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 2025-2026 Undergraduate Science Case Competition: Innovations in Reproductive Health

Chris Kachi, HBSc [1]*, Sarah Hussain, HBSc [2], Moumita Dutta [1]

[1] Department of Translational and Molecular Medicine, Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5
[2] Department of Chemistry and Biomolecular Sciences, Faculty of Science, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

*Corresponding Author Details: ckach031@uottawa.ca

Abstract
The SciNapse Undergraduate Science Case Competition (USCC) offers undergraduates the chance to craft an innovative research proposal. In this competition, a case study is provided, and students conduct comprehensive literature reviews — including scholarly publications, reports, and studies — to identify and connect crucial elements, which then form the basis of a supporting hypothesis. They also design a methodology to assess the validity of their hypothesis. This year's case focused on the intricate and often overlooked realm of reproductive health, exploring its significant effects on human health, disease, and wider ecological systems. In teams of 1-4, undergraduate students engaged with the challenge by crafting innovative research proposals aimed to catalyze breakthroughs and deepen our understanding of reproductive health. In total, the 2025-2026 USCC attracted 910 undergraduate students from 14 universities across North America. The top 10% 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 https://scinapsescience.com.

Keywords: SciNapse USCC; undergraduate research; science case competition; reproductive health

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

Rewiring the Brain-Uterus Axis: Oxytocin and Social Buffering in Endometriosis

Eva Hutcheson, HBSc Student [1], Serena Khouri, HBSc Student [1], Shyla Oommen, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Endometriosis is a chronic inflammatory condition in which endometrial-like tissue proliferates outside the uterus. It causes severe pelvic pain, hormonal dysregulation, and infertility, yet its symptoms often exceed the severity of visible lesions. Increasing evidence suggests that this disconnect occurs because endometriosis involves a dysfunctional brain-uterus axis, where psychological stress, social environment, and neural sensitization contribute directly to pain and reproductive outcomes. Chronic stress elevates glucocorticoids, disrupts progesterone signalling, alters ovulation, and worsens lesion inflammation. Meanwhile, neuroimaging studies in affected individuals reveal abnormal activation in the amygdala, insula, and medial prefrontal cortex-regions that integrate pain, emotion, and hormonal feedback. This project proposes that positive social interaction, acting through oxytocin-dependent neural pathways, can reshape disease progression. Using a surgically induced rat model of endometriosis, the study examines how social buffering and oxytocin influence lesion growth, neuroinflammation, hormonal balance, and pain behaviour under chronic stress. Five experimental groups will compare the effects of stress, isolation, social buffering, exogenous oxytocin, and oxytocin-receptor blockade on neural, immune, and reproductive physiology.

We predict that stress and isolation will amplify pain, increase lesion innervation, disrupt estrous cycling, and heighten central sensitization, while social buffering and oxytocin will normalize HPA-axis activity, reduce inflammation, stabilize hormonal rhythms, and improve reproductive markers. Demonstrating that social experience can directly influence uterine pathology and fertility-related hormones would position endometriosis as a multisystem disorder shaped by psychosocial and neuroendocrine factors. This work offers a foundation for innovative, integrative treatments that extend beyond pelvic surgery and hormonal suppression.

PTFE-Induced Infertility in Human Oocytes Through Mitochondrial Dysfunction and Ferroptosis

Demetrios Kokkoros, BHSc Student [1], Finlay Paterson, BHSc Student [1], Lakshdeep Hundal, HBSc Student [1], Nicholas Mykhaylov, HBSc Student [1]
[1] McMaster University, Hamilton, ON, Canada L8S 4L8

Microplastics have now been detected in all parts of the human body, including ovarian follicular fluid. This is a deeply concerning matter, as not even the reproductive system is immune to environmental contamination. Among these pollutants, Teflon microplastics (PTFE) are consistently the most abundant in human follicular fluid (FF), yet no study has characterized their effects on human oocyte quality. Existing reproductive-toxicology research focuses almost exclusively on polystyrene nanoplastics (PS-NPs), which degrade into reactive monomers and do not represent PTFE's fluorinated, inert structure. This study proposes the first direct investigation of PTFE using human cumulus–oocyte complexes (COCs) and focuses on mitochondrial dysfunction and ferroptotic lipid injury. FF typically contains 0.9-2.5 × 10^3 microplastic particles/mL, with PTFE present in more than half of IVF patients. Because the meiotic transition from germinal vesicle to metaphase II depends on uninterrupted mitochondrial ATP production and redox balance, mitochondrial disruption may directly impair oocyte competence. Ferroptosis, an iron-dependent form of regulated cell death driven by phospholipid peroxidation, provides a tenable mechanistic model linking PTFE exposure to oocyte injury. To evaluate mitochondrial membrane potential, lipid peroxidation, GPX4/ACSL4 expression, and cell-free mitochondrial DNA (cf-mtDNA), this proposal will expose human COCs to clinically relevant PTFE concentrations. PS-NPs will function as a ferroptosis-positive control, while MitoQ, Ferrostatin-1, and deferoxamine serve as potential rescue options. Through these experiments, we will determine whether PTFE poses an unrecognized threat to human reproductive capacity.

