
THE 12TH ANNUAL BIOCHEMISTRY
SYMPOSIUM
2022
Friday, 12th August
9 AM to 8 PM (Registration begins 8:30 AM)
The Fluvarium, St. John's, NL
MEMORIAL UNIVERSITY
BCGS
DESIGNED BY
SAMAN SALARI
VISHAL PANDYA

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Dr. Shaymanchand Mayengbam

ORGANIZING COMMITTEE

WELCOME ADDRESS

DR. MARK BERRY

HEAD OF DEPARTMENT,
DEPARTMENT OF BIOCHEMISTRY

Welcome to the 12th annual Biochemistry Symposium, a return to an in-person format, and an opportunity to see the first research outputs from our brand new home of the Core Sciences Facility. I trust everyone is looking forward to being able to engage face-to-face again as much as I am. That said, I would be remiss not to provide a note of caution. COVID-19 is by no stretch of the imagination over, and while there may no longer be a mask mandate, as leaders in Life Sciences research, we should be setting an example for following the science and evidence. Masking is still an important health protective measure, particularly in indoor settings where people will be in close quarters, such as a symposium. So please be considerate of others, and stay masked up when not giving a presentation. It may be slightly uncomfortable, but far less so than a week confined to solitary, or even worse a trip to ICU!

This year we are thrilled to be able to welcome Dr. Avi Chakrabartty from the University of Toronto as our keynote speaker and are very much looking forward to hearing about his work in protein folding. And as we all know from BIOC2201 – structure determines function, so there should be something for everyone in Dr. Chakrabartty's work. At last check our enrollments are once again incredibly healthy, soaring above 70. The symposium has certainly come a long way from its neonatal days of formation back in 2011, and I doubt anyone anticipated back then the symposium being the rousing success it has been. That success is largely due to the extraordinary dedication and hard work of the organizing students and faculty over-viewers each year, which leads nicely to a recognition of this year's organizing committee led by Sina Heravi, Vishal Pandya, Dr. Mayengbam and Dr. Park.

In closing, I wish everyone the best of luck with their presentations, and look forward to a day of vibrant, engaging, discussions, followed by the equally important awards presentations and social.

WELCOME ADDRESS

VISHAL PANDYA

2022 COMMITTEE CHAIR

The BCGS would like to welcome everyone's participation in the 12th Biochemistry Summer Symposium. Amidst these uncertain times, the committee has continued to work very hard toward maintaining and fulfilling pertinent expectations. However, this would not have been possible without everyone's support, including our sponsors, for which we are very grateful and hope to be able to continue receiving. As we look forward to reinstating our grounds in a new environment, we also wish everyone all the best in this exciting journey. It is our pleasure as a committee to see you all finally for an in-person event after a while. BCGS is so looking forward to this event.

MESSAGE OF SUPPORT

DR. TRAVIS FRIDGEN

ACTING DEAN OF SCIENCE

Congratulations to the BCGS for holding your 12th annual summer symposium. Without a doubt, communication is the cornerstone of research. Oral presentations are not just a means of showing what you have done but are especially important to provide a more personal and creative perspective on the new and exciting results you are communicating to your audience. It is an opportunity to get instant feedback to progress your work, and to pass on knowledge.

The art of oral presentation takes practice and patience. As an audience member, you might try to find where your own work fits in with the work of others, you may be able to help the presenter progress their work, or you may just learn something new—which is something we should actually try to do every day.

Congratulations to all of you for participating in the symposium and to those of you who helped to organize it. Good luck to everyone, and please have a safe and productive conference. Be respectful and be kind in your interactions with one another

KEYNOTE SPEAKER

DR. AVI CHAKRABARTTY

PROFESSOR, UNIVERSITY OF TORONTO
PRIVATE-SECTOR SCIENTIST

Avijit (Avi) Chakrabartty was born in Kolkata but raised in Edmonton, where he attended the University of Alberta to obtain his BSc degree in Med. Lab Science and his MSc degree in Experimental Pathology. He completed his Ph.D. in Clinical Biochemistry at the University of Toronto in 1990. Avi was a postdoctoral fellow at the Department of Biochemistry, Stanford University, where he held a fellowship award from the Medical Research Council of Canada. It was at Stanford that he developed an interest in protein folding and worked on both theoretical and experimental aspects of the protein folding problem. In 1994, Avi took up his position as Assistant Professor in the Department of Medical Biophysics, University of Toronto and Senior Scientist at the University Health Network.

The central focus of Avi's research is protein misfolding disease. The inability of cellular machinery to clear misfolded proteins is a recurring theme in many diseases that run the gamut from Alzheimer's disease to cancer. His work concentrates on degenerative diseases: Alzheimer's disease, amyotrophic lateral sclerosis, prion disease, light chain amyloidosis, and transthyretin amyloidosis disease. He utilizes protein structure-based design to produce therapeutic/ diagnostic antibodies and small molecules for ALS, prion disease and TTR amyloidosis.

Avi has published over one hundred scientific papers and holds several patents on therapeutic and diagnostic methods to combat protein misfolding disease. He consults for and has had sponsored research contracts with the following theranostic companies: Neurochem Inc., Caprion Inc., Amorfix Ltd., Prothena Inc., Sanofi S.A., and Novo Nordisk A/S. His most recent company collaboration with Prothena and Novo Nordisk has resulted in 5 approved patent applications and a successful phase 1 clinical trial for the monoclonal antibody drug (Prx004) for transthyretin amyloidosis.

Avi is now a consultant with Novo Nordisk working on a phase 2 clinical trial of Prx004. Avi has also received several awards, including the Annual Boehringer Mannheim Canada Graduate Student Prize for Excellence in Research, the Stuart Allan Hoffman Memorial Prize Awarded for Excellence in Graduate Research in Clinical Biochemistry, the Medical Research Council of Canada Postdoctoral Fellowship, the Premier's Research Excellence Award, presented by the Ministry of Energy, Science, and Technology, Government of Ontario, the David G. Dewar Research Award, Alzheimer Society of Canada, and the Sanofi iAward.

