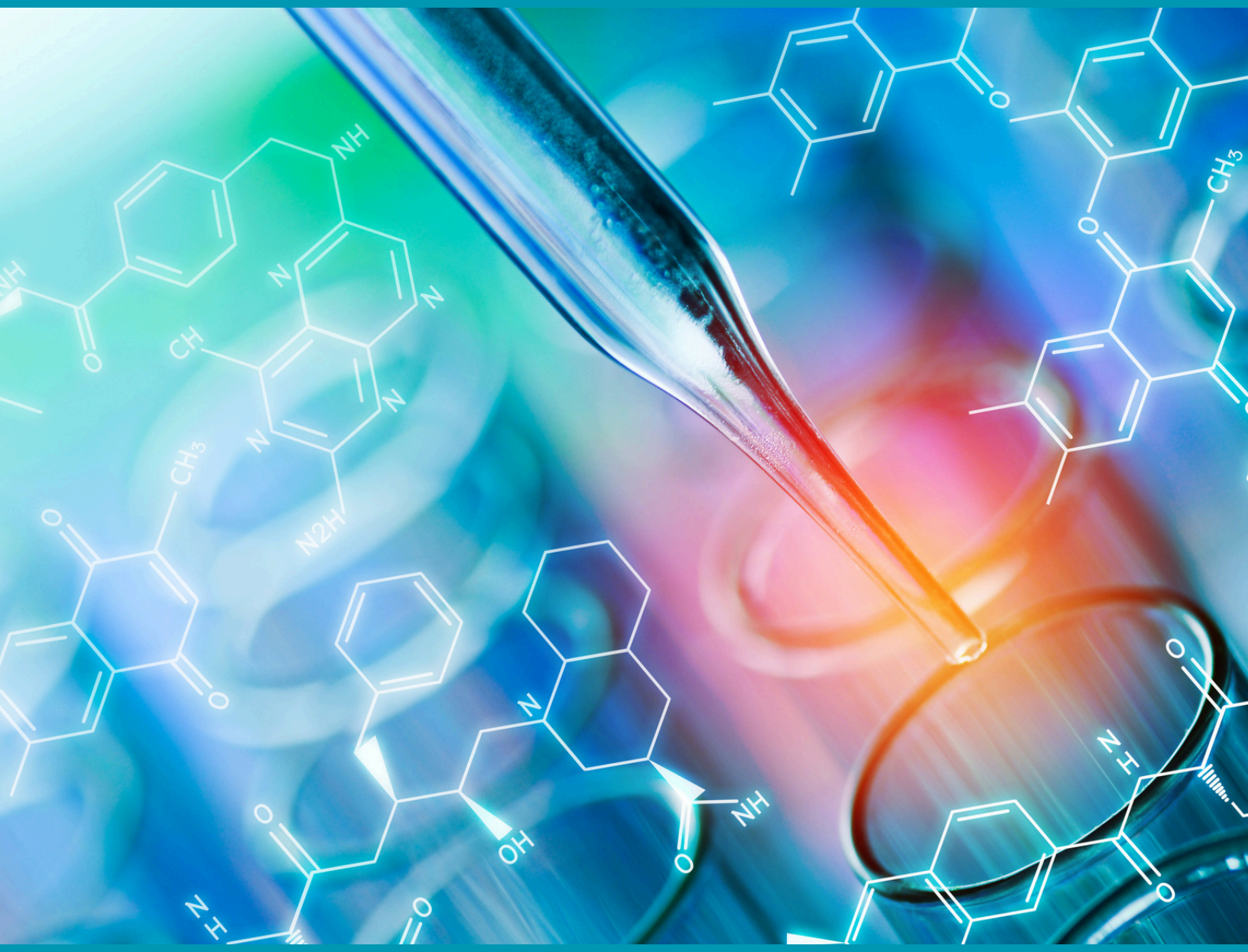
16TH ANNUAL BIOCHEMISTRY SYMPOSIUM



FRIDAY, JULY 31ST, 2026

Core Science Facility
Memorial University of Newfoundland and Labrador
St. John's, NL

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DR. ZAHRA FARAHNAK
Faculty Advisor

SYMPOSIUM SCHEDULE

MORNING

9:00AM - 12:00PM

9:00AM - DR. SHERRI CHRISTIAN & EMILY ROSSITER

Opening remarks

9:10AM - MOLLY GIBSON

Creation of a Nascently Fluorescent TAAR1 Protein Using CRISPR-Cas9 and Unnatural Amino Acid Technologies.

9:25AM - ALAIN MUTANGANA

Targeting the Gram-negative bacterial coat: multi-scale characterization of the O-antigen ligase (WaaL) across clinically relevant pathogens

9:40AM - BEAUTY DAM

Regulatory role of GRP75 in nuclear AGO2-mediated gene silencing and neuronal differentiation

9:55AM - GRANT KELLY

Adipocyte-secreted LOX is a Key Mediator of Mesenchymal-to-Epithelial Transition in Breast Cancer Cells

COFFEE BREAK

10:10AM - 10:55AM

10:55AM - ASHA DE SILVA

Parenterally fed neonatal piglets require more dietary methionine to optimize transmethylation than for protein synthesis

11:10AM - EMILY ROSSITER

Investigating the role of heme oxygenase-1 in macrophage cholesterol handling and lipid metabolism

SYMPOSIUM SCHEDULE

MORNING

9:00AM - 12:00PM

11:25AM - HANNAH WHITE

Docosahexaenoic acid (DHA) supplementation can potentially increase muscle n-3 fatty acids and muscle cross-sectional area in rats fed a high fat diet

11:40AM - SOGAND SASAN-MOGHADAM

Exploring the Role of Bacillus subtilis Teichoic acid in Cell Selectivity of AMPs

LUNCH

12:00PM - 1:00PM

AFTERNOON

1:00PM - 4:50PM

1:00PM - DR. KYLY WHITFIELD

Keynote presentation

2:00PM - POSTER PRESENTATIONS AND NETWORKING

Coffee available at this time

3:30PM - FATEMA ELGAMMAL

Comparing the Metabolic and Neurobehavioral Effects of Ulotaront vs Semaglutide in Diet Induced Obese Mice

3:45PM - KADY MEANEY

Examining the Associations Between Diet Quality, Student Success, and Cognitive Function in Undergraduates

SYMPOSIUM SCHEDULE

AFTERNOON

1:00PM - 4:50PM

4:00PM - RASHID JAFARDOUSTBOSTANI

CD24 Stimulation of Donor B Cells Enhances Extracellular Vesicle-Mediated GFP and IgM Transfer Through Actin-Dependent Macropinocytic Uptake

4:15PM - NARMADA WEERAKKODY

Presentation TBD.

4:30PM - KAITLYN MAYNE

Unraveling Signalling Dynamics of CD24 in B Cells

4:45PM - BIOCHEMISTRY GRADUATE SOCIETY

Closing remarks

EVENING

5:15PM - 8:00PM

5:15PM - AWARD CEREMONY AND RECEPTION

Join us in the CSF-1302 for an award ceremony followed by a reception with food and drinks in the Whale Atrium!

THANK YOU TO OUR VOLUNTEERS

HANNAH WHITE
MOLLY GIBSON
SOGAND SASAN-MOGHADAM
JULIAN SUTTON
HANNAH RIDEOUT
AMRITHA AMBY SINDHU
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THANK YOU TO OUR JUDGES

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WELCOME FROM THE BCGS

EMILY ROSSITER
BCGS CHAIRPERSON

On behalf of the Biochemistry Graduate Society (BCGS), it is my pleasure to welcome you to the 16th Annual Biochemistry Summer Symposium.
This year's symposium marks an exciting milestone as our first under the newly named Department of Human Biosciences. As our department enters a new chapter, this event continues a long-standing tradition of showcasing the outstanding research and achievements of our undergraduate and graduate students, postdoctoral fellows, and faculty.

The Summer Symposium is more than an opportunity to present research; it is a chance to share ideas, engage in thoughtful discussion, and strengthen the connections within our academic community. We hope today's presentations inspire curiosity, encourage collaboration, and highlight the innovative research across our department.

The BCGS is grateful to everyone who helped make this event possible, including our organizing committee, faculty and staff, volunteers, judges, sponsors, and, most importantly, our presenters. Your dedication, enthusiasm, and support continue to make this symposium a valued tradition.

We are also delighted to welcome our keynote speaker, Dr. Kyly Whitfield, and thank her for joining us to share her expertise.

Whether you are presenting your work, supporting a colleague, or joining us as a guest, thank you for being part of today's symposium. We hope you enjoy the presentations, make new connections, and take pride in the remarkable research being conducted within our department.

Welcome, and enjoy the symposium!

