegenerative diseases.

CSPG binding inhibition to promote axonal regeneration after spinal cord injury

Alyssa Thomas, HBSc Student [1], Christopher Kachi, HBSc Student [1], Nicholas Daoud, HBSc Student [1], Joseph Ghossein, HBSc Student [1]
[1] University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Spinal cord injuries (SCI) can cause severe effects such as loss of movement and sensation, leading to complete paralysis. While research continues to explore possible treatments, these have remained limited and, in most cases, not very effective. At the molecular level, it has been determined that chondroitin sulphate proteoglycans (CSPGs), a group of proteins, significantly inhibit neuronal growth after SCI. Following an injury, glial cells upregulate CSPGs which bind to various receptors in neurons and inhibit axonal growth. In this research proposal, we will explore methods to inhibit the binding of CSPGs to tyrosine-protein phosphatase sigma (PTPσ) receptors. More specifically, we propose synthesizing antibodies against the PTPσ receptor binding domain, thus blocking the binding between CSPG and this site. This proposal will be tested in vivo, producing and isolating desired antibodies from a group of mice. These antibodies will then be injected into another sample group to observe their effectiveness. This technique will further prevent the inhibition of axonal growth and promote the regain of neuronal function in SCI patients.

Delivery of short hairpin RNA by AAV9 vector to silence the amyloid for Alzheimer's disease

Jude Demyati, HBSc Student [1], Sitra Abdullahi, HBSc Student [1]
[1] University of Toronto Scarborough, Scarborough, Ontario, Canada M1C 1A4

Millions of victims fall prey to the life-threatening effects of Alzheimer’s Disease (AD). The hallmark of this catastrophic disease stems deep within the brain's neurons in a protein called the amyloid precursor protein (APP). This research proposal aims to provide a novel action plan for silencing the gene responsible for the progression of AD, propelling research on preventative treatment methods through the combination of several groundbreaking techniques. Using adeno-associated viral vectors (serotype AAV9), an intrathecal injection can be performed containing a strand of short hairpin ribonucleic acid (shRNA). The shRNA contains promoters that target APP and is injected into the cerebrospinal fluid (CSF) of transgenic mice with high amounts of the APP gene; this method will rely on blood brain barrier (BBB) damage to transfer AAV vectors through the CSF for non-invasive treatment. This gene therapy combination is not largely explored in AD treatment, but was selected based on literature review; thus, this proposal offers a distinct variation. In order to determine the efficacy of gene silencing, PET imaging and histology are used to analyze the effects of the injection by measuring the by-product of the APP gene, β-amyloid peptide (Aβ).

Infusion of lanosterol to restore cholesterol metabolism in R6/2 mice

Tony Lin, BSc Student [1], Abha Ranjitkar, BSc Student [1], Eden Samson, BHSc Student [1], Tracy Xie, BSc Student [1]
[1] University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Huntington's disease (HD) is an autosomal-dominant, neurodegenerative disease characterized by motor dysfunction, cognitive decline, and drastic behavioural changes. The mutant Huntingtin gene contains a CAG trinucleotide repeat resulting in a polyglutamine expansion in the mutant huntingtin protein (mHTT). While the mechanisms are not yet fully understood, mHTT is linked to the disruption of nerve and glial cells, particularly in their role in synthesizing cholesterol. Abnormal interactions between mHTT and sterol regulatory element-binding proteins (SREBP), which are transcription factors that control the cholesterol biosynthesis pathway, are implicated in HD. About 25% of the human body's total cholesterol is found in the brain and is involved in many vital roles, such as synaptogenesis, axonal growth, and is utilized to create efficient and effective synaptic transmissions. The biosynthesis of cholesterol is shown to be diminished in HD models, along with decreased levels of cholesterol precursor molecules. The lowered cholesterol levels may be linked to the symptoms and progression of the disease. We aim to increase cholesterol biosynthesis by infusion of lanosterol, a precursor to cholesterol, in R6/2 mice. As a result of restored cholesterol homeostasis, we expect to see amelioration in motor defects, cognitive performance, and striatal neuron survival rate in R6/2 mice compared to controls.