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SCHEDULE

MORNING

REGISTRATION (with coffee/tea and light snacks) | 8:30 - 8:50

Time	Speaker	Presentation
8:50 - 9:00	Dr. Mark Berry	Opening Remarks

SESSION 1 Moderator: Vishal Pandya

9:00 - 9:15	Thilini Kumarasinghe	Antioxidant-enriched fish oil emulsions on gut and liver function during prolonged parenteral feeding
9:15 - 9:30	Khandkar Shaharina Hossain	Effects of dietary vitamin B9 and B12 on gut morphology
9:30 - 9:45	Sathya Amarasena	Effect of vitamin B6 on gut microbial modulation of the kynurenine pathway metabolites in the host brain.
9:45 - 10:00	Zahra Aghaei	Effect of maternal exposure to polystyrene micro- and nanoplastics on placental and fetal development in a mouse model of pregnancy
10:00 - 10:15	Roya Shamsi	Sleep Duration and Quality in Children: Interactions with Food Choices, Energy Balance and Digital Screen-Time (Sleep - FAST)
10:15 - 10:30	Zack Clancy	Parenteral nutrition-induced hypersensitivity to insulin was alleviated by betaine and creatine supplementation in neonatal Yucatan miniature pigs

BREAK | 10:30 - 11:00

KEYNOTE

11:00 - 12:00	Dr. Avi Chakrabartty	Monoclonal therapies for protein misfolding diseases: turning antibodies into chaperones

SCHEDULE

AFTERNOON

LUNCH | 12:00 - 1:00

SESSION 2 Moderator: Efe Obade

1:00 - 1:15	Sina Heravi	Biological crowders' effects on alpha-synuclein structure
1:15 - 1:30	Innocent Uzochukwu Okagu	Maternal diets enriched in omega-3 polyunsaturated fatty acids boost accretion of docosahexaenoic acid-containing ethanolamine plasmalogens in fetal brain of C57BL/6 mice during gestation
1:30 - 1:45	Ava Rasouli	Dietary vitamin B6 and Pathophysiology of Non-alcoholic Fatty Liver Disease (NAFLD)
1:45 - 2:00	Arshad Shaikh	Exploiting cellular signalling in Streptomyces for antibiotic production
2:00 - 2:15	Chandrika Sewwandi Dissanayaka	Phenolic-protein Interactions: Molecular Docking and Simulations
2:15 - 2:30	Hong Dien (Anthony) Phan	CD24 regulates the PI3K/ROCK pathway to promote release of extracellular vesicles from B cell lymphocyte

POSTER SESSION | 2:30 - 3:15

POSTERS

Poster number	Presenter	Title
1	Alexandra Gamble	Relationship of TAAR1 to pro-inflammatory status in human macrophages.
2	Jude Power	The effect of cell lysate crowding on the fibrillization of Alpha-Synuclein
3	Veronica Castro	Signalling Pathway of CD24-mediated Extracellular Vesicles Release
4	Grant Kelly	Evaluating PeptiGel, a Synthetic Hydrogel, as an Effective Substitute for a Biologically Derived Matrix in a Three-Dimensional Co-Culture System
5	Zoë Rowe	Fructose and Energy Balance
6	Emma Jacobs	The Effects of Varying Parenteral Nutrition and Insulin Administration Regimens on the Risk of Hyperglycemia in Hospitalized Patients

SCHEDULE

SESSION 3 Moderator: Innocent Uzochukwu Okagu

3:15 - 3:30	Efe Obade	Addition of soy lecithin to enhace the bioavailability and incorporation of fish oil - OMEGA-3-FATTY ACID
3:30 - 3:45	Modeline Longjohn	Exploring blood extracellular vesicles as novel biomarkers for tracking pediatric B-cell acute lymphoblastic leukemia
3:45 - 4:00	Sean Ezekiel	Developing a potent clinical inhibitor of GGPPS via engineered dimer mutants of the enzyme
4:00 - 4:15	Saman Salari	Investigation on the regulation of mevalonate kinase
4:15 - 4:30	Kandeepan Karthigesu	In Vitro Effect of Vitamins C and E on Lipid Peroxidation of Lipid Emulsion for Total Parenteral Nutrition
4:30 - 4:45	Renan Danielski	Study of the bioaccessibility of phenolic compounds from tropical fruits and their by-products using an in vitro gastrointestinal digestion model

EVENING MIXER & AWARDS | 5:30 - 8:00

Royal Canadian Legion Pleasantville Branch 56
66 The Blvd, St. John's, NL

THILINI
KUMARASINGHE
Department of Biochemistry, Memorial University of Newfoundland

Antioxidant-enriched fish oil emulsions on gut and liver function during prolonged parenteral feeding

Antioxidant-enriched fish oil emulsions on gut and liver function during prolonged parenteral feeding. Intravenous feeding is essential in newborn infants who are extremely premature or present with gastrointestinal tract complications. The survival and development of such infants depend on parenteral nutrition (PN), yet PN-related liver disease and gut atrophy are unavoidable. Clinicians increasingly choose fish oil emulsions as the lipid source in PN because vegetable-based oil emulsions exacerbate parenteral nutrition-associated liver disease (PNALD). PN complications may be related to high levels of oxidative stress contributed by PN as lipids and other nutrients in the diet are oxidized when exposed to light and ambient temperatures prior to delivery. Lipid emulsions are enriched with vitamin E to reduce the peroxidation of long-chain fatty acids. This study aims to evaluate the effectiveness and safety of fish oil emulsions supplemented with greater concentrations of antioxidant vitamins (E and C) on the health of the gut and liver when used for long-term parenteral feeding.