WELCOME FROM THE DEPARTMENT

DR. ROBERT BERTOLO
DEPARTMENT HEAD

Welcome to the 16th annual Department Symposium! I suppose it could be considered the first "Human Biosciences" Symposium as well! This event has become a key annual event for our graduate and undergraduate research students, featuring the excellent research ongoing in HUBI. The symposium is particularly important for our graduate students, who not only present their fascinating research, but also plan, raise funds, select and arrange the keynote speaker, and organize all of the many details necessary for this event to run seamlessly. These are not trivial tasks, and I applaud all of the work that goes on behind the scenes by the organizing committee (Emily Rossiter, Fatema Elgammal, Narmada Weerakkody, Ihechi Marcus, Anna-Marie Cooney, Asha De Silva, and Ashley Stanford) to make this event a great success. And, of course, we need to congratulate our faculty mentor, Dr. Zahra Farahnak, without whom none of this would have been possible. This event is such a wonderful event to attend because of the excellent, well-prepared research presentations by our superb students. The symposium is a key opportunity for our undergraduate students to experience research presentations and interactions, and for our graduate students to present new, exciting research and add another presentation to their CV. I cannot overestimate the importance of having a strong list of presentations for future applications for awards and jobs. And I am delighted to welcome Dr. Kyly Whitfield, our fabulous keynote speaker. You will be captivated by her unending enthusiasm and passion for research and mentoring students!
As you enjoy the symposium, please acknowledge the folks who put the work into making this event a success, and interact with your colleagues and trainees to make it a great social event! Congratulations to all!

KEYNOTE SPEAKER

DR. KYLY WHITFIELD

DEPARTMENT CHAIR, ASSOCIATE PROFESSOR, DEPARTMENT OF APPLIED HUMAN NUTRITION, MOUNT SAINT VINCENT UNIVERSITY

Dr. Kyly Whitfield is an Associate Professor and Chair of the Department of Applied Human Nutrition at Mount Saint Vincent University in Halifax, where she leads the Milk and Micronutrient Assessment (MAMA) Lab. She is passionate about maternal and infant nutrition, with interests in identifying and combating micronutrient deficiencies and understanding feeding during 'the first thousand days' window, from conception through 2 years. A major focus of the MAMA Lab is identifying culturally appropriate public health interventions to combat micronutrient deficiencies in low-resource settings, especially thiamine deficiency among breastfed infants in Southeast Asia. Another area of interest is examining breastfeeding culture and social infant feeding norms in Canada.

KEYNOTE ABSTRACT

THIAMINE DEFICIENCY IN CAMBODIA: BERIBERI AND BEYOND

Thiamine (vitamin B1) deficiency has been described as 'the forgotten disease of Asia', often viewed as a historic relic, but unfortunately deficiency remains widespread in low-resource settings where rice is the dietary staple, including throughout South and Southeast Asia, numerous Pacific Island Nations, and throughout Africa. Thiamine deficiency causes potentially fatal beriberi, but more recent evidence suggests that early life sub-clinical thiamine deficiency, not severe enough to trigger clinical assessment and treatment, leads to persistent language and motor delays. Thiamine deficiency may put millions of infants at risk of neurocognitive deficits, undercutting life-long well-being and productivity. In this keynote, Whitfield will explore thiamine as an often-overlooked micronutrient of immense importance to early life development through a series of studies conducted (and ongoing) in Cambodia and other low- and middle-income countries with monotonous, carbohydrate-dense diets.

POSTER PRESENTATIONS

#1 - ISAAC OMEH

Microarray analysis of gene expression and metabolic pathways underlying hypertension and adiposity in intrauterine growth-restricted Yucatan pigs

#2 - ANNA-MARIE COONEY

Dietary Vitamin D May Reduce Fat Accumulation in Female Rats on a High-Fat Diet

#3 - NIMEESHA VANKADARA

Identifying the Purinergic Receptor Targeted by TAAR1 During ATP-Induced Inflammatory Signalling

#4 - YAR MALUAL

Comparison of SCFA profiles in cecal and fecal samples from semaglutide- and ulotorant- treated mice.

#5 - SUSHMITHA RAMAKRISHNA

Regulatory role of Tbx3 in nuclear miRNA-guided gene regulation in the pathogenesis of triple-negative breast cancer

#6 - ALEXANDER POWER

Investigating the Hemolytic Activity of Antimicrobial Peptides Using Molecular Dynamics Simulations

#7 - MACKLIN PELLEY

Investigating the Interactions of Chinese Red Sage Natural Products with p38 MAPK in Triple-Negative Breast Cancer

#8 - ANDREW HICKEY

Computational Investigation of the Structure-Activity Relationship of the Non-Hemolytic Antimicrobial Peptide Thurincin H

POSTER PRESENTATIONS

#9 - IHECHI MARCUS

Role of MicroRNA-132 in Regulating Cholesterol Efflux and Foam Cell Formation in Macrophages

#10 - ALYSSA DOBER

Docosahexaenoic Acid supplementation may attenuate high-fat diet-induced reductions in muscle cross-sectional area in rats

#11 - AGAPE POMROY

Investigating the Impact of Glucose, Fructose, and Glucolipotoxic Conditions on Lipid Accumulation in Adipocytes

#12 - CHATURRVI MARUTHIREDDY

Investigating the Autologous Extracellular Vesicle Transfer in B-lymphoblastic Leukemia Cells Stained with a Lipophilic Dye

#13 - KERTHIKA DEVI A

High-Pressure Processing as a Green Pre-treatment Strategy for Enhanced Saponin Recovery from North Atlantic Sea Cucumber

#14 - AMRITHA AMBY SINDHU

Fish oil versus flaxseed oil: A comparative study on pregnancy and fetal neurodevelopment

#15 - KYLE WARREN

The Ligand-Dependency of hTAAR1 Conformational Ensembles: A Scaffold for Allosteric Site Characterization

#16 - LEAH MARCANTONIO

An investigation of the influence of adipocytes and adipocyte-derived lysyl oxidase on the plasticity of MDA-MB-231 breast cancer metastatic variants

POSTER PRESENTATIONS

#17 - RUMANA IRFAN

Evaluation of Oxidant Load in Neonatal Parenteral Nutrition With and Without Antioxidant Supplementation

#18 - KAITLYN GATHERALL

Optimizing Arginine Intake in Parenteral Nutrition for Neonates

#19 - SAMANTHA NORMAN

Determining the Mechanism of Stability for B-Cell Receptors Transferred by Extracellular Vesicles

ORAL PRESENTATION ABSTRACTS

CREATION OF A NASCENTLY FLUORESCENT TAAR1 PROTEIN USING CRISPR-CAS9 AND UNNATURAL AMINO ACID TECHNOLOGIES.

MOLLY GIBSON & MARK D. BERRY

Trace amine-associated receptors (TAARs) are a family of G protein-coupled receptors with 6 functional TAAR isoforms in humans. Unlike most GPCRs, TAAR1 appears to be predominantly intracellular, although the precise sub-cellular compartment has rarely been conclusively demonstrated. This study aims to establish an approach for real-time TAAR1 protein tracking to be achieved within living immune cells, while the protein remains functional and at physiological levels in the cell. Using CRISPR-Cas9, tryptophan 18 within the TAAR1 protein sequence was targeted and mutated to create an amber stop codon in this position effectively creating a TAAR1 knock-out cell line that was confirmed using immunocytochemistry. The resulting cell line then underwent a secondary transfection with a synthetic aminoacyl tRNA synthetase that recognizes the amber stop codon as a coding sequence for the unnatural, fluorescent tryptophan derivative ANAP. This will simultaneously re-express TAAR1 and create a nascently fluorescent TAAR1 protein once ANAP is introduced to the cells. Preliminary studies have confirmed the feasibility of this approach. On-going studies will optimize the secondary transfection, to allow future work to determine the sub-cellular localization of TAAR1 under different immune cell activation conditions, as well as live cell imaging to determine TAAR1 turn-over kinetics.

ORAL PRESENTATION ABSTRACTS

TARGETING THE GRAM-NEGATIVE BACTERIAL COAT: MULTI-SCALE CHARACTERIZATION OF THE O-ANTIGEN LIGASE (WAAL) ACROSS CLINICALLY RELEVANT PATHOGENS

ALAIN MUTANGANA & KATIE WILSON

Antibiotic resistance is one of the most pressing threats to global health, and Gram-negative bacteria are among the hardest to treat. They are enclosed by a double-membrane envelope that keeps many antibiotics out and shields the cell from the immune system. A key component of this protective outer membrane is lipopolysaccharide (LPS), and its biosynthetic pathway has emerged as a promising target for the development of new antibiotics. The inner-membrane enzyme O-antigen ligase (WaaL) carries out a crucial late step of LPS assembly, attaching the O-antigen chain to the lipid A–core. Disrupting this step leaves bacteria more vulnerable and shows to cause death, making WaaL an attractive drug target. Yet despite its importance, the structural and mechanistic basis of WaaL function remains poorly understood. In this study, we employed a multi-scale computational approach to investigate the structure–dynamics–function relationships of WaaL within modeled, near-native membrane environments from four pathogens: E. coli, E. cloacae, K. pneumoniae, and P. aeruginosa. Long-timescale, low-resolution (coarse-grained) simulations revealed that the protein embeds within the membrane and reshapes its local environment to match the hydrophobic region, while preferentially recruiting negatively charged lipids that form transient, functionally supportive interactions. Higher-resolution simulations are now being used to examine how the substrate engages the active site and to develop a mechanistic model of the ligation reaction. Overall, this research aims to identify features that are shared or unique across species and to guide the design of broad-spectrum or species-specific inhibitors.