Inflammation-Responsive, Trophoblast-Targeting Nanoparticles for Localized Delivery of Nicotinamide Riboside to Restore Mitochondrial Function in Placental Organoids

Aidan Wang, HBSc Student [1], Jonathan Liu, HBSc Student [1], Phoenix Hsuen, HBSc Student [2], Sailesh Badri, HBSc Student [2]
[1] University of Toronto, Toronto, ON, Canada M5S 3E2
[2] Queen's University, Kingston, ON, Canada K7L 3N6

Pregnancy complications, such as preeclampsia, are often caused by placental dysfunction which are described by inflammation and oxidative damage. These sources of placental stress impair trophoblast function and proper metabolism in the mitochondria. One cause of this dysfunction is the depletion of nicotinamide adenine dinucleotide (NAD+), an important cofactor in redox reactions. A vitamin B3 derivative, known as nicotinamide riboside (NR), has been shown to increase intracellular NAD+ levels, restoring regular function in the mitochondria. However, current NR administration during pregnancy raises concerns of off-target side-effects that modify existing metabolic function. Targeted strategies that restore placental NAD+ have not been explored prior to this study.
We propose an inflammation-responsive nanoparticle system that is engineered to deliver NR selectively to placental tissue. NR-loaded and biodegradable PLGA–PEG nanoparticles were functionalized with a trophoblast-specific ligand and coated with a reactive-oxygen-species (ROS)-labile thioketal shell. Using human trophoblast-derived placental organoids as an in vitro model, we will test the hypothesis that these nanoparticles ("NAD+ replenishers") preferentially accumulate in placental tissue and release NR under inflammatory conditions induced by TNF-α or lipopolysaccharide (LPS). Data measured include intracellular NAD+ levels, mitochondrial membrane potential, oxygen consumption rate, cell viability, and cytokine secretion. If successful, this study will be a proof-of-concept for placenta-specific NAD+ restoration to mitigate inflammation-associated placental dysfunction while minimizing unwanted exposure.

Potential Application of Implanting Stromal Cell Endometrial Organoids to Improve uNK Cell Growth in Endometriotic Tissues

Claire Wang, HBSc Student [1], Lily He, HBSc Student [1], Lucy Wang, HBSc Student [2], Vicky Zhao, HBSc Student [3]
[1] University of Toronto, Toronto, ON, Canada M5S 3E2
[2] McMaster University, Hamilton, ON, Canada L8S 4L8
[3] McGill University, Montreal, QC, Canada H3A 0G4

Endometriosis severely impacts many women around the world, but remains a continually underfunded field of study in both treatment and understanding. Endometriosis is a chronic inflammatory condition where endometrial tissue grows outside the uterus. A key factor to infertility in endometriosis is the defective decidualization of endometrial stromal cells (ESCs), which

disrupts signals required in uterine natural killer cell (uNK) proliferation and abundance. This paper proposes the implantation of an endometrial organoid co-cultured with stromal cells into the uteri of mice with induced endometriosis to study its potential as a treatment. Organoids are self-organizing structures derived from stem cells that can mimic functional organs and have been shown to increase the restoration of damaged tissues after implantation in vivo. The mice would subsequently be bred with male mice on a 1:1 scheme, with their uteri harvested 11 days after confirmed pregnancy to measure uNK and stromal cell count using immunohistochemistry. Results will be compared with those of untreated mice with and without endometriosis. The organoid implantation is anticipated to increase uNK abundance, thereby restoring stromal-immune communication and potentially offering a new strategy for combating endometriosis and related reproductive challenges.

If You Can't Swim, You Can't Win: Investigating the Effects of Novel Synthetic Steroid RU1968 at Varying Concentrations on Reversible CatSper Channel Inhibition

Bridget Stewart, HBSc Student [1], Kimberly Jiang, BHSc Student [1], Naba Aziz, BHSc Student [1], Ryder Stephen, HBSc Student [1]
[1] Queen's University, Kingston, ON, Canada K7L 3N6

Non-hormonal male contraception is a growing field of study within reproductive health and has positive implications on maternal and infant health and gender equity. It redistributes the responsibility of family planning onto both men and women and widens the choices for contraception, improving accessibility if current female or male contraceptives are not viable for certain individuals. Thus, it is important to remember that male contraception is an interdisciplinary women's health issue as well as a men's health issue. This study aims to target inhibition of the sperm-specific CatSper channel as a method of non-hormonal male contraception using RU1968, a steroid-derived molecule. The CatSper channel is a Ca2+ channel whose activation allows the influx of Ca2+ ions, inducing hyperactivated motility which is essential for the penetration of oviductal mucus and the zone pellucida. Since the CatSper channel is found exclusively in sperm, its reversible inhibition has been at the forefront of research into non-hormonal male contraceptives. HC-05645 was one of the leading reversible inhibitors of the CatSper channel, thus has had its properties extensively studied. RU1968 is a comparatively new discovery, however it has displayed similar inhibitory effects and selectivity, giving it the potential to be an effective inhibitor with minimal toxicities.