We hypothesized that an antioxidant-supplemented lipid emulsion will increase gut blood flow, reduce gut atrophy, and ameliorate PNALD status in newborn piglets treated with prolonged intravenous feeding. Our objectives are to assess whether a mixed-lipid emulsion that contains fish oil (SMOFlipid®) will improve n-3 PUFA status, reduce indices of PNALD, and enhance gut structure and function. Enhancing the antioxidant concentration of this commercial lipid emulsion may contribute to important therapeutic applications in newborn infants. (Supported by OFI-VRF)

KHANDKAR
SHAHARINA HOSSAIN
Department of Biochemistry, Memorial University of Newfoundland

Effects of dietary vitamin B9 and B12 on gut morphology

Vitamin B9 and B12 play a crucial role as a cofactor in various enzymatic reactions, such as one-carbon (1C) metabolism. Our gut harbours bacteria that can produce these vitamins. However, bacterially synthesized vitamins are limited, and dietary intake is vital in meeting the host's daily requirements. We have previously shown that vitamin B6 deficiency alters gut microbiota and its metabolites. Here, we investigated the effects of vitamin B9 and B12 deficiency on gut microbiota, gut morphology, and metabolites linked to 1C metabolism using a rodent model. Sprague–Dawley rats (N = 47) were fed either control(n=15), low B9 (LB9, n=16) and low B12 (LB12, n=16) diet for six weeks. Body weights were measured weekly. At the end of the study, blood, cecal matter and tissue samples were collected for various biochemical analyses. We found significant differences in the gut morphology. Histological data of the colon shows increases in wall thickness (p=0.02), submucosal thickness (p=0.03), and mucosal height (p=0.01) in LB12 group compared to the control group. Similarly, colon muscle thickness was significantly increased in LB9 rats compared to the control rats (p=0.02). However, we didn't find any significant difference in the body weight, organ weights, and colon length between the groups. Interestingly, we observed a higher secretion of porphyrins from the animal's eyes and nose of the vitamin-deficient group than in the control group, indicating potential higher metabolic stress in those animals. Further studies, such as 16s RNA sequencing of the gut microbial community and targeted metabolites analysis, are needed to fully understand the gut-related effects of dietary vitamin deficiencies on the host.

SATHYA
AMARASENA
Department of Biochemistry, Memorial University of Newfoundland

Effect of vitamin B6 on gut microbial modulation of the kynurenine pathway metabolites in the host brain

The kynurenine pathway (KP) of L-tryptophan metabolism produces several metabolites, which can be either neurotoxic or neuroprotective. Most of the enzymes involved in this pathway require certain B-vitamins as cofactors. For instance, vitamin B6 is required for kynureninase and kynurenine aminotransferase enzymes which catabolize kynurenine to anthranilic acid or 3-hydroxyanthranilic acid, and kynurenine to kynurenic acid or xanthurenic acid, respectively. KP is also the primary pathway to convert L-tryptophan to NAD, a central coenzyme linked to energy production. Similar to mammalian cells, gut microbes metabolize L-tryptophan to produce KP metabolites and other neurologically active molecules such as serotonin and indole. Some of these molecules are permeable to the blood-brain barrier. Because of the profound roles of vitamin B6 in KP, B6 deficiency can significantly impair the homeostasis of KP metabolites. Such imbalance in KP metabolites has been shown to be associated with several chronic diseases, including neurological disorders. Vitamin B6 deficiency also impacts gut flora, and germ-free condition increases blood-brain barrier permeability. Recently, the potential gut microbial manipulation of KP metabolites to prevent or treat neurodegenerative diseases has attracted a great deal of attention. Here, we are interested in understanding the impacts of dietary micronutrients, such as vitamin B6, on the microbiota-gut-brain axis via modulating the KP.

ZAHRA
AGHAEI
Department of Chemistry, Memorial University of Newfoundland

Effect of maternal exposure to polystyrene micro- and nanoplastics on placental and fetal development in a mouse model of pregnancy

Plastics are ubiquitous and when released into the environment, they break down into smaller particles termed microplastics (MPs). These microparticles can be ingested by organisms and potentially accumulate in tissues and organs. Recently, MPs were found in the placentas of healthy women, raising the concern that plastic exposure may have an impact on pregnancy and fetal development. In this project, we studied the effect of maternal exposure to micro- and nanoplastics on placental and fetal growth and on placental metabolism using experimental mice. CD-1 pregnant mice were exposed to 5 μm polystyrene microplastics (PS-MPs) and, 50 nm polystyrene nanoplastics (PS-NPs) in filtered drinking water at one of four environmentally-relevant concentrations (0 ng/L (controls), 102 ng/L, 104 ng/L, 106 ng/L) from embryonic day 0.5 to embryonic day 17.5 (full term is 18.5 days). While the placental weights were constant in all groups at embryonic day 17.5, there was a significant effect on fetal weights, with a dose-dependent decrease in weight in the MP- and NP-exposed fetuses ($p<0.0001$). Maternal exposure to PS-MPs and PS-NPs also impacted the structure of the placenta and resulted in shorter umbilical cord lengths in all MP- and NP-exposed groups. Placental metabolite profiles were determined using 1H high-resolution magic angle spinning magnetic resonance spectroscopy.The relative concentration of lysine ($p=0.003$) and glucose ($p<0.0001$) in the placenta were found to decrease with increasing MP concentration. This study highlights the impact of MP and NP exposure on pregnancy outcomes and that efforts should be made to minimize exposure to plastics, particularly during pregnancy.

ROYA
SHAMSI
Department of Biochemistry, Memorial University of Newfoundland

Sleep Duration and Quality in Children: Interactions with Food Choices, Energy Balance and Digital Screen-Time (Sleep - FAST)

Introduction: Obesity in children is a major risk for many conditions such as insulin resistance, type 2 diabetes, and cardiovascular diseases. Hence, it has become one of the major priorities for public health. To address this issue, it is essential to identify modifiable lifestyle habits linked to childhood obesity including physical activity, sleep, screen time, and eating patterns. Sleep is one of the neglected issues for clinicians, and recent research has shown that sleep patterns can predict Body mass index and macronutrient intake.
Methods: Twenty-two children aged 9-12 were recruited from Newfoundland. Anthropometric data were collected, and BMI was calculated, and percentiles were determined using international reference values. All participants completed demographic, dietary habits, and screen time questionnaires. Sleep duration and quality and physical activity data were obtained using both actigraphy and questionnaires.
Results: Participants were 10.5 (±1.2) years old including 40.9% males, 59.1% females, and 68.2% Caucasian. The mean waist circumference was 65.7 cm (±9.3). Subjective and objective mean sleep duration over seven days were (9:26±0:51, 9:24±0:46 and 6:32±0.53 hours) reported by children, parents and collected by actigraphy, respectively. Self-reported data showed the daily screen time less than 2 hours was 23% for weekdays and 9% for weekends.
Conclusion: Our data suggest that screen time does not affect sleep duration or quality, regardless of sex. We are still analyzing data the following outcome variables: dietary habits and physical activity and how they might affect sleep duration and quality.