ORAL PRESENTATION ABSTRACTS

REGULATORY ROLE OF GRP75 IN NUCLEAR AGO2-MEDIATED GENE SILENCING AND NEURONAL DIFFERENTIATION

BEAUTY DAM & PAVAN KUMAR KAKUMANI

MicroRNAs (miRNAs) are essential regulators of gene expression and act primarily through the Argonaute 2 (AGO2) protein during post-transcriptional gene silencing. While miRNA-AGO2 interaction is well established in the cytoplasm, AGO2 also functions in the nucleus and mitochondria to influence core processes, such as neuronal differentiation. Dysregulation of this pathway is implicated in neurodegenerative conditions like Parkinson's disease (PD). However, the exact mechanisms that guide AGO2 to these different locations outside the cytosol, and how they affect neuronal fate, remain unclear. Here, we aim to address this knowledge gap by investigating the role of the mitochondrial chaperone protein GRP75, which translocates to the nucleus during neuronal differentiation. We hypothesize that GRP75 regulates AGO2's subcellular localization to the nucleus and miRNA function underlying neuronal differentiation. We will use RA-BDNF-induced differentiation, nuclear fractionation, transfection, immunoprecipitation, and western blotting to determine whether GRP75 promotes AGO2 nuclear localization and to assess the disruptive effect of its PD-associated variant, P509S. We anticipate that wild-type GRP75, unlike the pathogenic variant, will aid nuclear miRISC assembly, promoting neurogenic outcomes. Our findings will highlight GRP75 as a novel regulator of compartment-specific AGO2 function, potentially providing essential insights into neuronal development and how its disruption contributes to neurodegenerative diseases.

ORAL PRESENTATION ABSTRACTS

ADIPOCYTE-SECRETED LOX IS A KEY MEDIATOR OF MESENCHYMAL-TO-EPITHELIAL TRANSITION IN BREAST CANCER CELLS

GRANT KELLY, NIKITHA K. PALLEGAR, EMAN ELBAKRY, PERE MIQUEL MORLA-BARCELO, ALICIA M. VILORIA-PETIT, & SHERRI L. CHRISTIAN

Metastasis is the leading cause of death in breast cancer (BC) patients. Metastatic risk is correlated with obesity, defined by an overabundance of adipocytes. Adipocytes secrete proteins that cause epithelial-to-mesenchymal transition (EMT) in BC cells. The cells lose cell-cell interactivity and gain motility, enabling dissemination during early stages of metastasis. The role of adipocytes in later stages[CSL2.1], however, is not known. We have previously shown that adipocytes cause BC cells to undergo mesenchymal-to-epithelial transition (MET) which is the reverse of EMT. MET promotes metastatic colonization as the re-acquired epithelial characteristics promote tumour growth. We discovered that adipocytes secrete the enzyme LOX which may mediate MET in BC cells. Once cleaved by ADAMTS2, LOX remodels the extracellular matrix, which plays a significant role in EMT and MET. Therefore, we sought to determine if adipocyte-secreted LOX is necessary to induce MET in BC cells. Chemical and genetic inhibition of LOX restored the mesenchymal phenotype of BC cells in co-culture. Furthermore, single cell RNA sequencing on co-cultures revealed LOX inhibition leads to increased overall expression of EMT genes in BC cells. Together, these data show LOX activity is critical in BC cell MET. The exact mechanism of action is unknown but appears to involve ADAMTS2. BC cells lacking ADAMTS2 retained their mesenchymal phenotype, even after co-culture.[CSL3.1] Overall, we have demonstrated the crucial roles of LOX and ADAMTS2 in adipocyte-driven BC MET which may be a significant promoter of metastatic colonization. However, further work is needed to confirm LOX and ADAMTS2's roles in vivo.

ORAL PRESENTATION ABSTRACTS

PARENTERALLY FED NEONATAL PIGLETS REQUIRE MORE DIETARY METHIONINE TO OPTIMIZE TRANSMETHYLATION THAN FOR PROTEIN SYNTHESIS

ASHA DE SIVA, JANET BRUNTON, & ROBERT BERTOLO

Total Parenteral Nutrition (TPN) is a live-saving diet strategy given intravenously to preterm neonates who cannot feed by mouth. A key nutrient in TPN is methionine, which is needed for synthesis of protein and key metabolites, as well as for regulating gene expression patterns that can program risk of diseases later in life. We have shown that if methionine is diverted to one metabolite, it is then unavailable for other metabolites. Hence, it is essential to establish the amount of methionine required for each metabolite. Previous studies have determined a methionine requirement for TPN-fed pigs based only on the methionine requirement for whole-body protein synthesis. It is still unclear how much methionine is used to synthesize important metabolites like creatine, phosphatidylcholine, and DNA methylation. We hypothesized that piglets require more dietary methionine for these non-protein pathways. We used 18 neonatal piglets and fed them with different methionine levels in a TPN diet and measured the synthesis of various metabolites. We found that higher levels of methionine were needed to support metabolites synthesis than that for overall whole-body protein synthesis. These findings provide recommendations for optimizing methionine levels in neonatal TPN to better support early development and long-term health.

ORAL PRESENTATION ABSTRACTS

DOCOSAHEXAENOIC ACID (DHA) SUPPLEMENTATION CAN POTENTIALLY INCREASE MUSCLE N-3 FATTY ACIDS AND MUSCLE CROSS-SECTIONAL AREA IN RATS FED A HIGH FAT DIET

HANNAH WHITE, ANNA-MARIE COONEY, NARGES GHORBANI BAVANI, ELIZABETH CHIA, & ZAHRA FARAHNAK

Recent evidence suggests vitamin D and docosahexaenoic acid (DHA) can reduce fat accumulation, improving muscle metabolism. This study assesses the impact of vitamin D, DHA and co-supplementation on muscle metabolism in rats fed a high fat (HF) diet. Eight-week-old male and female Sprague-Dawley rats (n=95) were randomly assigned to either control (AIN-93M), HF diet (35% kcal fat), HF+low vit. D (D) (0.05 IU/g), HF+D (3 IU/g), HF+DHA (1% dietary weight), or HF + DHA (1%) + D (3 IU/g) for 8 weeks. Muscle grip strength was measured at week 0, 4 and 8, along with collection of blood samples and muscle tissues (quadriceps, gastrocnemius and soleus). Data was tested using Linear Mixed Model and Spearman's correlation ($P<0.05$). Total muscle mass was higher in HF+DHA+D (12.05+0.8 g) (Mean + SEM) than controls (10.20+0.8 g,$P < 0.001$), with males having greater muscle mass ($P<0.001$). Forelimb+hindlimb muscle strength was higher in HF+DHA+D (1033.43+81.1 (gram force)) than control (1007.95+61.0,$P<0.001$). Interestingly, females exhibited higher muscle strength to weight ratios ($P<0.001$). Quadriceps muscle fibre cross-sectional area was higher in HF+DHA (4001.62+345.2 μm2) than HF (3351.80+130.5,$P=0.059$), with higher area and fibre density in males ($P<0.001$). Total n-3 PUFAs in serum and quadriceps were higher in the HF+DHA (12.03+1.7(%), 18.45+0.5, respectively) and HF+DHA+D (10.95+1.2, 19.26+0.4, respectively) groups than in the control (4.64+0.4, 11.28+0.4, $P<0.001$). Serum n-3 PUFAs correlated positively with muscle n-3 PUFAs ($r=0.74$, $P<0.001$). Preliminary findings showed DHA and DHA+D increased muscle n-3 PUFAs and cross-sectional area, suggesting a potential role of DHA in preserving muscle.

ORAL PRESENTATION ABSTRACTS

INVESTIGATING THE ROLE OF HEME OXYGENASE-1 IN MACROPHAGE CHOLESTEROL HANDLING AND LIPID METABOLISM

EMILY ROSSITER & ROBERT J. BROWN

Cardiovascular disease remains the leading cause of mortality worldwide, with atherosclerosis underlying most heart attacks and strokes. Macrophages play a central role in disease progression through lipid uptake, foam cell formation, and impaired cholesterol homeostasis. Heme oxygenase-1 (HO-1), encoded by HMOX1, is a stress-inducible enzyme that protects against oxidative stress and inflammation and is highly expressed in atherosclerotic plaques. Previous work from our laboratory identified HMOX1 as one of the most highly upregulated genes in macrophages exposed to lipoprotein lipase hydrolysis products, suggesting that HO-1 is activated in response to lipid stress. Whether HO-1 also regulates macrophage cholesterol handling and lipid metabolism, however, remains poorly understood. The objective of this study is to determine how HMOX1 knockdown influences macrophage cholesterol handling and lipid metabolism. THP-1-derived macrophages were transfected with HMOX1-targeting siRNA, and knockdown was confirmed by Western blotting. Preliminary characterization included assessment of proteins involved in cholesterol transport and lipid metabolism, together with Oil Red O staining to evaluate intracellular lipid accumulation. These studies establish a foundation for investigating the role of HO-1 in macrophage cholesterol homeostasis. Ongoing studies will determine how HO-1 regulates macrophage cholesterol handling through complementary analyses of gene expression, cholesterol efflux, lipidomic profiling, and oxidative stress, providing a more comprehensive understanding of its contribution to atherosclerosis.