A Longitudinal Study Determining the Influence of Relaxin-1 in Mitigating Inflammation of Prostate Glands Associated with Adjuvant-Induced Chronic Prostatitis in Rln1-Knockout Murine Models

Arinie Prabaharan, BMSc Student [1], Ella Stanescu, BMSc Student [1], Sonakshi Sengupta, BMSc Student [1], Veronika Chan, BMSc Student [1]
[1] Western University, London, ON, Canada N6A 3K7

Chronic prostatitis is a condition that affects male reproductive health by inflaming the prostate gland. Although its etiology is not well understood, studies have suggested that H2 relaxin, a hormone encoded by the human RLN2 gene and produced by the prostate, has anti-fibrotic properties that could reduce collagen accumulation and remodel the extracellular matrix of fibrotic tissue within the prostate. This study aims to investigate, using adult male (C57BL/6) mice, if the expression of their orthologous Rln1 gene would influence the magnitude of prostate inflammation in adjuvant-induced prostatitis. A longitudinal study with a sample of 60 C57BL/6 mice will be conducted by initially identifying if the mice have the wild-type or Rln1-deletion gene. Each of these groups will then be randomly assigned to either the experimental (LPS-induced inflammation) or the control (no induced inflammation) condition. Over 28 days, through phlebotomies and prostate tissue collection, the presence and quantity of pro-inflammatory cytokines (IL-6/TNF-alpha) and visual evidence of inflammation will be used to draw conclusions. It is anticipated that Rln1-deletion gene mice will have higher cytokine levels as well as a higher inflammation score of their prostate tissue. These results suggest that relaxin could be utilized in the treatment of chronic prostatitis as well as an anti-fibrotic agent for localized use.

Exploring the Potential of the eNOS/cGMP/PKG Pathway as a Targetable Axis for Impaired Follicular Activation in Primary Ovarian Insufficiency

Emma Liu, BHSc Student [1], Isaac Tang, BHSc Student [1], Sifan Liu, BHSc Student [1], Steven Yu, BHSc Student [1]
[1] McMaster University, Hamilton, ON, Canada L8S 4L8

With a prevalence of up to 3.5% for women under 40, the loss of normal ovarian function is a significant clinical problem. This condition, known as primary ovarian insufficiency (POI) is characterized by the premature or abnormal activation of the follicular reserve. POI has associations with severe conditions like osteoporosis, cognitive impairment, and infertility if left untreated. Recent studies have linked the activation of follicles, which is controlled by the PI3K/AKT pathway and transcription factor FOXO3a, to an upstream pathway involving eNOS. The phosphorylation of FOXO3a via activated AKT promotes its binding to nuclear export proteins and subsequent degradation in the cytoplasm. Without FOXO3a acting as a transcriptional repressor, the oocyte can exit its dormant state. Using a target radiation mouse model to simulate POI conditions, this study aims to experiment with the pathway's functionality in damaged ovaries. The radiation increases the production of reactive oxygen species (ROS) that induce cellular and DNA damage, accelerating follicular apoptosis. By upregulating cGMP and inhibiting eNOS via L-NAME, we aim to verify if the eNOS/AKT/PI3K pathway can continue activating follicles without its upstream mediators. If this study supports the continuation of the eNOS/AKT/PI3K pathway despite the higher level of ROS caused, it provides significant evidence for the pathway's potential as a targetable axis. This understanding could potentially allow for future development of fertility treatments that account for the small follicle reserve of POI patients.

Protective Effects of Vitamin K2 (MK-7) on Mitochondrial Integrity and Investigating the Potential Protective Effects of Menaquinone-7 on Mitochondrial Integrity and Ovarian Reserve–Related Functions in Oxidative Stress–Exposed Human Granulosa-Like Cells

Ilyass Najjar, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Infertility affects approximately one in six individuals of reproductive age worldwide, and much of it is due to female factors including diminished ovarian reserve (DOR). Since females are born with all the oocytes they will ever have, any damage to granulosa cells and their mitochondria accelerates ovarian aging and DOR. One of the most significant contributing factors to accelerated ovarian aging is oxidative stress; the imbalance of reactive oxygen species (ROS) and antioxidant defenses thereof compromises mitochondrial performance, leading to damaged mitochondrial DNA (mtDNA) and ultimately apoptosis. Thus far, one molecule that has been shown to beneficially impact other non-ovarian systems via mitochondrial electron transport, enhancement of quality-control pathways, and reduction of oxidative cell death is vitamin K2. While little is known about menaquinone in ovarian granulosa-like cells, this study aims to test MK-7 to test whether mitochondrial homeostasis and mtDNA integrity can be maintained despite oxidative stress induced by hydrogen peroxide exposure. Granulosa-like KGN (human granulosa-like cell line) cells will be MK-7 pretreated (0.1-10 μM) before sublethal H2O2 exposure, and outcomes assessed will be mitochondrial membrane potential, ATP production, mtDNA copy number, intracellular ROS, cell viability, apoptosis, and anti-Müllerian hormone (AMH) secretion. It is hypothesized that MK-7 will limit H2O2-induced mitochondrial dysfunction, ROS accumulation, mtDNA depletion, apoptosis, and AMH decrease. Should such mitochondrial protection be observed in granulosa-like cells, it will be the first novel mechanistic data suggesting vitamin K2 might provide advantages in an effort to maintain ovarian reserve-driven function to justify subsequent translational studies in the field of ovarian aging and infertility.