ZACK
CLANCY
Department of Biochemistry, Memorial University of Newfoundland

Parenteral nutrition-induced hypersensitivity to insulin was alleviated by betaine and creatine supplementation in neonatal Yucatan miniature pigs

Parenteral nutrition (PN) is a lifesaving nutritional strategy used in cases where infants cannot tolerate oral feeding, thus requiring an intravenous infusion of nutrients. Prolonged PN can induce metabolic and physiological complications ranging from impaired gut functioning to non-alcoholic fatty liver disease and hyperglycemia. Furthermore, gut and liver functions are critical for maintaining proper methyl metabolism and thus are essential for transmethylation reactions, including creatine and phosphatidylcholine synthesis and DNA methylation. Betaine and creatine metabolism play integral roles in methylation reactions by replenishing methyl supply and sparing methyl demand, respectively. Moreover, methyl deficiencies and PN have been associated with an increased risk of glucose intolerance and insulin sensitivity. Therefore, we propose methyl-supplementation, in the form of betaine and creatine, as a means to increase methyl group availability. We hypothesize that control PN feeding will increase insulin resistance and induce a state of glucose intolerance compared to a breastfed control. While methyl-supplemented PN will enhance methionine availability for methylation reactions, thereby modulating DNA methylation and improving glucose and insulin homeostasis. 7-10 day old Yucatan piglets received one of three interventions: control PN, betaine + creatine-supplemented PN or sow-fed for 12 days. The effect of low birth weight was investigated using intrauterine growth restricted (IUGR) piglets placed on control PN. Glucose metabolism in vivo was assessed using an intravenous glucose tolerance test (IVGTT) and insulin sensitivity test (IST). Glucose tolerance and insulin sensitivity were not affected by birth weight but were significantly affected by the use of PN. Compared to sow-fed piglets, piglets fed control PN had higher glucose clearance rates and lower plasma glucose concentration, suggesting a cellular hypersensitivity to circulating insulin; this response was alleviated in piglets on methyl-supplemented PN. The mechanism by which betaine and creatine reduce cellular hypersensitivity to insulin remains unclear and warrants further investigation.

DR. AVI CHAKRABARTTY
Department of Medical Biophysics, University of Toronto

Monoclonal therapies for protein misfolding diseases: turning antibodies into chaperones

Transthyretin amyloidosis (ATTR) is a classic protein misfolding disease, where the tetrameric protein transthyretin (TTR) dissociates into monomers prior to aggregation as amyloid fibrils. While deposition of the amyloid in peripheral nerves causes polyneuropathy (ATTR-PN) and mobility impairments, amyloid deposition in cardiac tissue causes cardiomyopathy (ATTR-CM) and heart failure in the absence of effective treatment. ATTR-CM and ATTR-PN can both occur in the same individual. ATTR-CM has previously been classified as a rare disease; however, with the advent of new diagnostic imaging techniques the disease is being found to be present in 13% of cases of heart failure with preserved ejection fraction. Notably, 3-5% of African descent individuals possess a mutation (V122I) in the TTR gene that predisposes them to ATTR-CM. We have developed a treatment for ATTR-CM/PN that involves the generation of conformation-specific antibodies that entrap non-native conformations of TTR but leaves the native state untouched. These antibodies facilitate native state folding, prevent aggregation of TTR, and actively causes immune clearance of misfolded TTR. In collaboration with Prothena Inc., a humanized version of the monoclonal antibody (PRX004) was tested in a phase 1 clinical trial. The trial initially involved 20 symptomatic individuals with hereditary ATTR-CM. PRX004 was shown to be tolerated safely and have appropriate absorption, distribution, metabolism, and excretion characteristics. Importantly, PRX004 was shown to reduce the concentration of misfolded TTR circulating in the blood by up to 70% and show concomitant improvements in neuropathy and cardiac function. Favorable changes in neuropathy impairment score (NIS) and cardiac global longitudinal strain (GLS) measured by ECHO were observed in all patients. A phase 2 clinical trial for PRX004 will be conducted by Novo Nordisk later this year. The approach we used to develop monoclonal therapeutics for ATTR can be used as a platform for generating similar therapeutics for other protein misfolding diseases.

SINA
HERAVI
Department of Biochemistry, Memorial University of Newfoundland

Biological crowders' effects on alpha-synuclein structure

Alpha-synuclein is an intrinsically disordered protein located in presynaptic neurons; it plays a leading role in vesicle and neurotransmitter release and regulation. In Parkinson's disease, the dysregulation and aggregation of alpha-synuclein lead to Lewy-body deposits and then cell death. The crowded environment inside living cells with a diverse range of macromolecules could be a major factor in alpha-synuclein dysfunction. We evaluated the structure of the alpha-synuclein in the presence of Ficoll70 as an artificial crowder and in bacterial cell lysate as a biological crowder. We found that in the absence of crowders and at physiological temperature and pH, there is no conformational change in the protein, yet there is a slight change in the presence of the artificial Ficoll70 crowders. However, alpha-synuclein's NMR peaks disappeared after 48 hours likely due to its aggregation. In summary, the artificial crowder Ficoll70 didn't have a significant effect on the structure of alpha-synuclein. However, the cell lysate with its natural biocomplexity facilitated alpha-synuclein aggregation at physiological temperature.