Phage therapy targeting gut-microbiome *Akkermansia* as Parkinson's disease treatment

Agnes Chan, BHSc Student [1], Marco Leung, BHSc Student [1]
[1] Queen's University, Kingston, Ontario, Canada K7L 3N6

Parkinson's disease (PD) is a detrimental neurodegenerative disease and recent literature has linked the gut microbiome to PD development. One well-studied bacterium in the gut microbiome is *Akkermansia* and it produces short-chain fatty acids (SCFAs). Many studies have found an abundance of *Akkermansia* in individuals with PD. Although controversial, SCFAs may be implicated in the pathogenesis of PD. We propose to investigate the impact of *Akkermansia* by comparing PD and healthy control stool samples treated with *Akkermansia* bacteriophages. The expected results are that the *Akkermansia* bacteriophages will be able to selectively kill *Akkermansia* while leaving other bacterial populations intact within the stool sample.

Potential application of PHA-543613 to increase α7 nicotinic acetylcholine receptor expression and reduce seizure susceptibility in temporal lobe epilepsy

Rachel Leong, BHSc Student [1], Gaon Park, BHSc Student [1], Richard Ma, BHSc Student [1],
Vivian Chang, BHSc Student [1]
[1] McMaster University, Hamilton, Ontario, Canada L8S 4L8

The field of epilepsy research is ever-changing, with new findings exposing greater insights into the physiological mechanisms of seizures as well as potential therapeutic targets for treatment. Current primary treatments take the form of anti-epileptic drugs (AEDs), which act either by inhibiting ion channels, or by repairing the glutamate and GABA balance within the brain. New research has pointed to α7-nicotinic acetylcholine receptors (α7-nAChRs) in the brain as a potential therapeutic target for epilepsy due to its implications in calcium influx increase, glutamate transporter expression, and glutamate and GABA modulation. It is thus hypothesised that PHA-543613, a novel, highly selective agonist with a strong affinity to α7-nAChRs, would produce stronger anti-epileptic effects with fewer side effects than existing treatment methods. The effects of α7-nAChR agonists on epileptic activity will be measured using electroencephalography (EEG) in vivo and neurochemical analyses ex vivo. Seizures will be induced using pilocarpine solutions in C57BL/6 wild-type mice, then either PHA-543613 (a strong α7-nAChR agonist), cytisine (a partial α7-nAChR agonist), or vehicle control (0.9% saline) will be administered via microtubule injections into the mice hippocampi. Following EEG measurements, the mice will be anesthetized prior to hippocampus excision and preservation. After tissue homogenization and centrifugation, GABA and glutamate concentrations will be measured in the resulting supernatant using enzyme-linked immunosorbent assay (ELISA) and spectrophotometry, respectively. The results of this experiment will provide further insight into the field of epilepsy research by examining the possible application of PHA-543613 to ameliorate the electrical and neurochemical characteristics of epilepsy.

The effect of prebiotics and probiotics on the severity of symptoms in Autism Spectrum Disorder following exposure to antibiotics during early stages of development

Iliana Keritses, BSc Student [1], Laura Kostwinder, BSc Student [1], Maria Tavares, BHSc Student [2], Joyce van Paassen, BMSc Student [2]
[1] University of Guelph, Guelph, Ontario, Canada N1G 2W1
[2] Western University, London, Ontario, Canada N6A 3K7

The human microbiota consists of 10-100 trillion symbiotic microbial cells that are critical for one's digestive system, immune system and reducing neurological conditions. With early exposure to antibiotics, an individual's microbiome composition is negatively affected by reducing the diversity of microbial species found in the microbiome and can lead to an imbalance in the Gut-Brain-Axis (GBA). While more information is required to analyze the direct relationship between the GBA and neurological disorders, evidence suggests that individuals with a poor microbiome and an imbalanced GBA have an increased risk of developing neurological disorders, specifically Autism Spectrum Disorder (ASD). To improve the microbiome, exposure to a high prebiotic and probiotic diet in the early stages of life can reintroduce beneficial bacteria back into the microbiome and improve microbial diversity. Probiotics can improve the microbiome and restore balance to the GBA for the rest of an individual's lifetime. With the introduction of a prebiotic and probiotic diet and a balanced GBA, there is a possibility to reduce the severity of ASD symptoms for those with ASD. By reducing the severity of ASD symptoms, the life expectancy of those with severe ASD can potentially be extended and allow them to live a more independent lifestyle.