ORAL PRESENTATION ABSTRACTS

EXPLORING THE ROLE OF BACILLUS SUBTILIS TEICHOIC ACID IN CELL SELECTIVITY OF AMPS

SOGAND SASAN-MOGHADAM, MICHAEL MORROW, & VALERIE BOOTH

Antimicrobial peptides (AMPs) are a growing approach to fight against antibiotic resistance in pathogenic bacteria. However, AMP interactions with the bacterial cell envelope components they encounter before reaching the cell membrane have not been studied. Additionally, the role of bacterial cell-envelope layers in AMP selectivity in the presence of blood cells (RBCs) remains unclear. This study probes AMP MSI-78-induced permeabilization in mixtures of RBCs and bacteria. We use zeta-potential measurements to quantify surface charge and membrane permeabilization in wild-type and teichoic acid (TA)-deficient Bacillus subtilis and RBC envelopes, thereby analyzing TA-dependent changes in net negative charge. We also use SYTOX Green flow cytometry to assess bacterial cell-envelope permeabilization and AMP selectivity in the presence of MSI-78, with or without RBCs. We found that MSI-78 peptides may damage the cell envelope through mechanisms that extend beyond simple charge interactions. Together, the data support a model in which removing wall teichoic acids (WTAs) destabilizes the B. subtilis cell envelope, even without MSI-78 peptides, while lipoteichoic acid (LTA) deficiency has a smaller effect compared to W-type cells. Certain TAs on the bacterial surface likely attract MSI-78 through specific interactions and modulate its ability to permeabilize the cell envelope. However, the unique lipids and proteins in the RBCs protect them from permeabilization. Overall, charge-based attraction and cell envelope composition are important factors in AMP selectivity. Some information on AMP-TA interactions may be useful for designing more selective and microbe-specific antibiotics.

ORAL PRESENTATION ABSTRACTS

COMPARING THE METABOLIC AND NEUROBEHAVIORAL EFFECTS OF ULOTARONT VS SEMAGLUTIDE IN DIET INDUCED OBESE MICE

FATEMA ELGAMMAL, MARK D. BERRY, & SCOTT V. HARDING

Type 2 diabetes and obesity are prevalent global metabolic disorders that frequently co-exist with metabolic and neurobehavioral comorbidities. Semaglutide is an effective anti-glycaemic and weight-loss agent that targets the glucagon-like peptide-1 receptor (GLP-1R). However, its clinical use is associated with limitations, including inaccessibility, pancreatitis risk, implied lifelong therapy, and potential psychiatric side effects. Ulotaront is a trace amine-associated receptor-1 (TAAR1) agonist, primarily developed as an antipsychotic, that has recently demonstrated a favourable metabolic profile. Interestingly, TAAR1 is co-expressed with GLP-1R in several tissues and shares similar signalling cascades, suggesting these agonists may share common therapeutic utility, although no direct comparison has been made. We will directly compare semaglutide and ulotaront across metabolic, cognitive, and behavioural outcomes while accounting for diet, sex, and age as biological variables. Male and female C57BL/6J mice will undergo baseline phenotyping following 8 weeks of a standard diet. Mice will then receive either standard chow or a high-fat/high-sugar diet (HFHS) for 16 or 36 weeks while receiving semaglutide or ulotaront. Metabolic and behavioural assessments, including glucose tolerance, body weight, food intake, energy expenditure, anxiety-like behaviour, alcohol preference and cognitive performance, will be conducted throughout each experimental panel. Preliminary *in vivo* findings suggest that ulotaront ameliorates HFHS diet-induced metabolic dysfunction. The research outcomes could identify interventions capable of simultaneously addressing metabolic and behavioural health, and ulotaront may find new therapeutic relevance beyond its traditional antipsychotic endpoints.

ORAL PRESENTATION ABSTRACTS

EXAMINING THE ASSOCIATIONS BETWEEN DIET QUALITY, STUDENT SUCCESS, AND COGNITIVE FUNCTION IN UNDERGRADUATES

KADY MEANEY & AMY TODD

Nutrition plays a significant role in cognitive function, including positive associations with healthy diet and academic success. Growing research in this area has measured many particular aspects of diet, such as fruit and vegetable intake, however overall diet quality is scarcely addressed. Furthermore, there is limited research assessing the diet quality of students pursuing Science, Technology, Engineering, and Mathematics (STEM) in particular. STEM students experience frequent dropouts and lack of self-efficacy, impacting their post-secondary experience. Therefore, for the purpose of this study, we define "Student Success" as a combination of GPA, retention, and student experience. In this study, we evaluate the association between diet quality and student success within the population of undergraduate STEM students at Memorial University of Newfoundland. A quantitative online survey was distributed to undergraduates in STEM departments at MUN to collect data on demographics, lifestyle factors, and student success measures. Survey participants were then given the option to continue to complete diet recalls through the Automated Self-Administered 24-Hour Dietary Assessment Tool (ASA24), used to measure diet quality. To evaluate the association between diet quality and student success using a multivariable path analysis model, we adjust for previous academic experience, and multiple lifestyle factors which have been extensively studied for their impact on academic success in higher education students. Future work extends this framework by examining pathways connecting diet quality and student success through neural and behavioural measures of cognitive function.

ORAL PRESENTATION ABSTRACTS

CD24 STIMULATION OF DONOR B CELLS ENHANCES EXTRACELLULAR VESICLE-MEDIATED GFP AND IGM TRANSFER THROUGH ACTIN-DEPENDENT MACROPINOCYTIC UPTAKE

RASHID JAFARDOUST & SHERRI CHRISTIAN

Extracellular vesicles (EVs) are membrane-bound nanoparticles released by most cell types that mediate cell-to-cell communication by transferring functional biomolecules, including proteins, nucleic acids, and lipids. Because the biological effects of EVs depend on their successful delivery to recipient cells, understanding the mechanisms that regulate EV uptake is essential for defining how EVs influence cellular function. Engagement of the cell surface protein CD24, which is highly expressed during B-cell development, stimulates EV secretion from B cells; however, the mechanisms by which these EVs are taken up by recipient B cells remain unclear. Here, we characterized the mechanism of EV uptake. EVs isolated from CD24-stimulated donor cells were incubated with recipient cells over multiple time points to determine the kinetics of GFP and IgM (B cell receptor) transfer by flow cytometry. Next, recipient cells were pretreated with pharmacological inhibitors targeting actin remodelling, dynamin-dependent endocytosis, or PI3K signalling for 30 minutes, then simultaneously incubated with EVs and the same inhibitors, followed by detection of GFP and IgM by flow cytometry. We found that recipient cells incubated with CD24-stimulated EVs exhibited significantly increased GFP and IgM uptake compared with control EVs across multiple time points (2–72 h), with the maximum uptake at 36 h. In addition, this study showed that EV uptake required actin remodelling but was independent of dynamin and PI3K. These findings support actin-dependent macropinocytosis as a major mechanism of EV internalization.

ORAL PRESENTATION ABSTRACTS

UNRAVELING SIGNALLING DYNAMICS OF CD24 IN B CELLS

KAITLYN E. MAYNE, & SHERRI L. CHRISTIAN

B lymphocytes (B cells) undergo strict regulation during development to protect against foreign pathogens and prevent autoimmunity. CD24, a surface protein on immature B cells that functions to regulate developmental apoptosis, lacks a cytoplasmic signalling domain and its mechanism of signalling is unknown.
We hypothesized that CD22 (Siglec-2), a B–cell–restricted protein, acts as a functional co-receptor for CD24. Both proteins are enriched in the bone marrow during early B cell development where they regulate apoptosis. Additionally, CD24 has been shown to interact with Siglec proteins in other cell types, supporting the potential for CD24-CD22 interaction. Using flow cytometry, we found CD24 activation significantly increases CD22 surface expression, independent of new protein synthesis, indicating that CD24 unmasks existing CD22 surface receptors. siRNA knockdown confirmed this increase reflects altered CD22 epitope availability rather than a change in protein abundance. Confocal microscopy revealed CD24-CD22 co-localization following CD24 stimulation, while FRET analysis confirmed physical proximity within 10 nm that increases upon CD24 activation. We are now testing phosphorylation of proteins downstream of CD22 to determine if CD24 engagement activates CD22 signalling. Together, these findings provide strong evidence for a direct CD22-CD24 interaction that increases upon CD24 stimulation, supporting our hypothesis that CD22 is a functional signaling partner for CD24 in B cells. Future work will determine whether this interaction regulates CD24-mediated apoptosis and extracellular vesicle secretion during B cell development.