INNOCENT
UZOCHUKWU ORAGU
Department of Biochemistry, Memorial University of Newfoundland

Maternal diets enriched in omega-3 polyunsaturated fatty acids boost accretion of docosahexaenoic acid-containing ethanolamine plasmalogens in fetal brain of C57BL/6 mice during gestation

The lipid composition of the brain changes with age and diet. Phosphatidylethanolamine (PE) is the major phospholipid in the brain, of which ethanolamine plasmalogens (EPs) are the most abundant. Plasmalogens possess plasmenyl-linkage at the sn-1 position, and promote neurogenesis, synaptogenesis and myelination. Little is known how maternal diet influences the levels of EPs in fetal brain during gestation. We hypothesized that maternal diets having an adequate quantity of n-3 polyunsaturated fatty acids (PUFA) will support the accretion of EPs-enriched species that are important in fetal brain development and function. Eight-week-old female C57BL/6 mice were divided into four groups (n=8), and were fed semi-purified diets containing 20% w/w fat with low or high n-3 PUFA composed of n-6:n-3 of 40:1 and 5:1, respectively, for two weeks prior to mating. The females were maintained on assigned diets until either gestation day (GD)12.5 or 18.5 when fetal brains were dissected for lipidomic analyses using UHPLC/MS/MS. Data were analyzed using X-Calibur and LipidSearch softwares, and statistical analysis was performed using Graphpad Prism. Higher levels of docosahexaenoic acid (DHA)-EPs {PE(16:0p/22:6); PE(18:0p/22:6); PE(18:1p/22:6)} were observed in fetal brain of high n-3 PUFA mice at both GD, compared to low n-3 PUFA mice. Oleic acid {PE(16:0p/18:1)}, arachidonic acid {PE(16:0p/20:4)}, and adrenic acid {PE(16:0p/22:4); PE(18:0p/22:4)}-containing EPs decreased as gestation progressed. We are the first to demonstrate that maternal diet rich in n-3 PUFA promoted the accretion of DHA-containing EPs in fetal brain as early as gestation day 12.5, which may have crucial roles in neurogenesis, synaptogenesis and myelination.

AVA
RASOULI

Department of Biochemistry, Memorial University of Newfoundland

Dietary vitamin B6 and Pathophysiology of Non-alcoholic Fatty Liver Disease (NAFLD)

Non-alcoholic fatty liver disease (NAFLD) is a chronic liver condition affecting many people worldwide. It begins with simple steatosis (hepatic lipid accumulation) and further develops into non-alcoholic steatohepatitis (NASH), fibrosis, irreversible liver conditions (cirrhosis), and hepatocellular carcinoma (HCC). NAFLD is a multifactorial disorder, and currently, it has no approved pharmacotherapeutic management. Several studies have indicated the association between dietary micronutrients and the development of NAFLD. One specific example is the impairment of one-carbon (1C) metabolism. Vitamin B6 works as a cofactor in some of the 1C metabolism enzymatic reactions, which regulates the packaging and secretion of triglyceride (TG) in very-low-density lipoprotein (VLDL). Low vitamin B6 causes impairment in 1C metabolism. This affects VLDL secretion and ends in NAFLD manifestation. We aim to study the effects of dietary B6 supplementation on the onset and progression of NAFLD through its role in 1C metabolism for eight weeks. We will induce NAFLD in male C57BL/6J mice with a high-fat, high-sugar (HFHS) diet, high (n=8) and low (n=8) in vitamin B6, and compare it to a control group (n=8). We will then monitor different biological specimens (plasma, urine, and fecal samples) with metabolomics assays such as proton nuclear magnetic resonance (1H-NMR) and liquid chromatography mass spectrometry (LC-MS). We will also collect liver tissue samples for histopathological examinations. We hope that Vitamin B6 administration regulates hepatic fat accumulation and works as a therapeutic management for NAFLD.

ARSHAD SHAIKH

Department of Biology, Memorial University of Newfoundland

Exploiting cellular signalling in Streptomyces for antibiotic production

The discovery of bacterial specialized metabolites and their use as antibacterial, antifungal and anticancer agents establish one of the most significant advances in medicine, but the growing threat of antibiotic resistance needs to be addressed. This requires the development of new antimicrobial compounds using new approaches. The Streptomyces synthesize a large variety of specialized metabolites and have numerous biosynthetic gene clusters (BGCs) responsible for their production. Based on the numbers of BGCs present in different Streptomyces species, it has been found that up to 90% of specialized metabolites are not produced under laboratory conditions and are known as “cryptic” since their identities and functions are unknown. Therefore, it is important to understand cellular signalling pathways that are responsible for the adaptation of bacteria in diverse environmental conditions, to activate such “cryptic” BGCs for specialized metabolites production for exploitation. Our focus is on Streptomyces clavuligerus, a producer of clavulanic acid, which is a potent β-lactamase inhibitor. Various genes situated in signalling pathways of S. clavuligerus are involved in the regulation of clavulanic acid either positively or negatively, making them good candidates for study. Overexpression and mutation of these genes can lead to increased clavulanic acid production and in the activation of
cryptic” BGCs.

CHANDRIKA
SEWWANDI DISSANAYAKA
Department of Biochemistry, Memorial University of Newfoundland

Phenolic-protein Interactions: Molecular Docking and Simulations

Phenolic compounds are ubiquitous plant secondary metabolites that possess various biological activities and are known to interact with proteins, altering their structure and properties. Therefore, interactions between these compounds gained increasing attention due to their potential benefits for human health and the food industry. Phenolic compounds and proteins can form complexes via covalent linkages and/or hydrophobic, electrostatic, van der Waals forces and hydrogen bonding known as non-covalent linkages. This presentation describes possible mechanisms of phenol-protein complex formation, their physiological action and activities that are important in the food industry, and possible outcomes in the terms of molecular docking and simulation analysis. According to studies so far, conformational changes of the protein upon binding of polyphenols can lead to the folding or unfolding of the protein molecules, forming insoluble or soluble complexes. The concentration of polyphenols, their molecular weight and structure, ions/cofactors and conditions of the system determine the precipitation or solubilization of the complex, affecting their nutritional and functional properties as well as their bioactivities. Molecular docking and simulation studies of phenolic-protein interactions allows comprehensive virtual screening of competitive/non-competitive and site-specific/non-specific conjugation of phenolics with different protein targets and facilitate understanding the effect of manipulated compounds. Thus, combining molecular docking and simulation studies with experimental techniques is vital for better understanding of the reactions that take place during digestion to engineer and manufacture novel food materials with desirable functional and pharmacological properties.