A novel Alzheimer's disease treatment: Staurosporine as a CHT1 enhancer

David Gou, BHSc Student [1], Priyansh Patel, BSc Student [1], Zachary Smith, BSc Student [1], Laura Weiler, BEng Student [1]
[1] McMaster University, Hamilton, Ontario, Canada L8S 4L8

Alzheimer's disease (AD), the most common type of dementia, is a progressive neurodegenerative disease affecting over 55 million patients worldwide. With no cure and an estimated annual global cost of US$1 trillion, there is an urgent need for novel cures and treatment pathways. Cholinergic signalling dysfunction and loss of acetylcholine (ACh) receptors have been strongly linked to the pathogenesis of AD. Thus, the cholinergic neuronal pathway is a promising therapeutic target for AD which has gained increasing attention in recent years. Choline uptake relies on the high-affinity choline transporter, CHT1, and is the rate-limiting step of ACh synthesis. While acetylcholinesterase (AChE) inhibitors have traditionally been the major focus for AD therapeutic agents, increasing choline uptake is a relatively unexplored field. However, increasing surface levels of CHT1 is insufficient to change long-term choline uptake due to gene-related regulation mechanisms. As a result, CHT1-enhancing compounds may open the door to new treatments. Staurosporine (STS), a compound derived from bacteria commonly known for its anti-cancer properties, demonstrates potential as a positive modulator of CHT1. This study will measure the effects of STS on CHT1 activity in vitro using a high-affinity choline-uptake assay (HACU) on SH-SY5Y human neuroblastoma cells.

Treating Huntington's disease with MSCs secretions

Luna Khayat, BSc Student [1], Sara Tfaily, BSc Student [1], Yara Samaha, BSc Student [1]
[1] University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Huntington's disease (HD) is a hereditary neurodegenerative disorder characterized by impaired muscle coordination and mental decline in late stages. HD is characterized by the accumulation of intranuclear mutant huntingtin and degeneration mainly in the basal ganglia. The onset and progression of HD are broadly influenced by inflammation caused by the pro-inflammatory activation of microglia. Mesenchymal stem cells (MSCs), specifically derived from the umbilical cord, have emerged as a novel therapy for various neurodegenerative diseases, including Alzheimer's disease and Parkinson's disease. Transplantation of MSCs has shown promising outcomes by decreasing behavioral deficits in animal models of HD. However, they have limited capability to differentiate into neurons in vivo. Thus, its effect is due to their secreted factors providing growth factor support and protection against neuronal death. In this study, we will investigate the impact of the umbilical cord-MSCs secretions (secretome) on behavioral and neuropathological outcomes in an HD mouse model. In addition to evaluating HD motor symptoms, the striatal volume and neuronal intranuclear inclusion, as well as activated microglia, will be measured in an R6/2 mouse model in order to assess the effect of the secretome on the pathology and progression of the disease. Thus, this study will provide a potential cell-free therapy not only for alleviating HD symptoms, but a neuroprotective and anti-inflammatory effect that reduce the progression of HD.

Upper Division

Amyloid-beta hairpin inhibitor (ABHI): A novel peptidomimetic inhibitor to prevent fibrillization in Alzheimer's disease

Anna Tymofyeyeva, BMSc Student [1], Romanch Shah, BMSc Student [1], Kris Gandhi, BSc Student [2]
[1] Western University, London, Ontario, Canada N6A 3K7
[2] University of Toronto Mississauga, Mississauga, Ontario, Canada L5L 1C6