Combination Therapy for Early-Onset Pre-Eclampsia: sFlt-1 e15a Silencing and VEGF-A mRNA Restoration in a Guinea Pig Model

George Zeng, BMSc Student [1], Jason J. Chu, BHSc Student [2], Ryan I. Moffatt, BHSc Student [2], Yifan Han, BHSc Student [2]
[1] Western University, London, ON, Canada N6A 3K7
[2] McMaster University, Hamilton, ON, Canada L8S 4L8

Pre-eclampsia is a pregnancy-specific hypertensive disorder characterized by maternal endothelial dysfunction and placental maldevelopment. Unfortunately, this disorder affects ~8% of pregnancies worldwide and contributes significantly to both maternal and neonatal morbidity/mortality. Early-onset pre-eclampsia (EO-PE), appearing before 34 weeks of gestation, is often caused by impaired trophoblast invasion and incomplete spiral artery remodeling. It is also commonly associated with

chronic placental hypoxia and the release of the anti-angiogenic factor soluble fms-like tyrosine kinase-1 (sFlt-1). This is particularly evident in the placenta-specific e15a isoform. Current approaches targeting sFlt-1 via siRNA reduce circulating levels and lower maternal blood pressure in preclinical models, but do not fully restore placental vascular function.
Here, we propose a synergistic therapeutic strategy combining sFlt-1 e15a-targeted siRNA with PLGA-emulsified VEGF-A mRNA lipid nanoparticles (LNPs) to simultaneously suppress anti-angiogenic signals and enhance pro-angiogenic signaling. Using a placenta-on-chip model, we will first optimize delivery and confirm VEGF-A expression under hypoxic conditions. Afterwards, in vivo, pregnant guinea pigs will undergo reduced uteroplacental perfusion (RUPP) to model EO-PE and will receive either VEGF-A mRNA-LNP, sFlt-1 e15a siRNA, our proposed combination therapy, or no treatment. Mean arterial blood pressure will be monitored weekly, and placental vascularization and trophoblast function will be assessed. We hypothesize that combined therapy will more effectively restore placental angiogenic balance, reduce maternal hypertension, and improve fetal oxygenation and nutrient delivery compared to either monotherapy. This study provides a plausible and targeted approach to address the core symptoms of EO-PE and may serve as a proof-of-concept for combination therapeutics capable of improving maternal and fetal outcomes in early-onset pre-eclampsia.

Optimizing Nicotinamide Riboside Therapy for Placental Health: Mapping Dose-Timing-Sex Effects in a Synthetic Human Reproductive Model

Jake Heinbuch, BHSc Student [1], Luka Parikh, BHSc Student [1], Luke Heinbuch, BHSc Student [1]
[1] Queen's University, Kingston, ON, Canada K7L 3N6

Gestational obesity hinders placental development through chronic inflammation, impaired angiogenesis, and mitochondrial dysfunction, which compromises fetal growth and long-term health. The root mechanism driving these abnormalities is placental nicotinamide adenine dinucleotide (NAD+) depletion, caused by inflammation-induced poly(ADP-ribose) polymerase (PARP) overactivation. Recent studies illustrate that nicotinamide riboside (NR), a vitamin B3-derived NAD+ precursor, can restore placental NAD+ levels, improving mitochondrial function and fetal growth. However, therapeutic potential remains limited by three gaps: the optimal dosage, the optimal timing of administration, and whether male and female placental tissues respond differently. These gaps prevent translation of NR-based interventions into targeted therapies for improving placental function and fetal health in pregnancies affected by obesity. To address this, this study uses an in vitro trophoblast organoid model grown in a synthetic PEG-based hydrogel to test how NR dosage and timing affect placental cell function under mimicked obesity inflammatory stress. Organoids will be derived from XX and XY placental tissues and exposed to varying NR doses (0.05-1.0 mM) and treatment windows paralleling early, mid, and late organoid development. Outcomes of the study include NAD+ restoration, mitochondrial respiration, inflammatory cytokine levels, angiogenic signaling (PlGF, sFlt-1), as well as trophoblast differentiation markers. By generating the first response map correlating to sex, dose, and timing for NR in placental tissue, this study aims to identify the optimal NR treatment strategy and provide a framework for developing targeted therapies to improve placental health in obese pregnancies.