POSTER PRESENTATION ABSTRACTS

MICROARRAY ANALYSIS OF GENE EXPRESSION AND METABOLIC PATHWAYS UNDERLYING HYPERTENSION AND ADIPOSITY IN INTRAUTERINE GROWTH-RESTRICTED YUCATAN PIGS

ISAAC OMEH, TOUATI BENOUKRAF, & ROBERT F. BERTOLO

The study examined the genetic link between intrauterine growth restriction (IUGR), blood pressure, and obesity in Yucatan miniature pigs. Previous research showed IUGR pigs grew faster before maturity and had higher adult blood pressure than normal-weight littermates. Using microarray analysis, this study explored gene expression and metabolic pathways related to early blood pressure and fat development in IUGR pigs. Methods involved analyzing hepatic genes in male Yucatan pigs, categorizing them by function, and mapping them to KEGG pathways. Three IUGR/NW pairs with different blood pressure phenotypes were selected for analysis, with statistical adjustments for p-values. Results identified 49 upregulated genes across categories such as metabolism, growth, blood pressure, hormones, adiposity, size, sodium sensitivity, and appetite. KEGG mapping linked 34 genes to 79 pathways, primarily metabolic pathways, including steroid biosynthesis, fatty acid metabolism, and retinol metabolism. Twenty-seven downregulated genes were also categorized, mainly involved in lipid metabolism, energy, transport, protein processing, signalling, and disease-related functions. KEGG mapping showed that 15 of these genes were present in 17 pathways, mostly metabolic. Conclusions suggest these genes' roles in metabolism and potential as intervention targets or biomarkers. The pathway network highlights the complexity of early programming of adult diseases. (Funded by CIHR)

POSTER PRESENTATION ABSTRACTS

DIETARY VITAMIN D MAY REDUCE FAT ACCUMULATION IN FEMALE RATS ON A HIGH-FAT DIET

ANNA-MARIE COONEY, HANNAH WHITE, & ZAHRA FARAHNAK

Obesity is a health concern characterized by excess adiposity. Evidence suggests that vitamin D and docosahexaenoic acid (DHA) may reduce fat accumulation. This study assesses vitamin D, DHA, and their co-supplementation on fat accumulation. Eight-week-old male and female Sprague-Dawley rats (n=95) were randomly assigned to control or high fat (HF) diet (35% kcal), HF-low D (0.05 IU/g), HF-D (3 IU/g), HF-DHA (1% dietary weight), and HF-DHA (1%) + D (3 IU/g) for 8 weeks. Upon sacrifice, blood and adipose tissues were harvested. Serum vitamin D was measured using LC-MS/MS. Food intake, body weight and length were significantly higher in males ($P < 0.001$). HF-low D diet resulted in lower vitamin D concentrations, while HF-D produced higher concentrations (M:39.8 ± 2.50, F: 44.0 ± 1.03, $P < 0.001$). Female rats had higher intra-abdominal fat weight (mesenteric, retroperitoneal, urogenital) ($P < 0.001$) compared to control diet (23.6 ± 3.71) and HF-D (21.03 ± 1.58). Urogenital fat weight was similar between HF-D and control (9.6 ± 0.65) groups. This trend was not observed in male epididymal fat. Preliminary findings suggest that high vitamin D supplementation may mitigate high-fat diet induced fat accumulation, particularly in females, highlighting sex differences in response to vitamin D.

POSTER PRESENTATION ABSTRACTS

IDENTIFYING THE PURINERGIC RECEPTOR TARGETED BY TAAR1 DURING ATP-INDUCED INFLAMMATORY SIGNALLING

NIMEESHA VANKADARA, MARK D. BERRY, & DAVID BARNES

Trace amine-associated receptor 1 (TAAR1) has emerged as a potential regulator of immune function, with growing evidence suggesting a role in modulating inflammatory responses. Previous work in Dr. Berry's laboratory demonstrated that TAAR1 activation selectively inhibits ATP-induced tumour necrosis factor (TNF) secretion in macrophages without affecting lipopolysaccharide (LPS)-induced responses, indicating that TAAR1 targets specific inflammatory pathways rather than acting as a general suppressor of inflammation. Furthermore, prior studies showed no effect of TAAR1 activation on non-purinergic damage-associated molecular pattern (DAMP) signalling, including uric acid and succinate, suggesting selective modulation of purinergic pathways. Despite these findings, the specific purinergic receptor regulated by TAAR1 remains unknown. This project aims to identify the purinergic receptor subtype involved in TAAR1-mediated modulation of ATP-induced inflammatory signalling. Extracellular ATP signals through both P2X ionotropic receptors and P2Y G protein-coupled receptors, each capable of driving pro-inflammatory cytokine secretion, with previous concentration-response curves suggesting P2X7 and P2Y12 as likely candidates.. Selective pharmacological antagonists targeting each will be used in and TNF secretion quantified using enzyme-linked immunosorbent assay (ELISA). By identifying the receptor subtype regulated by TAAR1 this work will provide new insights into the receptor-specific mechanisms controlling DAMP-induced inflammation. These findings will improve our understanding of TAAR1-mediated immune regulation and may support the development of more targeted therapeutic strategies for inflammatory diseases.

POSTER PRESENTATION ABSTRACTS

COMPARISON OF SCFA PROFILES IN CECAL AND FECAL SAMPLES FROM SEMAGLUTIDE- AND ULOTORANT- TREATED MICE

YAR MALUAL, MARK D. BERRY

Obesity and type 2 diabetes mellitus (T2DM) are associated with impaired glucose regulation. While semaglutide, a GLP-1 receptor agonist, is the standard for T2DM treatment, TAAR1 agonist Ulotaront is a promising alternative with a different mechanism of action. Semaglutide treatment has been associated with changes in the gut metabolome; however, the effects of TAAR1 agonists on the gut metabolome are mostly unknown. Targeted gas chromatography-mass spectrometry (GC-MS) is used in this project to identify and quantify short-chain fatty acids (SCFAs) in cecal and fecal samples collected from control and high-fat/high-sugar (HFHS) mice treated with semaglutide or Ulotaront. SCFA profiles will be compared across the different groups as indicators of microbial metabolic activity. If Semaglutide and Ulotaront produce similar SCFA profiles, this may suggest shared metabolic adaptations despite different receptor targets. Alternatively, different SCFA profiles would suggest that TAAR1 agonists influence host-microbial activity differently compared to GLP-1 receptor agonists.

By comparing microbial metabolites following these two treatments, this project aims to determine whether TAAR1 agonism is associated with a unique metabolome that may provide insight into its broader metabolic effects. This work could contribute to future studies on gut-brain-metabolic interactions associated with TAAR1 agonism. The use of metabolite profiling with microbiome sequencing could help determine whether alterations in microbial metabolism contribute to the therapeutic effects of TAAR1-targeted drugs.

POSTER PRESENTATION ABSTRACTS

REGULATORY ROLE OF TBX3 IN NUCLEAR MIRNA-GUIDED GENE REGULATION IN THE PATHOGENESIS OF TRIPLE-NEGATIVE BREAST CANCER

SUSHMITHA RAMAKRISHNA & PAVAN KUMAR KAKUMANI

MicroRNAs (miRNAs) are small non-coding RNAs that regulate gene expression through Argonaute-2 (AGO2)–mediated RNA interference (RNAi) in the cytoplasm. While miRNA–AGO2 interactions are well characterized in the cytoplasm, emerging evidence indicates that AGO2 also localizes to the nucleus of embryonic stem cells. However, the molecular mechanisms regulating nuclear AGO2 activity and its associated cofactors in triple-negative breast cancer (TNBC) remain poorly understood. T-box transcription factor (TBX3), a transcriptional factor frequently overexpressed in TNBC, promotes proliferation, stemness, and tumor progression. Preliminary co-immunoprecipitation data suggest that AGO2 directly interacts with TBX3; however, the functional role of this interaction in nuclear miRNA-mediated gene regulation remains unclear. This study aims to investigate the regulatory the role of TBX3 in regulating nuclear miRNA-guided gene silencing in TNBC. We hypothesize that TBX3 interacts with components of the nuclear RNAi machinery to facilitate miRNA-mediated gene silencing. Using TNBC cell models, we will employ subcellular fractionation to separate the nuclear and cytoplasmic compartments and assess the localization of AGO2. Transfection will be used to manipulate TBX3 expression levels. Immunoprecipitation followed by western blotting will allow us to examine potential interactions between TBX3 and AGO2 or other RNAi-associated proteins. Finally, RT-qPCR will be used to determine whether TBX3 influences AGO2 nuclear localization and promotes the assembly of nuclear RNAi machinery. Overall, this study will provide mechanistic insight into TBX3-dependent regulation of nuclear miRNA-mediated gene silencing and may reveal a previously unrecognized regulatory pathway contributing to TNBC progression