HONG DIEN (ANTHONY) PHAN
Department of Biochemistry, Memorial University of Newfoundland

CD24 regulates the PI3K/ROCK pathway to promote release of extracellular vesicles from B cell lymphocyte

CD24 is a small glycophosphatidylinositol-linked protein localized to lipid rafts on the plasma membrane and regulates B cell development and maturation in the bone marrow. We previously reported that the engagement of CD24 on B lymphoma cells causes the release of extracellular vesicles (EVs). EVs are membrane-encapsulated nanosized particles containing bioactive cargo, including proteins, lipids, and genetic materials. We next found that stimulation of CD24 on donor cells promotes EV trafficking of lipid and membrane proteins between B lymphocytes to transport functional receptors to recipient cells. However, the underlying mechanisms that govern EV formation in response to the engagement of CD24 remain elusive. Thus, this study aimed to determine how CD24-mediated EV release is regulated. To address this issue, we continued to employ a model system where donor cells expressing palmitoylated GFP (WEHI-231-GFP cells) were stimulated and then co-incubated with recipient cells expressing palmitoylated tdTomato that lack membrane-bound IgM (WEHI-303-tdTomato cells). Donor cells were treated with chemical inhibitors of select signaling pathways and EV transfer to recipient cells was monitored. Our findings provide clear evidence that PI3K and ROCK signaling pathways, likely via regulation of the actin cytoskeleton and ceramide production, mediate the release of EVs by CD24.

EFE
OBADE
Department of Biochemistry, Memorial University of Newfoundland

Addition of Soy Lecithin to enhance the bioavailibity and incorporation of fish oil - OMEGA-3-FATTY ACID

Long-chain omega-3 polyunsaturated fatty acids (LC omega-3 PUFA) are important in regulating inflammation, maintaining cell membrane integrity, and required for healthy growth and development. For omega-3 fatty acids to perform their biological function, they must first be bioavailable to the target tissues. Studies have shown that numerous factors affect their bioavailability, such as molecular carrier (ethyl ester (EE), triacylglycerol (TAG), phospholipid (PL), positional distribution, and high-fat consumption.While fatty fish is the best dietary source of LC omega- 3 PUFA, fish oils supplements are a common source for many people. Phospholipids, such as phosphatidylcholine, may improve the efficiency at which the omega-3 fatty acids are utilized in target tissues when added to fish oil. 24 adult Yucatan miniature pigs (females, 6-8 months old) were used in this study and divided into three iso-caloric diet groups (Western diet (WD), western diet plus fish oil (FO), and western diet plus fish oil and phosphatidylcholine (FO-PC)); they were fed ad-libitum for four hours per day. The objective of this study was to determine if 1) the FO group affected the distribution of fatty acids in various organs compared to WD and 2) if PC added to fish oil enhanced the bioavailability of omega-3 fatty acids. After four months of feed intake various tissue samples were collected to analyze different fatty acid distribution. Fatty acid and lipid analysis was assessed in various organs and tissues. There was no difference in omega-6 fatty acids among the three treatment groups, and the saturated fatty acid level was increased in the WD group compared to other treatment groups. The level of omega-3 fatty acid was significantly increased in the FO group compared to the WD group.

Interestingly, PC supplementation had the highest distribution and incorporation of omega-3 fatty acids in the brain (12.3%±5.6%) and backfat (1.6% ±0.4%) compared to the other diet groups. In contrast, the FO group had the highest distribution in the liver (14.6% ±5.4%) ($p<0.05$). This study's outcomes could identify the beneficial effects of supplemental phosphatidylcholine with fish oil in improving the bioavailability of omega-3 fatty acids.

MODELINE
LONGJOHN

Department of Biochemistry, Memorial University of Newfoundland

Exploring blood extracellular vesicles as novel biomarkers for tracking pediatric B-cell acute lymphoblastic leukemia

B cell acute lymphoblastic leukemia (B-ALL) is a hematological malignancy in which immature B lymphoblasts accumulate and out crowd other cells in the blood, from which they can infiltrate the bone marrow (BM) and cerebrospinal fluid (CSF). B-ALL is one of the most prevalent pediatric cancers, with a cure rate of up to 90%, which reduces to 30-50% upon relapse. A key prognosticator of B-ALL relapse is measurable residual disease (MRD), where lymphoblasts that evade cytotoxic effects of chemotherapy persist in the BM and CSF. Current methods for tracking MRD are invasive, laborious and cannot be done frequently enough, which necessitates exploring new methods. For this, we look to extracellular vesicles (EVs) – lipid membrane-bound nanoparticles which carry bioactive cargo. Previous studies showed that cancer cells secrete >104 EVs, which can cross membrane barriers and thus be important in B-ALL MRD. To explore this approach, we first tried identifying a suitable EV isolation technique by comparing peptide-affinity, polyethylene glycol, and size exclusion chromatography-based methods. Next, we analyzed the size, concentration, and nucleic acid cargo of healthy donor (HD) and B-ALL blood plasma EVs. Preliminary data showed that the peptide-affinity-based method isolated a more comprehensive EV population than the others. Also, B-ALL plasma had more EVs than HD, with an enrichment of 30-160 nm. Furthermore, we found a nucleic acid cargo signature of EVs, differentiating B-ALL from HD. The next direction will be validating the identified nucleic acid signature in blood, BM, and CSF.

SEAN
EZEKIEL
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Developing a potent clinical inhibitor of GGPPS via engineered dimer mutants of the enzyme

The enzyme geranylgeranyl pyrophosphate synthase (GGPPS) catalyzes the production of geranylgeranyl pyrophosphate (GGPP), an isoprenoid that plays a key role in cell signalling. Owing to its implications in cellular processes involved in multiple forms of cancer as well as Alzheimer's disease, GGPPS has been studied extensively. However, efforts to elucidate its detail structure and design a clinically relevant inhibitor have been mostly unsuccessful.