Investigating Vascular Co-Option as a Non-Angiogenic Survival Mechanism in Endometriosis: A Research Protocol

Ben Chernin, BHSc Student [1], Manaar Hussain, BHSc Student [1], Nawal Youssef, BHSc Student [1]
[1] Queen's University, Kingston, ON, Canada K7L 3N6

Endometriosis is a chronic, estrogen-dependent condition characterized by the ectopic growth of endometrium-like tissue, leading to pelvic pain, infertility, and significant reductions in quality of life. A principal feature of endometriosis is the ability of ectopic lesions to survive and proliferate in hypoxic environments. Traditional models attribute this vascular support to hypoxia-driven sprouting angiogenesis; however, the limited effects of anti-angiogenic therapies indicate that angiogenesis alone cannot fully explain lesion persistence, suggesting the participation of additional vascular strategies. In oncology, resistance to angiogenic blockade commonly stems from vascular co-option, a non-angiogenic mechanism that permits tumor cells to multiply by hijacking pre-existing host vessels. Because endometriosis lesions exhibit malignant-like traits, including invasion, recurrence, and microenvironmental remodeling, it is plausible that they may similarly adopt alternative vascular survival strategies. Using a rat autologous transplantation model, lesions will be collected at sequential time points (days 3-28) and compared with a SHAM-operated control group using histological, immunohistochemical, and 3D imaging approaches. H&E staining defines lesion-host architecture, while immunohistochemistry with endothelial (CD31 and VE-cadherin), mural (α-SMA and TAGLN), and angiogenic activation markers (Ki-67 and ESM1) differentiate mature host vessels from angiogenic sprouts. Complementary 3D tissue clearing and confocal microscopy enables high-resolution reconstruction of vessel continuity and assessment of lesion volume relative to vascular branching. This work seeks to establish whether endometriotic lesions can expand along pre-existing vasculature, suggesting a previously unrecognized

vascular mechanism. Identifying vascular co-option in endometriosis would enrich current models of disease pathophysiology and introduce new non-angiogenic targets for therapy.

Targeted Macrophage Engineering for Lesion-Specific Endometriosis Immunotherapy

Arthur Rupiya Kinja, BScN Student [1], Hoorain Sadiq, BMSc Student [2], Isabel Hodara, BMSc Student [2], Manal Abdul-Rahim, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5
[2] Western University, London, ON, Canada N6A 3K7

Endometriosis is a chronic inflammatory disease that is defined as the presence of endometrium-like tissues outside the uterus that cause pain and infertility. Although hormonal therapies and surgery can help manage symptoms, they do not prevent the recurrence of lesions. Currently, there are no antigen-specific or lesion-targeted immunotherapies for endometriosis, which is why more precise treatment approaches are needed.
To address this gap, this study evaluates whether dual-antigen synNotch CAR-macrophages can selectively target superficial peritoneal endometriosis lesions, the most common subtype of the disease. In this model, endometriosis will be surgically induced in C57BL/6 mice, and engineered EpCAM-activated FAP synNotch CAR-M will be administered intraperitoneally. To test this, five experimental groups will be included: a sham control, a disease control, unengineered macrophages, single-antigen CAR-M, and the dual-antigen synNotch CAR-M. Lesion burden, stromal fibrosis, cytokine profiles and off-target effects will be evaluated at study completion.
We expect that synNotch CAR-M will reduce lesion size and local inflammation while showing minimal off-target activity due to the dual antigen activation requirement. Overall, this approach may provide a more precise, lesion-specific immunotherapy for superficial peritoneal endometriosis and contribute to future advances in reproductive immunology.

Upper Division

Effect of Microplastics in Menstrual Products on the Dysbiosis of the Vaginal Microbiome in Relation to Bacterial Vaginosis

Elise O'Callaghan, HBSc Student [1], Justen Mattias Chan, BMSc Student [2], Tianni Zhao, BMSc Student [2], Yu Tong (Amy) Zheng, BMSc Student [2]
[1] Queen's University, Kingston, ON, Canada K7L 3N6
[2] Western University, London, ON, Canada N6A 3K7

As plastic use continues to grow globally, microplastics (MPs) have become an increasing concern for human health. Emerging studies show that MPs have been found in different areas of the female reproductive system. However, given the limited research on female reproductive health in scientific studies, the relationship between MPs and dysbiosis of the vaginal microbiome, a significant factor in bacterial vaginosis (BV), remains unclear. *Lactobacillus crispatus* plays a crucial role in fighting infections and makes up a significant population of the vaginal microbiome. This study aims to better understand how MPs can influence pre-established vaginal microbiomes by aiding in the growth of BV-associated bacteria, like *Gardnerella vaginalis*. By co-culturing VK2/E6E7 human vaginal epithelial cells with *L. crispatus* and *G. vaginalis* in varying concentrations of MPs, a dose-response curve was created to analyze biofilm production in the presence of MPs. It was hypothesized that the MPs would aid and increase *G. vaginalis* biofilm production, thus leading to a decrease in *L. crispatus* populations. Findings may clarify whether MPs contribute to BV onset or persistence and inform the development of safer menstrual products.