Alzheimer's disease (AD) is a progressive neurodegenerative disease that is characterized by the accumulation of amyloid-beta (Aβ) plaques in the brain. The Aβ protein exists in several forms, with the isoforms most conducive to neurodegeneration being soluble, N-truncated, and pyroglutamate modified. Dimeric and oligomeric Aβ fibrils also appear to play a major role in neurodegeneration when compared to their monomeric counterparts—a dangerous component of AD as fibrils readily form. A pseudo-β-hairpin structure has recently been identified in the N-terminal region of Aβ monomers and has been shown to correspond to its unique fibrillization behaviour. In this study, we design and synthesize a novel Aβ hairpin inhibitor (ABHI) using peptidomimetics, that will bind to the N-terminal β-hairpin structure with high affinity and specificity to prevent fibrillization, thus preventing further neurotoxicity. We isolated and cultured Aβ monomers from an AD pathology mouse model for assessment of our inhibitor. After performing trials with the cultures, we found that, compared to the control and a previously characterized aggregation inhibitor, our peptidomimetic inhibitor resulted in the greatest reduction of Aβ oligomerization. As such, our inhibitor offers a multitude of therapeutic options for AD prevention, although further in vivo and clinical research is needed before implementation.

Effect of different learning types on periaxonal space width and cognitive functions

Aliya Ali Shaikha, HBSc Student [1], Julien Hazel Barredo, HBSc Student [1], Wania Qamar, HBSc Student [1], Zaynab Azeem, HBSc Student [1]
[1] University of Toronto Scarborough, Scarborough, Ontario, Canada M1C 1A4

Periaxonal space is a region in the neuron that has been found to affect membrane resistance induced by myelination. This space can be modulated by neuronal activity. Previous research has shown that increased spatial learning increases neuronal activity, reduces the width of periaxonal space, and increases the neuron's conduction velocity. However, no other forms of learning have been tested. The impact of the periaxonal space on cognitive function has also not been established. Our study will investigate the effects of different types of learning (spatial, auditory, and motor) on the width of periaxonal space and its effect on various cognitive functions: processing time, working memory, long-term memory, and short-term memory.

In-vitro p53-suppressed brain organoids to model pathogenesis of acquired ataxia

Madison Hunter, HBSc Student [1], Rubina Sardar, BSc Student [2], Shayamikka Ravichandran, HBSc Student [3]
[1] McMaster University, Hamilton, Ontario, Canada L8S 4L8
[2] Western University, London, Ontario, Canada N6A 3K7
[3] University of Toronto Scarborough, Scarborough, Ontario, Canada M1C 1A4

The cerebellum is an important region in the hindbrain that is specialized for interdisciplinary functions such as motor execution, balance, coordination and cognition. Lesions or tumours in the cerebellum may result in a type of cerebellar ataxia known as acquired cerebellar ataxia. Recent developments in studying pathogenesis involve the use of in-vitro brain organoids to model the characteristics of neurons as they develop. The etiology of acquired ataxia is not well understood. However, brain organoids can be used to model disease progression of acquired ataxia to direct future research toward a better understanding of the disease and potential treatments. In order to achieve this, CRISPR/Cas9 technology will be used to supress the gene, p53, to model tumour-induced ataxia and mimic a non-hereditary tumour in the cerebellum. The formation of the lesions in relation to time will be observed to monitor disease progression.

Novel probiotic treatment for reduction of long-term memory loss in Alzheimer patients

Tyler Pereira, BSc Student [1]
[1] University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Alzheimer's disease (AD) is a neurodegenerative disorder that mainly afflicts a large percentage of the elderly population. AD can cause many problems, most notably long-term memory loss. Many studies have observed a decreased frequency of substance P (SP) in the hippocampus of AD patients. In this paper, we propose a novel strategy to limit this loss of long-term memory using probiotics transformed with SP in the colon of mice. Long-term memory will be measured through Morris Water Maze (MWM) tests over a few months to look for improvements. We are hopeful that our treatments will establish a new therapeutic option for AD patients to slow the progression of long-term memory loss.

Potential synergistic interactions between curcumin and piperine on remyelination in multiple sclerosis induced mouse optic nerves