Modern drug discovery and design have relied on the synthesis and screening of large numbers of compounds to identify potential candidates, a costly and time-consuming process. This process has become streamlined in the past two decades using computer-aided drug design approaches such as structure-based drug design and one of its subset methods, structure-based virtual screening. Structure-based drug design relies on the 3D structure of the drug target determined by experimental techniques such as X-ray crystallography and NMR to investigate the ligand-protein binding interactions in atomic-level detail. A current obstacle with structure- based drug design is that GGPPS is not amenable to X-ray crystallographic methods.

In my presentation, I discuss how I am employing computer-aided drug design in the search for novel inhibitors of GGPPS. I will also briefly introduce an interesting solution that may solve the difficulty in using GGPPS with X-ray crystallography that our lab has been developing.

SAMAN
SALARI
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Investigation on the regulation of mevalonate kinase

The mevalonate (MVA) pathway is a fundamental metabolic pathway for isoprenoid synthesis in the cell. Mevalonate kinase (MK) is an enzyme within this pathway that converts mevalonic acid to mevalonate-5-phosphate, using ATP as a phosphoryl donor. A mutation in the gene encoding MK causes mevalonate kinase deficiency which is affecting the immune system. Farnesyl pyrophosphate (FPP), a downstream metabolite of MVA pathway, was shown to inhibit human MK (hMK). Crystallographic analysis of rat MK indicated that FPP inhibits the enzyme by blocking its ATP binding site. Interestingly, FPP is also involved in the feedback inhibition of farnesyl pyrophosphate synthase (FPPS), the enzyme responsible to produce FPP. This provides evidence for multilayered control of MVA pathway and raises the possibility of yet undiscovered feedback loops. In this study, we aim to further investigate the regulation of hMK. Our questions are: 1) Can FPP-mimicking inhibitors of FPPS also inhibit hMK? 2) Can other endogenous metabolites inhibit hMK? 3) if so, what are the atomic details of the enzyme-inhibitor interaction? The first step is to establish a reliable enzyme assay protocol. To this end, I have expressed and purified a recombinant form of hMK. Using a pyruvate kinase/lactate dehydrogenase-coupled assay, I will examine the effect of FPPS inhibitors on hMK activity. In the meantime, I will screen a large virtual library of endogenous metabolites to identify other potential physiological effectors of hMK. A better understanding of this enzyme is important for that of MVA pathway and may lead to novel therapeutic insights.

KANDEEPAN KARTHIGESU
Department of Biochemistry, Memorial University of Newfoundland

In Vitro Effect of Vitamins C and E on Lipid Peroxidation of Lipid Emulsion for Total Parenteral Nutrition

Parenteral administration of nutrients, which refers to total parenteral nutrition (TPN), is vital to neonates with prematurity or low-birth weight. It is prepared by mixing elemental nutrients with lipid emulsion, which results in the generation of peroxidation. Such neonates may also have an immature antioxidant system, which cannot neutralize overwhelming oxidants from TPN. Several studies have suggested that adding antioxidants may improve the neonates' antioxidant status and reduce complications, including liver disease and gut atrophy. However, the in vitro effect of antioxidant vitamins C and/or E supplementation for TPN on the peroxide levels is still unknown. Thus, we hypothesized that adding vitamin C and/or E to the lipid emulsion will reduce the generation of peroxide levels. We aimed to assess the lipid peroxidation of the commercially available lipid emulsion SMOFlipid . We used THP-1 human monocytic cells to assess the peroxidation. Light-protected SMOFlipid (1%) was added to the cells and incubated for 16 hours at 37°C. After incubations, cells and media were separated. Cell viability and metabolic activity were determined using a Trypan blue exclusion and MTT assay, respectively. Lipid peroxidation was measured from the supernatant using a thiobarbituric acid reactive substances (TBARS) assay. Cell viability or metabolic activity of THP-1 cells was not affected by increasing concentration of vitamin C from 0 to 40 µM, while the metabolic activity was gradually increased by increasing concentrations of vitamin E from 0 to 160 µM (n=3; p 0.05). The vitamin C treated cells (5-40 µM) did not reduce the TBARS levels (n=3; p 0.05), while increasing levels of vitamin E treated cells reduced the TBARS levels (at 0 µM= 1.17 µM vs at 40 µM=0.72 µM (n=1)). We conclude that vitamin E reduced the peroxide generation by THP-1 cells. Further studies are needed to examine the synergistic effect of vitamins C and E for SMOFlipid and all-in-one TPN to examine the peroxide levels.

RENAN
DANIELSKI
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Study of the bioaccessibility of phenolic compounds from tropical fruits and their by-products using an in vitro gastrointestinal digestion model

The tropical fruits guava, jerivá, butiá, and their processing discards (e.g., seeds, peels, pulp leftovers) are excellent sources of phenolic compounds with antioxidant capacity. To efficiently exert their bioactivities, phenolics must be released from the matrix and become available for intestinal absorption following digestion. However, there is no information about the bioaccessibility of phenolics from guava, jerivá, butiá, and their waste fractions. Therefore, an in vitro gastrointestinal (GI) digestion model consisting of oral, gastric, small, and large intestinal phases was used to assess the bioaccessibility of tropical fruits' phenolics. The digested extracts were analyzed for total phenolic (TPC) and total flavonoid (TFC) contents, as well as antiradical activity against DPPH and ABTS radicals and ferric reducing antioxidant power (FRAP). The results showed that jerivá and butiá phenolics were mainly released after gastric and large intestinal digestion, while guava phenolics were predominantly released in the large intestinal phase. Extracts from the gastric phase showed the best results in DPPH and FRAP assays, except for butiá seeds, where the large intestinal extract demonstrated the highest antiradical potential. Nevertheless, large intestinal extracts from jerivá pulp, guava pulp, and butiá (pulp and seeds) were the most efficient at inhibiting ABTS radical. These differences may be due to possible modification of phenolic structures throughout GI digestion, directly impacting their antioxidant activity. Overall, phenolic bioaccessibility was low to moderate (3-54%). Future in-progress studies include the assessment of the effect of encapsulation on the phenolics of tropical fruits as a strategy to increase their bioefficiency.