A Biodegradable PLGA-PEG Nanoparticle Encapsulating sFlt-1 siRNA Reduces Anti-Angiogenic Signaling and Improves Maternal Outcomes in Preeclampsia

Abdelrahman Fahmy, HBSc Student [1], Abhijit Sinha, HBSc Student [1], Diana Munteanu, BHSc Student [1], Jean-Paul Azzi, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Preeclampsia is a dangerous pregnancy complication that affects 2-8% of pregnancies worldwide, causing more than 50,000 maternal deaths and over 500,000 fetal deaths each year. It is caused by poor placental development, leading to maternal organ damage and poor fetal growth. Preeclampsia is associated with elevated sFlt-1 levels in the placenta, causing

angiogenic signalling and damage to uterine blood vessels. Earlier attempts to lower sFlt-1 with siRNA used older PEG-PLA nanoparticles that had safety concerns and showed only mild effects in normal pregnant mice. This proposal develops a safer nanoparticle made from the biodegradable polymer PLGA10K-PEG5K, a common biodegradable polymer where the 10K:5K ratio controls core firmness and surface stability. We first tightly pack the sFlt-1 siRNA using a helper molecule called PEI-C14, then encapsulate it within a PLGA core and form a protective PEG shell. PEG is widely used in FDA-approved drugs because it improves circulation time and reduces immune clearance, which are major advantages during pregnancy. These 50-60 nm particles load siRNA efficiently, remain stable in blood, and escape endosomes well inside placental cells, making them far more effective and safer than older PEG-PLA systems. We will test this nanoparticle in human trophoblast cells under low-oxygen stress to mimic early preeclampsia. This will show whether the particles can enter the cells and lower sFlt-1, thereby restoring healthier signals that support blood-vessel growth. These results will form an important first step before moving toward future testing in pregnancy models.

The Impact of Vaginal Dysbiosis on Inflammatory Markers in an Induced Endometriosis Model

Cadence Nash, HBSc Student [1], Joudie Afara, HBSc Student [1], Mashaal Duranai, HBSc Student [2]
[1] Western University, London, ON, Canada N6A 3K7
[2] Toronto Metropolitan University, Toronto, ON, Canada M5B 2K3

Endometriosis is an inflammatory condition that is characterized by the ectopic growth of endometrium-like tissue outside of the uterus, leading to symptoms like chronic pain and infertility. New studies highlight that patients with endometriosis frequently have vaginal dysbiosis, but it remains unclear if these microbial differences contribute to the heightened inflammatory environment associated with the disease. The purpose of this study is to determine whether dysbiosis of the vaginal microbiome worsens inflammation in a surgically induced mouse model of endometriosis. In order to test this hypothesis, endometriotic lesions will be introduced in C57BL/6 mice and maintained with exogenous 17β-estradiol supplementation. Mice will be assigned to either a topical clindamycin treatment group, designed to disrupt the vaginal microbiome, or an untreated control group. Based on prior research showing increased dysbiosis in endometriosis and the anti-inflammatory role of Lactobacillus-dominated vaginal microbiota, we expect clindamycin-treated mice to exhibit higher levels of inflammatory markers and lower vaginal pH compared to the controls. The findings from this study may help to establish a causative link between vaginal dysbiosis and endometriosis-associated inflammation, which could support future studies in non-hormonal microbiome-based therapies.

Recharging the Obese Placenta: Dual Targeting of PARP1 and NAD+ to Restore Trophoblast Mitochondrial Function in Human Organoids and a Gestational Obesity Mouse Model

Bryan Lin, HBSc Student [1], Lena Draca, HBSc Student [1], Majd Al-Aarg, HBSc Student [1], Tai Adewoye, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Maternal obesity now complicates nearly one-third of pregnancies and is strongly linked to preeclampsia, fetal growth restriction, and long-term cardiometabolic disease in offspring, yet no therapies directly target the placenta where much of this risk arises. Chronic low-grade inflammation in obese pregnancies activates poly(ADP-ribose) polymerase-1 (PARP1), a DNA-repair enzyme that consumes nicotinamide adenine dinucleotide (NAD+), leading to NAD+ depletion, mitochondrial dysfunction, and impaired trophoblast fusion and angiogenic signalling. We propose that this PARP1-driven "energy crisis" is a central, druggable mechanism of placental dysfunction in gestational obesity and that simultaneously blocking PARP1 and replenishing NAD+ can restore placental health and fetal growth. To test this, we will first use human trophoblast organoids grown on tunable polyethylene glycol hydrogels in a transwell system that models lean versus obese placental environments. We will expose organoids to lean or obese-inflammatory media and treat obese conditions with vehicle, nicotinamide riboside (an NAD+ precursor), a PARP1 inhibitor (olaparib), or the combination, then measure NAD+/NADH ratios, PARylation, mitochondrial respiration, trophoblast fusion markers, and angiogenic factors. In parallel, a diet-induced gestational obesity model in C57BL/6 mice will mirror these five treatment groups to assess maternal metabolism, placental bioenergetics and structure, and fetal growth at gestational day 17.5. By integrating mechanistic organoid studies with an in vivo pregnancy model, this project will clarify whether PARP1-driven NAD+ depletion is sufficient to disrupt placental function and whether dual PARP1 inhibition plus NAD+ supplementation can "recharge" the obese placenta, informing future placenta-targeted therapies for high-risk pregnancies in Canada.