Anthea Mavridis, BSc Student [1], Ashley Nicholls, BSc Student [1]
[1] University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Multiple sclerosis (MS) is a chronic autoimmune disease characterized by myelin sheath degradation along with debilitating chronic symptoms. The proportion of the population that is affected by MS is increasing along with the socioeconomic impact that the disease has on its bearers. Recent studies turn to herbal medicine, which has used natural compounds, such as turmeric, to alleviate the symptoms of ailments for centuries. Curcumin, a compound of turmeric (*Curcuma longa*), has demonstrated regenerative properties towards remyelination in cases of MS. Further research suggests piperine, the primary protein of black pepper (*Piper nigrum*), enhances the bioavailability of curcumin, indicating synergistic behavior in myelin regeneration. This study will test the potential synergistic effects of curcumin and piperine on action potential (AP) conduction velocity in the mouse optic nerve. It is hypothesized that the mixed compound will have synergistics effects on axon remyelination in MS induced mice. Due to the role of the myelin sheaths as electrical insulators, AP velocity (speed at which the signal can travel) will be used as a proxy for remyelination. It is predicted that when exposed to both curcumin and piperine, AP conduction velocity will increase synergistically, indicating greater remyelination. Treatment groups with both curcumin and piperine will display the greater-than-additive AP conduction velocity compared to treatment groups of a single compound.

Therapeutic application of exosome-encapsulated nanoparticles containing plasmid DNA expressing heat shock protein to treat Parkinson's disease

Simrit Rana, BHSc Student [1], Nyah Shah, BHSc Student [1]
[1] McMaster University, Hamilton, Ontario, Canada L8S 4L8

Parkinson's disease (PD) is classified as a proteinopathy, mainly due to its characteristic accumulation of Lewy bodies or aggregates of proteins containing α-synuclein (α-syn) which induce death of dopaminergic neurons. The hallmark treatment involves administering L-DOPA to increase dopamine production, yet many complications exist including dyskinesia, excessive and unwanted motor movements. Nanoparticles present as a promising alternative field of therapeutics to overcome limitations posed by traditional pharmaceuticals in the treatment of PD. As drug-delivery vehicles, nanoparticles possess an enhanced ability to traverse the blood brain barrier (BBB) and the ability to localise delivery of disease therapeutics, thereby enhancing dosing efficacy. This paper addresses the issue of nanoparticles' immunoreactivity and lack of target specificity through the use of exosome-encapsulated nanoparticles (ENPs) containing plasmid DNA expressing heat shock protein 70 (HSP70). ENPs possess a suitable half-life stability within the human body, traverse the BBB efficiently and protect contents from degradation through the host cell's phagocytic mechanisms. By targeting misfolded proteins of α-syn, HSPs have the potential to attack an early step in pathogenesis by directing α-syn to the ubiquitin–proteasome system (UPS). This mechanism is thus effective at stopping the polymerization cascade forming Lewy bodies. Plasmid DNA represents an efficacious method of delivering HSP genetic material into cells as they have a very large DNA packing capacity. Therefore, pairing an ENP based drug delivery vehicle with HSP70 Plasmid DNA is a novel and versatile therapeutic strategy for PD, as well as other neurodegenerative diseases with the appropriate ENP chemical and biological modifications.

The therapeutic potential of HDAC inhibitors for dopamine deficiency in Alzheimer's disease

Andrew Huynh, HBSc Student [1], Jillian Cruzana, BSc Student [1], Sushmita Kiri, HBSc Student [1]
[1] University of Toronto Mississauga, Mississauga, Ontario, Canada L5L 1C6

HDACs (histone deacetylases) are enzymes with down-regulatory epigenetic functions. Various HDACs have been shown to contribute to the pathogenesis of neurodegenerative diseases such as Alzheimer's disease by reducing synaptic plasticity and promoting the accumulation of neurotoxic proteins. A novel frontier of research with great potential to yield treatments for Alzheimer's disease is the application of epigenetic regulation to neurotransmitter rehabilitation. Seeing as the cognitive impairment and memory deficits of Alzheimer's disease have been linked to dopamine deficiency in the brain, the usage of HDAC inhibition to reverse HDAC downregulation of dopamine metabolic pathways may increase dopamine levels and have a positive behavioural effect on memory. This research proposal hypothesizes that the inhibition of HDAC enzymes can upregulate hippocampal dopamine concentrations and therefore improve memory in Tg2576 transgenic mice models of Alzheimer's disease. Three experiments are proposed to investigate the hypothesis. Firstly, the eighteen types of HDAC enzymes will be individually injected into mice to identify those which decrease hippocampal dopamine concentration. Secondly, an HDAC inhibitor for each of the identified HDACs will be chosen and longitudinally injected into the mice while hippocampal concentration is measured using microdialysis. Finally, sulpiride (a dopamine receptor antagonist) will be injected into the hippocampus while the mice undertake the Morris water maze task in order to see whether any increases in hippocampal dopamine resulting from HDAC inhibition lead to behavioural improvements in memory.