POSTER PRESENTATION ABSTRACTS

INVESTIGATING THE HEMOLYTIC ACTIVITY OF ANTIMICROBIAL PEPTIDES USING MOLECULAR DYNAMICS SIMULATIONS

ALEXANDER POWER & KATIE WILSON

One of the largest growing health threats in the world of modern medicine is antibiotic-resistant bacteria. Through various genetic mutations, these harmful bacteria can resist traditional antibiotics, and currently, very few effective treatments for these pathogens exist. One potential alternative to conventional antibiotics are antimicrobial peptides, or AMPs. AMPs are small peptides that can target and disrupt bacterial membranes, making them a promising avenue to fight antibiotic-resistant bacteria. A major issue, however, is that some AMPs have been shown to disrupt mammalian membranes, showing hemolytic activity against red blood cell (RBC) membranes. This renders AMPs much less useful in fighting antibiotic-resistant bacteria. In this study, we employed extensive molecular dynamics simulations to investigate the molecular mechanisms by
which AMPs modulate the biophysical properties of the RBC membrane. Notably, RBC membrane models that contain the full biochemical complexity are a novel area of research, meaning our computational analysis also provides a new view into the behaviour of the RBC membrane. Collectively, our findings provide valuable molecular insights that can guide the rational design of AMPs with enhanced selectivity toward pathogenic bacterial membranes while minimizing hemolytic activity.

POSTER PRESENTATION ABSTRACTS

INVESTIGATING THE INTERACTIONS OF CHINESE RED SAGE NATURAL PRODUCTS WITH P38 MAPK IN TRIPLE-NEGATIVE BREAST CANCER

MACKLIN PELLEY, HUCK GROVER, VALERIE BOOTH, & SHERRI CHRISTIAN

Breast cancer affects 1 in 8 women and results in up to 13% of cancer-related deaths in Canada, making the development of improved therapies a research priority. Triple-negative breast cancer (TNBC) is an aggressive breast cancer subtype with limited treatment options, making the need for improved therapeutic strategies. Natural products have shown promise as potential anticancer agents, including compounds derived from the Chinese red sage plant, Salvia miltiorrhiza. Previous research investigating synthetically prepared red sage natural products showed various cellular responses. Tanshinone IIA demonstrated a dose-dependent reduction in cell viability, while tanshinone IIA anhydride and normiltirone produced a hormetic response, characterized by increased viability at lower concentrations and inhibition at higher concentrations. Protein target prediction and docking analyses suggested p38α MAPK, a stress-response protein that regulates cell survival, apoptosis, and inflammation, as a potential target. Computational approaches were used to investigate the molecular interactions between the natural products and p38α. Molecular docking predicted the anhydride-containing compounds preferentially bind within a lipid-binding pocket, while tanshinone IIA docked to the hinge region. Molecular dynamics (MD) simulations were performed to evaluate the stability and behaviour of these protein-ligand complexes. MD simulations demonstrated that the anhydride-containing ligands remained within the lipid-binding pocket. Ligand binding displaced water molecules from the pocket, supporting a hydrophobic binding mechanism for the anhydride-containing compounds and their molecular interactions. Computational predictions are currently being tested through Western blot analysis to measure p38 phosphorylation after natural product exposure.

POSTER PRESENTATION ABSTRACTS

COMPUTATIONAL INVESTIGATION OF THE STRUCTURE-ACTIVITY RELATIONSHIP OF THE NON-HEMOLYTIC ANTIMICROBIAL PEPTIDE THURINCIN H

ANDREW HICKEY, DANANJANA WIJERATHNE, & KATIE WILSON

Antimicrobial resistance is a global health threat, attributing to over one million deaths in 2019 alone. Bacteria are becoming increasingly resistant to traditional antibiotics, creating more severe infections, prolonged hospitalizations, and increased healthcare costs. Antimicrobial peptides (AMPs) are small, typically cationic peptides that are promising alternatives to current antibiotics, as they are less susceptible to antimicrobial resistance. However, AMPs have a tendency to exhibit off-target activity against mammalian red blood cells. Thurincin H is an AMP that does not exhibit hemolytic activity, making it a promising next-generation therapeutic candidate. In this study, we investigate the structure-activity relationship of Thurincin H, complemented by experimental collaborations, to provide molecular insights that guide the optimization of its purification process. More specifically, we analyzed three experimentally reported Thurincin H mutants and compared their structural and dynamic properties with those of the wild-type peptide. Collectively, our findings provide molecular insights that may guide the optimization of the Thurincin H purification process while preserving its antimicrobial activity, thereby supporting the development of next-generation antimicrobial peptide therapeutics.

POSTER PRESENTATION ABSTRACTS

ROLE OF MICRORNA-132 IN REGULATING CHOLESTEROL EFFLUX AND FOAM CELL FORMATION IN MACROPHAGES

IHECHI MARCUS & ROBERT J. BROWN

Atherosclerosis is a chronic inflammatory disease characterized by the accumulation of cholesterol-laden macrophages within the arterial wall, leading to foam cell formation and plaque development. Efficient removal of excess cholesterol from macrophages is mediated by ATP-binding cassette transporter A1 (ABCA1), which initiates reverse cholesterol transport and protects against lipid accumulation. Previous work from our laboratory demonstrated that activation of the PI3K/Akt signalling pathway impairs macrophage cholesterol efflux without reducing ABCA1 protein expression, suggesting that cholesterol transport can be regulated through signalling-dependent mechanisms. Recent RNA sequencing studies also identified microRNA-132 (miR-132) as differentially expressed in macrophages exposed to lipid hydrolysis products, implicating miR-132 as a potential regulator of macrophage cholesterol homeostasis. This study investigates whether miR-132 contributes to impaired cholesterol efflux by suppressing phosphatase and tensin homolog (PTEN), thereby enhancing Akt signalling and functionally impairing ABCA1-mediated cholesterol transport. We hypothesize that increased miR-132 expression promotes intracellular lipid accumulation and foam cell formation, whereas inhibition of miR-132 restores cholesterol efflux. To test this hypothesis, THP-1 macrophages will be transfected with miR-132 mimic or antimiR-132, followed by assessment of ABCA1, PTEN, and Akt expression using quantitative PCR and Western blotting. Functional consequences will be evaluated using ApoA-I-mediated cholesterol efflux assays and Oil Red O staining to assess intracellular lipid accumulation. Understanding the role of miR-132 in regulating macrophage cholesterol metabolism may provide new mechanistic insights into atherosclerosis and identify potential therapeutic targets for reducing foam cell formation and cardiovascular disease progression.

POSTER PRESENTATION ABSTRACTS

DOCOSAHEXAENOIC ACID SUPPLEMENTATION MAY ATTENUATE HIGH-FAT DIET-INDUCED REDUCTIONS IN MUSCLE CROSS-SECTIONAL AREA IN RATS

ALYSSA DOBER, HANNAH WHITE, ELIZABETH CHIA, & ZAHRA FARAHNAK

Obesity impairs muscle health, which may impact muscle function and metabolism. Recent evidence suggests that docosahexaenoic acid (DHA) can improve muscle metabolism. This study examines the effect of DHA on muscle metabolism in rats fed a high fat (HF) diet. Male and female Sprague-Dawley rats (n=46) aged eight weeks old were randomly assigned to control (C)(AIN-93M), HF (35% kcal fat) or HF+DHA (1% dietary weight) diets for 8 weeks. Blood samples and muscle mass (bilateral quadriceps) were collected. Fatty acid profile was assessed using gas chromatography (GC) to determine DHA%, EPA%, and omega-3 index in the quadriceps. ImageJ was used to calculate average quadriceps myofiber cross-sectional area (CSA) from images taken with a Zeiss light microscope. A General Linear Model and Spearman's correlation ($P<0.05$) were used to test the data. Data are presented as mean±SEM. Quadriceps CSA was higher in the HF+DHA group (4001.62um²±345.20) than HF (3351.80um²±130.47, $P=0.054$), with higher CSA in males ($P<0.001$). Rat length and quadriceps weight were higher in males in all three diets ($P<0.001$). The omega-3 index (DHA+EPA) was higher in the HF+DHA group (17.024%±0.48) than in the control (8.88%±0.52, $P<0.001$) and HF (8.91%±0.48)($P<0.001$). Females had a higher omega-3 index than males in all three diets ($P<0.05$). Quadriceps CSA was positively correlated with quadriceps weight ($P<0.001$, $r=0.54$) and rat length ($P=0.001$, $r=0.51$). Our preliminary findings suggest that DHA supplementation increased omega-3 deposition in the quadriceps muscle and can increase quadriceps myofibre CSA.