The use of CRISPR base-editing of ARID1A mutation in endometrial organoids to improve endometrial receptivity

Jessica Chiem, HBSc Student [1], Rachel Phang-Lyn, HBSc Student [2], Rubeen Zaidi, HBSc Student [1], Sarah Al-Zubaidi, HBSc Student [3]
[1] University of Toronto, Toronto, ON, Canada M5S 3E2
[2] University of Waterloo, Waterloo, ON, Canada N2L 3G1
[3] University of Guelph, Guelph, ON, Canada N1G 2W1

Endometriosis is an estrogen-dependent disorder in which the uterine endometrial tissue is present outside of its expected location. It is additionally well known that there is a genetic component to endometriosis. Individuals with immediate family members affected by endometriosis have a seven times higher chance of developing the disorder, and over 50% of individuals with endometriosis are infertile. Scientific exploration of the genetic modules of this disorder may allow us to explore therapeutic approaches to increase fertility. This is crucial as many genetic mutations are expressed atypically, and thus, may be associated with endometrial receptivity. Specifically, ARID1A mutations have been associated with poor endometrial receptivity and poor implantation success rates. Although the ARID1A mutation is most typically associated with endometriosis-related ovarian cancers, this proposal aims to explore the significance of ARID1A in benign disorders. Within fertility, ARID1A is understood to enable estrogen and progesterone production and thus, is vital during early pregnancies as it facilitates decidualization. The objective is to assess whether gene correctional therapy on the ARID1A mutation may allow for increased estrogen and progesterone responsiveness and regulation of cytokine signalling. The methodology includes employing CRISPR base-editing correction on ARID1A mutations using hormone-responsive human endometrial organoids. Organoids are subdivided into three experimental groups (n=3) and are electroporated with ribonucleoprotein complexes to assess pathological differences.

Extracellular Vesicle Therapy for Endometriosis-Associated Infertility: A Mouse Model Study

Charlotte Poirier, BScH Student [1], Daria Timoshenko, BScH Student [1], Emily Ma, BScH Student [1], Kiana Labbaf, BScH Student [1]
[1] Queen's University, Kingston, ON, Canada K7L 3N6

Endometriosis is a common reproductive disorder associated with infertility and is characterized by ectopic endometrial growth, and dysregulated hormonal and inflammatory signaling. Unfortunately, effective non-surgical therapies remain limited. Our proposal explores a therapeutic strategy that increases miRNA-30d concentration using engineered extracellular vesicles in syngeneic mouse models. Endometriosis-like lesions will be induced into the mice via intraperitoneal injection of decidualized endometrial fragments collected from donor mice in proestrus. miRNA-30d will be loaded into extracellular vesicles using electroporation and introduced into recipient mice for cellular uptake. Fertility outcomes in mice will be assessed by inducing pseudopregnancy through cervical manipulation, followed by non-surgical artificial insemination. Pregnancy rates will be compared across both treated and control groups. Our approach aims to improve understanding of the mechanisms involved in endometriosis-associated infertility and evaluate a targeted method to restore normal endometrial receptivity.

Targeting β-Arrestin-Mediated Oxytocin Receptor Desensitization as a Novel Strategy to Prevent Postpartum Hemorrhage

Samuel Martial, BHSc Student [1], Audrey Tatiotsop Kenou, BHSc Student [1], Benejah Moleko, HBSc Student [1], Eva Sadiot, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Uterine atony, the inability of the uterus to contract properly after birth, is the most common cause of postpartum hemorrhage (PPH), the main cause of maternal death globally. Despite being the first-line uterotonic, oxytocin's therapeutic efficacy decreases with protracted labor because of Oxytocin Receptor (OXTR) desensitization. This mechanism, which is mediated by β-arrestin/AP2-driven internalization, removes OXTR from the cell surface and disrupts the Gαq-dependent Ca2+ signaling that is required for sustained contractions. We propose preventing or reversing OXTR internalization by pharmacologically blocking the β-arrestin/AP2 interaction. We hypothesize that if the β-arrestin inhibitor Barbadin is applied after oxytocin-induced desensitization, then myometrial contractility will be restored through preserved receptor surface availability and reactivation of downstream Ca2+ signaling. This idea will be tested using ex vivo myometrial strips from Sprague-Dawley rats at the end of gestation. Extended oxytocin treatment will cause OXTR desensitization, after which tissues will be treated with Barbadin, oxytocin, carbetocin, atosiban, or a vehicle. Contractile function will be measured using

force recordings in the organ bath, and whether Barbadin stabilizes OXTR at the membrane and restores Gαq/PLCβ signaling will be evaluated using mechanistic tests like surface biotinylation of OXTR, confocal imaging of receptor localization, and Fluo-4 Ca imaging. If successful, this study will give the first experimental proof that stabilizing OXTR trafficking can restore uterine responsiveness, opening up a new approach for the development of novel "oxytocin-based" medications to prevent PPH globally.