Vitamin K supplementation for prevention of teratogenic neurological calcification in fetuses with Congenital Zika Syndrome

Erin Ford, BHSc Student [1], Emma Griffin, BHSc Student [1], Veda Sharpe, BHSc Student [1],
Simon Zacharias Bezanson, BHSc Student [1]
[1] Queen's University, Kingston, Ontario, Canada K7L 3N6

Congenital Zika Syndrome (CZS) manifests in infants who have been exposed to the ZIKA virus (ZIKV) *in utero*. Recent studies have documented the correlation between bone morphogenetic protein (BMP) and fetal brain calcification in these infants; however, potential treatment avenues remain unaccomplished. Since 2010, the increase in ZIKV cases has engendered a plethora of fetal neurodevelopmental defects including brain calcification which permanently impairs neurological function. The dominant pathogenic explanation results from osteogenic factor upregulation. Previous research has identified this underlying factor, highlighting a key potential mechanism by which to combat the neurodegenerative impacts of ZIKV. Since the correlation between ZIKV and BMP is novel, studies addressing the mitigation of infant brain calcification are limited. We use data from established ZIKV research to determine the potential utilization of matrix Gla protein (MGP) to inhibit the BMP pathway which calcifies fetal neural tissue. We founded our rationale on evidence showing (a) the efficacy of MGP in combating BMP-dependent calcification, and (b) the activation of MGP with Vitamin K2 (VK2). Our findings indicate that low-dose maternal VK2 supplementation could provide a potential avenue for prevention of brain calcification *in utero* after vertical transmission of ZIKV. The proposed treatment would be the first of its kind, providing affected populations with a low-cost intervention for neurological damage caused by CZS, decreasing the burden of disease as ZIKV prevalence grows.

Conflicts of Interest

The authors declare that they have no conflict of interests.

Authors' Contributions

RK: President of SciNapse and Chair of the USCC planning committee, assisted authors with their abstract submissions, drafted the conference abstract booklet, and gave final approval of the version to be published.
AM: President of the Undergraduate Research Initiative, served on the planning committee for the USCC, drafted the conference abstract booklet, and gave final approval of the version to be published.

Acknowledgements

We want to acknowledge the entire SciNapse Executive Committee for playing an integral role in making this year's USCC a success. The team that helped make this competition possible includes: Rebecca Krnel as President of SciNapse and Chair of the planning committee, Andréanne Ménard as President of URI, Justen Choueiry and Jebreel Alayche as co-Vice-Presidents of Finances of URI, Misha Kaniyath as Communications Director, Jade Gamelin Kao as Social Media and Marketing

Coordinator, Marilou Poitras as Mentorship Lead, Sarah Heffernan as Scientific Advisor, Dan Celestin as External Campus Coordinator, Giselle Joseph and Adam Omari as uOttawa Campus Leaders, Marisa Levy as Event Leader, Eve Desjardins and Rita Murali as Graphic Designers, Jin-Young Bang and Almamy Diop as Project and Statistics Coordinators, and Saajan Dekiwadiya-Patel as Sponsorship and Statistics Coordinator. The USCC would not have been successful without the contribution and work of the multiple different chapters of the SciNapse USCC situated across other universities in Ontario. We want to acknowledge all the professors and graduate students that participated as judges for the competition. Your valuable time and feedback to the participants has had a positive impact on their future success.

Funding
The SciNapse USCC is funded by the Undergraduate Research Initiative and contributions from Campus Vibez uOttawa.

Article Information
Managing Editor: Jeremy Y. Ng
Article Dates: Received Mar 01 23; Published Mar 14 23

Citation
Please cite this article as follows:
Krnel R, Ménard A. SciNapse 2022-2023 Undergraduate Science Case Competition: Neuroscience and the future. URNCST Journal. 2023 Mar 14: 7(3). https://urncst.com/index.php/urncst/article/view/469
DOI Link: https://doi.org/10.26685/urncst.469