POSTER PRESENTATION ABSTRACTS

INVESTIGATING THE IMPACT OF GLUCOSE, FRUCTOSE, AND GLUCOLIPOTOXIC CONDITIONS ON LIPID ACCUMULATION IN ADIPOCYTES

AGAPE POMROY & SCOTT HARDING

3T3-L1 cells are mouse embryonic fibroblasts that can differentiate into adipocytes through the activation of a transcriptional cascade in which the master regulators C/EBPα and PPARγ are rapidly expressed. Together, these proteins activate adipocyte genes that produce the differentiated phenotype. Adipocytes are the primary cell type in adipose tissue and store excess calories as triglycerides within lipid droplets. Adipose tissue consists of two distinct types: white adipose tissue (WAT) and brown adipose tissue (BAT). WAT is critical for energy storage, endocrine communication, and insulin sensitivity, whereas BAT is essential for non-shivering heat production, which helps regulate internal body temperature without muscle movement. These tissues also differ in cellular morphology. White adipocytes are typically larger and unilocular, containing one large lipid droplet that pushes the organelles to the cell periphery. Brown adipocytes contain multiple lipid droplets and are enriched with mitochondria. Recent research has also examined beige adipocytes, which share characteristics of both WAT and BAT. The transformation of WAT into brown or beige adipose tissue, commonly referred to as browning or beiging, may be influenced by dietary factors. In this study, Oil Red O was used to stain neutral lipids, allowing lipid droplet formation and lipid accumulation to be analyzed. The aim of this study was to determine how adipocytes respond to varying dietary treatments. By identifying differences in lipid accumulation and adipocyte morphology, this study may provide insight into how specific dietary treatments alter adipocyte characteristics and lipid storage, contributing to metabolic dysregulation.

POSTER PRESENTATION ABSTRACTS

INVESTIGATING THE AUTOLOGOUS EXTRACELLULAR VESICLE TRANSFER IN B-LYMPHOBLASTIC LEUKEMIA CELLS STAINED WITH A LIPOPHILIC DYE

CHATURRVI MARUTHIREDDY, KAITLYN E. MAYNE, RASHID JAFARDOUSTBOSTANI, & SHERRI L. CHRISTIAN

Extracellular vesicles (EVs) are nanoparticles mediating intercellular communication by transferring molecules between cells. In healthy B lymphocytes, EVs regulate immune responses by suppressing excessive activation or amplifying the response. However, in malignant B lymphocytes, EVs promote therapy resistance and tumour growth. B-cell Acute Lymphoblastic Leukemia (B-ALL) is a blood cancer characterized by abnormal B lymphocyte production, and the role of EVs in B-ALL progression remains unclear. In order to track EV uptake, the UoC-B1 B-ALL cell line will be stained with the dye Neuro-DiO, which diffuses into the cell membrane and is thus incorporated into secreted EVs, enabling fluorescent visualization of these EVs. Our main objective is to determine if UoC-B1-derived EVs are taken up by UoC-B1 recipient cells. Preliminary testing focused on identifying an optimal Neuro-DiO dye concentration for cell staining by evaluating multiple concentrations (0.75-10 µM) and staining with a Live/Dead stain to assess cell death from the Neuro-DiO. Results showed cell death of 13.8%, 10.9%, and 15.7% at 2.5, 5, and 10 µM, respectively, but less than 10% at 0.75 and 1.25 µM. The 0.75 µM Neuro-DiO concentration showed the lowest cell death (4.77%) while maintaining a high fluorescence intensity of stained cells. Thus, 0.75 µM of Neuro-DiO will be used in subsequent experiments. After cell staining, secreted EVs will be isolated by size-exclusion chromatography, characterized by nanoparticle tracking analysis, and co-cultured with recipient UoC-B1 cells for flow cytometry analysis to determine whether autologous EV uptake occurs.

POSTER PRESENTATION ABSTRACTS

HIGH-PRESSURE PROCESSING AS A GREEN PRE-TREATMENT STRATEGY FOR ENHANCED SAPONIN RECOVERY FROM NORTH ATLANTIC SEA CUCUMBER

KERTHIKA DEVI A, DEEPIKA DAVE, & FEREIDOON SHAHIDI

The Northern Atlantic sea cucumber (Cucumaria frondosa) is an abundant marine echinoderm found in Atlantic Canada. It is considered a delicacy in several East Asian countries for its rich source of bioactive compounds with diverse pharmacological activities, including antioxidant, anti-inflammatory, anticancer, and antidiabetic properties. Among these bioactive compounds, saponins are considered a key constituent because of their broad therapeutic potential, especially known for their anti-cancer activity. However, their extraction, characterization, and utilization remain limited due to their structural complexity, low extraction yields, and toxicity concerns. High-pressure processing (HPP), a non-thermal and green technology widely used for food preservation, has recently emerged as a promising pretreatment for enhancing the extraction of bioactive compounds while minimizing thermal degradation. However, its use as a pretreatment to improve saponin extraction from C. frondosa has received limited attention. In this study, the body wall, internal organs, and flower of C. frondosa were pretreated at 150, 350, and 550 MPa to investigate the effect of different pressures of HPP on saponin extraction. Among the treatments, 350 MPa produced the highest yield, resulting in a 1.6-fold increase in total saponin content in the internal organs compared with the untreated control. These findings demonstrate the potential of HPP pretreatment to enhance saponin recovery, particularly from processing discards such as internal organs, supporting sustainable biomass valorization and the development of high-value functional ingredients from marine resources.

POSTER PRESENTATION ABSTRACTS

FISH OIL VERSUS FLAXSEED OIL: A COMPARATIVE STUDY ON PREGNANCY AND FETAL NEURODEVELOPMENT

AMRITHA AMBY SINDHU, DIEN HONG PHAN, BRANDON DOUGLAS DALTON, & SUKHINDER KAUR CHEEMA

Omega-3 (n-3) polyunsaturated fatty acids (PUFAs) are important during pregnancy and are critical for fetal brain growth and development. While most studies have focused on marine-derived n-3 PUFAs, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), comparatively little is known about the effects of the plant-derived n-3 PUFA, alpha-linolenic acid (ALA), on maternal health and pregnancy outcomes. This is the first study that directly compares the effects of flaxseed oil- and fish oil-enriched diets on pregnancy outcomes, maternal metabolic regulation, and fetal brain development. Female C57BL/6 mice (8 weeks old) were fed four different diets: 10% safflower oil (SO) – source of n-6 PUFA, 3% fish oil + 7% safflower oil (FO), 3% flaxseed oil + 7% safflower oil (FL3), or 10% flaxseed oil (FL10), respectively, for 2 weeks before mating and throughout pregnancy. Females were sacrificed on gestation day (GD) 18.5 (end of third trimester), and maternal blood, fat, placenta, and fetal brains were collected; the number of fetuses and resorption sites were recorded. Our preliminary findings showed fetal resorption sites in the SO, FO, and FL3 groups, with the highest incidence in the SO group, whereas no resorption sites were found in the FL10 group. Except FO, a number of dams from all other groups delivered between GD 17.5 and 18.5. Placental and fetal brain fatty acid profiles will be measured to investigate whether the same amount of flaxseed oil or fish oil incorporates the same amount of DHA in the fetal brain. Future analysis will investigate whether flaxseed oil and fish oil have similar effects on maternal metabolic regulation. The findings will help in understanding requirements for a plant-based n-3 PUFA maternal diet for optimal health of both mothers and offspring.

POSTER PRESENTATION ABSTRACTS

THE LIGAND-DEPENDENCY OF HTAAR1 CONFORMATIONAL ENSEMBLES: A SCAFFOLD FOR ALLOSTERIC SITE CHARACTERIZATION

KYLE WARREN, MARK D. BERRY, & KATIE WILSON

Human trace amine-associated receptor 1 (hTAAR1) is an aminergic G protein-coupled receptor (GPCR) that regulates neurotransmitter signalling within the central nervous system, alongside nutrient-dependent hormone secretion in the periphery. Consequently, hTAAR1 has emerged as a promising therapeutic target for disorders including schizophrenia, depression, amphetamine addiction, and diabetes. Despite its therapeutic potential, no selective hTAAR1 antagonists (receptor inhibitors) have been identified, limiting both therapeutic development and detailed mechanistic investigations of hTAAR1 signalling. Moreover, the high structural similarity of the orthosteric binding pockets across aminergic GPCRs complicates the design of hTAAR1-selective ligands by significantly increasing the risk of undesirable off-target effects. Consequently, allosteric binding pockets represent an attractive alternative for structure-guided drug design, as these less conserved and structurally distinct binding interfaces can enhance drug-receptor selectivity while preserving potent modulation of receptor activity. Nonetheless, it remains unknown whether hTAAR1 harbours cryptic allosteric binding sites and, if so, whether their formation is coupled to ligand binding at the orthosteric site similarly remains an open question. To address this knowledge gap, we performed long-timescale molecular dynamics simulations of hTAAR1 embedded within its native-like lipid membrane environment, spanning inactive, partially active, and fully active receptor states. Collectively, these simulations will provide novel insights into the conformational dynamics governing hTAAR1 activation while establishing a structural framework for subsequent mixed-solvent molecular dynamics simulations to identify and characterize activity-modulating allosteric binding sites.