Metabolic Regulation of Secretoneurin Signalling: SCG2/SN Control of GnRH-LH Pulsatility in Obesity-Associated Infertility

Ahmad Haris Jamal, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Obesity is a leading cause of infertility because it disrupts the pulsatile release of gonadotropin-releasing hormone (GnRH) and luteinizing hormone (LH) within the hypothalamic-pituitary-gonadal (HPG) axis. Current treatments use GnRH analogues and exogenous gonadotropins to induce ovulation, but they do not re-establish the underlying neuroendocrine pulse generator, so fertility often declines once therapy stops. Secretoneurin (SN), a peptide derived from the prohormone secretogranin-2 (Scg2), has recently been shown in zebrafish to be essential for female fertility: loss-of-function mutations in the zebrafish scg2 gene cause a severe LH secretion and ovulation defect that can be specifically rescued by synthetic SN. In mammals, the prohormone secretogranin-2 (SCG2) and its derived peptide secretoneurin (SN) are expressed in hypothalamic neuroendocrine neurons and LH-producing gonadotrophs, where SN amplifies GnRH-driven LH secretion. I hypothesize that high-fat-diet-induced obesity disrupts SCG2/SN signalling in hypothalamic and pituitary cells, blunting GnRHLH pulsatility and causing ovulatory failure, and that restoring SN can partially normalize LH pulses and fertility. To test this, I will define how closely the metabolic, hormonal, LH pulse, and fertility defects in obese female mice mirror those in Scg2 knockout (Scg2-/-) mice; map obesity-induced changes in SCG2/SN expression and processing in KNDy and GnRH neurons and LH gonadotrophs; and determine whether pulsatile SN delivery can rescue LH pulse dynamics and ovulatory function ex vivo and in vivo after obesity. This work will establish whether SCG2/SN is a mechanistic link between obesity and neuroendocrine infertility and will evaluate SN as a potential rhythm-based therapeutic target.

Impact of miR-29b Mimic on Expression of Matrix Metalloproteinase-2 and Matrix Metalloproteinase-9 in a Mouse Model of Endometriosis

Griff Bracchitta, HBSc Student [1], Jack Gillis, HBSc Student [1], Joey Li, HBSc Student [1], Keegan Yang, HBSc Student [1]
[1] University of Ottawa, Ottawa, ON, Canada K1N 6N5

Endometriosis is a pressing condition affecting 1 in 10 women globally and is caused by endometrial-like tissue growing in ectopic sites. One of the pathophysiological mechanisms of endometriosis is overexpression of the matrix metalloproteinase-2 and 9 genes (MMP2/9) which code for the MMP2 and MMP9 proteins respectively. MMP2/9 are proteinase enzymes which degrade the mesothelial basement membrane and extracellular matrix by targeted digestion of type IV collagen. Overexpression of MMP2/9 in endometriotic stromal cells facilitates an increase in the implantability of these cells, allowing them to establish ectopic lesions following their reflux via retrograde menstruation. Furthermore, MMP2/9 are implicated in angiogenesis, enabling vascularization and persistence of ectopic lesions. Because overexpression of MMP2/9 genes promotes lesion invasion, angiogenesis, and growth, inhibition of MMP2/9 has been investigated as a potential therapeutic approach that could lead to less lesion propagation, lessening the symptoms of endometriosis. MicroRNA-29b (miR-29b) has shown promise in inhibiting translation of MMP2/9 transcripts in various cell lines and cultures, and for that reason is the primary subject of our study. By injecting increasing concentrations of mouse-specific miR-29b in mouse endometrial stromal cells in vitro, incubating, then measuring MMP2/9 levels using a dose response western blot, we aim to identify the effect that miR-29b has on MMP2/9 expression. If miR-29b shows significant downregulation in the protein expression of MMP2/9, it could have future therapeutic relevance in humans as an effective drug treatment, lessening symptoms of endometriosis and reducing lesion spread.

Conflicts of Interest
The authors declare that they have no conflict of interest.

Authors' Contributions
CK: Co-President of SciNapse and Co-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.
SH: Co-President of SciNapse and Co-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.
MD: 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.

Acknowledgments
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: Chris Kachi and Sarah Hussain as Co-Presidents of SciNapse and Co-Chairs of the planning committee; Moumitta Dutta as President of URI; Jebreel Alayche as Vice-President of Finances of URI; Aysha Aftab as Vice-President of Outreach; Abha Ranjitkar as Vice-President of Operations; Joseph Ghossein and Hadassa Mathivathanan as Events Coordinators; Will Meisner as Communications Representative; Émilie Marion as Social Media and Website Coordinator; Tony Lin, Mae Padie, and Jade Gamelin Kao as USCC Mentors; Halimo Daher and Franciska Konji as uOttawa Campus Leaders; Rita Murali, Amy Shi, and Moumita Dutta as Graphic Designers; Ariel Tayo-Ajimoko as External Campus Coordinator; and Jin-Young Bang, Liza Zhu, and Benjamin Mugisha as Logistics and Statistics Coordinators. The USCC would not have been successful without the contribution and work of the multiple chapters of the SciNapse USCC situated across other universities in Ontario. Finally, we want to acknowledge all the professors, researchers, 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.

Article Information
Managing Editor: Jeremy Y. Ng
Article Dates: Received Feb 19 26; Published Mar 05 26

Citation
Please cite this article as follows:
Kachi C, Hussain S, Dutta M. Scinapse 2025-2026 undergraduate science case competition: innovations in reproductive health. URNCST Journal. 2026 Mar 05: 10(3). https://urncst.com/index.php/urncst/article/view/1066
DOI Link: https://doi.org/10.26685/urncst.1066

URNCST Journal
"Research in Earnest"

Funded by the Government of Canada | Canada