POSTER PRESENTATION ABSTRACTS

AN INVESTIGATION OF THE INFLUENCE OF ADIPOCYTES AND ADIPOCYTE-DERIVED LYSYL OXIDASE ON THE PLASTICITY OF MDA-MB-231 BREAST CANCER METASTATIC VARIANTS

LEAH MARCANTONIO, GRANT KELLY, SHERRI CHRISTIAN, & ALICIA VILORIA-PETIT

Breast cancer is the most diagnosed cancer among Canadian women, making up 26% of new cases. Triple-negative breast cancer (TNBC) is characterized by a lack of increased hormone and HER2 receptors, and its aggressive nature. Most cancer-related deaths result from metastatic disease, when cells separate from the tumour and colonize another site. This can occur through cells taking on a mesenchymal morphology to invade tissue and travel through the blood, and reversion back to a round, epithelial morphology to form new tumours. Adipose-secreted lysyl oxidase (LOX) has been shown to play a role in TNBC cells undergoing a partial mesenchymal-to-epithelial transition (MET) when cultured over adipocytes with Matrigel. In this project, MDA-MB-231 cells isolated from bone and brain metastases were cultured in 3D to investigate differences in morphology and protein expression. We hypothesize the adipose-rich bone marrow will be abundant in pro-LOX that, when activated, facilitates the partial MET required for tumour formation, so cells that preferentially colonize the bone should secrete higher levels of LOX-activating enzymes, like ADAMTS2. Western blots showed that adipocytes and their interactions with MDA-MB-231 variants promote active LOX and ADAMTS2 secretion, however, no pattern was observed between variants. Immunofluorescence shows the bone variants form colonies and appears to lose their elongated morphology when cultured in 3D, similar to but more noticeably than the parental line, while the brain variant tends to group together irrespective of adipocytes. Establishing a link between LOX and metastasis can give researchers a new target for prognoses and treatment of TNBC.

POSTER PRESENTATION ABSTRACTS

EVALUATION OF OXIDANT LOAD IN NEONATAL PARENTERAL NUTRITION WITH AND WITHOUT ANTIOXIDANT SUPPLEMENTATION

RUMANA IRFAN, THILINI KUMARASINGHE, JANET A. BRUNTON,& ROBERT F. BERTOLO

For premature and newborns who cannot tolerate sufficient enteral feeding, parenteral nutrition (PN) is a vital source of nutrients. However, during preparation, storage, and administration, nutrients in PN solutions are vulnerable to oxidative degradation, producing lipid hydroperoxides and reactive oxygen species. Neonates have immature antioxidant defense systems that render them susceptible to oxidative stress; as such, receiving an increased oxidant load contributed by these oxidative products may be especially problematic, contributing to organ and tissue damage. Earlier in vitro work from our research group demonstrated that antioxidant supplementation enhanced the oxidative stability of PN formulations, and led to the theoretical development of an “optimized” PN solution. This formulation was recently tested in vivo, in a study with neonatal piglets. The objective of this study was to assess the oxidant load delivered to the neonates when the diet was exposed to light and ambient temperatures, as occurs in the experimental and clinical environments. Specifically, this study's goal was to assess peroxide generation in newborn PN formulations after addition of the antioxidants selenium, glutathione (delivered as GSSG), vitamin E(α-tocopherol), and vitamin C (ascorbic acid). PN solutions were prepared in the lab with or without additional antioxidant nutrients and mixed with one of two commercial lipid products, SMOFlipid (Frenius-Kabi) or Intralipid (Baxter). The standard and antioxidant-supplemented optimized PN formulations exposed to ambient temperatures and light, and were sampled at 0, 6, 12, and 24 hours to measure the development of pro-oxidants over time. The ferric-xylenol orange (FOX) assay, which measures peroxide and lipid hydroperoxide concentrations as markers of primary oxidation, was used to quantify oxidative product production. To evaluate the impact of antioxidant supplementation on oxidative stability, peroxide concentrations were calculated from a hydrogen peroxide standard curve and compared among PN formulations. This work is important because enhancing the oxidative stability of PN solutions will aid in the creation of safer PN formulations with reduced oxidant exposure in susceptible neonatal patients.

POSTER PRESENTATION ABSTRACTS

OPTIMIZING ARGININE INTAKE IN PARENTERAL NUTRITION FOR NEONATES

KAITLYN GATHERALL, ASHA U. DE SILVA, ROBERT F. BERTOLO, & JANET A. BRUNTON.

The optimal arginine concentration in parenteral nutrition (PN) designed for neonates remains unclear. Arginine is a conditionally essential amino acid, and in a healthy infant, de novo synthesis of arginine occurs in the small intestinal mucosa during first-pass metabolism of dietary precursors, a process that is by-passed during PN feeding. Inadequate intake of arginine can impair urea cycle function, metabolic function, and growth.

Using a PN-fed neonatal piglet model, this study aims to use breakpoint analysis to determine the arginine intake required to optimize whole body and tissue specific protein synthesis, as well as creatine and nitric oxide synthesis. Yucatan miniature piglets (9-to-16-d old, n=15) were fitted with jugular and femoral venous catheters for diet infusion and blood sampling. On day 5, piglets were assigned to one of 15 isonitrogenous test diets, differing only in arginine, serine, and alanine content. On day 6, a 6-hour IV-infusion of stable isotope tracers was administered to assess whole body protein, creatine, and nitric oxide synthesis. Thirty minutes prior to necropsy, a flooding dose of phenylalanine stable isotope was to measure rates of tissue specific protein synthesis.

Preliminary analysis showed that plasma arginine concentrations increased in a dose-dependent manner with dietary arginine intake ($P<0.001$). In contrast, preliminary analysis suggested skeletal muscle fractional protein synthesis rate, which ranged from 7.1 - 17.1% per day, did not respond to increasing dietary arginine intake. Findings from this study will help refine arginine requirements for PN-fed newborns, optimizing neonatal nutrition and health.

POSTER PRESENTATION ABSTRACTS

DETERMINING THE MECHANISM OF STABILITY FOR B-CELL RECEPTORS TRANSFERRED BY EXTRACELLULAR VESICLES

SAMANTHA NORMAN, RASHID JAFARDOUSTBOSTANI, & SHERRI L. CHRISTIAN

Extracellular vesicles (EVs) are lipid bilayer bound particles which act as cellular messengers, transferring protein, receptors, and other cargo from one cell to another. The transfer of EVs from a donor cells to receptor cells can be stimulated by anti-CD24 antibodies. CD24 (Cluster of Differentiation 24) is a highly glycosylated protein anchored to the outer cell surface. The B-cell Receptor (BCR) is an important receptor mediating apoptosis or cell proliferation. The BCR can be transferred via EVs and is functional once incorporated into the recipient cell surface. BCR recycling occurs by internalisation of the receptor, and then recycling back to the cell surface. It is currently unknown how an EV-transferred BCR is recycled. The objective of this project is to determine the stability mechanism of B-cell receptors transferred by EV's.

To identify which pathway regulates the endocytosis, and thus stability, of the transferred BCR, dynamin, actin, and lipid rafts were inhibited with chemical inhibitors and the levels of EV-transferred BCR were determined using flow cytometry. BCR levels on samples inhibited with Dynasore, a dynamin inhibitor, were similar to the control (Dimethyl sulfoxide). Actin inhibitor Cyto D showed partial inhibition, and lipid raft inhibitor Methyl-beta-cyclodextrin resulted in full inhibition. Thus, stability of EV-transferred BCRs are regulated by dynamin independent, actin and lipid raft dependant mechanisms.

AWARDS TO BE PRESENTED

GRADUATE AWARDS

BOUNCE HEALTH INNOVATION AWARD (PH.D)
Best Ph.D oral presentation

RUNNER UP ORAL PRESENTATION
2nd place Ph.D oral presentation

BOUNCE HEALTH INNOVATION AWARD (M.SC)
Best M.Sc oral presentation

RUNNER UP ORAL PRESENTATION
2nd place M.Sc oral presentation

1ST PLACE POSTER PRESENTATION
Best graduate student poster presentation

RUNNER UP POSTER PRESENTATION
2nd place graduate student poster presentation

3RD PLACE POSTER PRESENTATION
3rd place graduate student poster presentation

UNDERGRADUATE AWARDS

1ST PLACE POSTER POSTER PRESENTATION
Best undergraduate poster presentation

RUNNER UP POSTER PRESENTATION
2nd place undergraduate poster presentation

3RD PLACE POSTER PRESENTATION
3rd place undergraduate poster presentation

AWARDS TO BE PRESENTED

PEOPLE'S CHOICE AWARDS

PEOPLE'S CHOICE ORAL PRESENTATION

Best scientific communication and riveting presentation as chosen by audience

PEOPLE'S CHOICE POSTER PRESENTATION

Best scientific communication, poster design, and presentation as chosen by audience