ALEXANDRA GAMBLE
Department of Biochemistry, Memorial University of Newfoundland

Relationship of TAAR1 to pro-inflammatory status in human macrophages

TAAR1 is a G protein-coupled receptor that has recently been associated with immune function. Macrophages are large mononuclear phagocytes that play essential roles in the immune system, including cytokine release, phagocytosis, and antigen presentation. Macrophages primarily exist in two polarization states. The M1 state is functionally pro-inflammatory, while the M2 state is anti-inflammatory. The M1 state is also associated with a pronounced metabolic shift toward glycolytic ATP production. We have previously shown that TAAR1 localization in human monocyte-derived macrophages shifts from nuclear to cytoplasmic after M1 stimulation. In mouse bone-marrow derived macrophages, TAAR1 agonism inhibits TNF secretion, but does not affect metabolic re-programming, associated with damage-associated-molecular-pattern (DAMP) induced M1 stimulation, but not the less clinically relevant lipopolysaccharide (LPS)-induced stimulation. Here we extend these results using the more accessible THP-1 cell line, a human acute monocytic leukemia line derived from a 1-year-old male that can be differentiated into macrophages. A 100-fold increase in TNF secretion confirmed LPS-induced M1 polarization. Using an anti-human TAAR1 antibody, as previously seen in primary human cells, TAAR1 localization shifted from nuclear in unstimulated cells to cytoplasmic following LPS treatment. Energy metabolism was then evaluated using the Agilent Seahorse XF Real-Time ATP Rate Assay. By measuring oxygen consumption rate (mitochondrial) and extracellular acidification rate (glycolytic) we observed, as expected, that LPS stimulation promoted glycolytic energy metabolism. TAAR1 agonists had no effect either alone or following LPS treatment. Since a similar lack of effect of TAAR1 agonists on LPS-mediated stimulation was previously seen in mouse macrophages, although responses to damage-associated-molecular-pattern (DAMP) signals were modified, future studies will examine the effect of TAAR1 agonists on human DAMP-signals.

JUDE
POWER
Department of Biochemistry, Memorial University of Newfoundland

The Effect of Cell Lysate Crowding On The Fibrillization of Alpha-Synuclein

Alpha-Synuclein is a presynaptic neuronal protein that has been determined to be a major contributor to the development and progression of Parkinson's disease within humans. The fibrillization of alpha-synuclein is of particular interest, as Parkinson's disease and many other neurodegenerative diseases are characterized by the formation of amyloid fibrils.

Macromolecular crowding in cells affects many biomolecular characteristics including protein-protein binding and fibril formation. This occurs through the excluded volume effect, where a higher concentration of macromolecules reduces the water activity of the solution, as well as through soft interactions between the protein and the crowder. Cell lysate is used as a macromolecular crowder for this study, as it provides an environment that mimics the crowded intracellular milieu. French Press is used to mechanically lyse bacteria. For my project, I studied the effect that the cell lysate crowder has on the fibrillization of alpha - synuclein compared to a known macromolecular crowder, ficoll70, which enhances fibrillization, as well as to the alpha-synuclein without crowder. The study used three different concentrations of each crowder; 50 mg/mL, 100 mg/mL, 200 mg/mL, at 25 °C with an alpha - synuclein concentration of 0.050 mM in order to compare the effects of the cell lysate to the ficoll70. To determine the impact of each crowder on the fibrillization of alpha-synuclein I performed a fluorescence assay using Thioflavin-T, which will bind to the amyloid fibrils that are formed over the 240 hour experiment.

VERONICA
CASTRO
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Signaling Pathway of CD24-mediated Extracellular Vesicles Release

CD24 is a glycophosphatidylinositol-linked cell surface receptor that has been associated with apoptosis, cancer, inflammation, and autoimmune disorders. CD24 is highly expressed on precursor B cells and plays a role in B cell selection and development. Dr. Christian's laboratory has found that CD24 stimulation triggers the release of extracellular vesicle (EVs) from murine immature B cells. EVs are membrane-encapsulated nanoparticles that allow the transfer of biomolecules between cells. EVs can modify cellular processes on the recipient cells and, therefore, are critical for cellular communication. However, the molecular pathway involved in CD24-mediate EV release has not been fully elucidated. Recent observations suggest that CD24-mediated EV release is dependent on phosphoinositide-3-kinase/protein kinase B (PI3K-Akt) and Rho-associated protein kinases (ROCK) pathway. Thus, we hypothesized that stimulation of CD24 triggers EVs release via PI3K activation, leading to phosphorylation of Akt, and ROCK pathway, inducing actin cytoskeletal remodelling. Our model used B cells labelled with palmitoylated green fluorescent protein (WEHI-231-GFP) and signaling inhibitors of PI3K and ROCK to study the regulation of CD24-mediated EV release. WEHI-231-GFP cells were stimulated with anti-CD24 for different time periods. Western blot analysis was performed to study changes in AKT, which is downstream of PI3K, and Cofilin, which is downstream of ROCK. CD24 stimulations induced phosphorylation of AKT, as well as dephosphorylation of Cofilin. Treatment of LY294002, which blocks PI3K, resulted in a reduction of CD24-induced phosphorylated AKT. Our research shows that CD24 induces activation of PI3K and ROCK signaling pathways.

GRANT
KELLY
Department of Biochemistry, Memorial University of Newfoundland

Evaluating PeptiGel, a Synthetic Hydrogel, as an Effective Substitute for a Biologically Derived Matrix in a Three-Dimensional Co-Culture System

The epithelial-to-mesenchymal transition (EMT) and its reverse process, the mesenchymal-to-epithelial transition (MET), are known to be critical steps in cancer metastasis. Adipocytes are known to be able to induce both EMTs and METs in triple negative breast cancer (TNBC) cells. Our lab previously established a three-dimensional (3D) co-culture system that more closely mimics conditions in vivo; revealing that TNBC cells undergo a partial MET when co-cultured with adipocytes. The system employs the use of a biologically derived matrix, Matrigel (Mg) (Corning Life Sciences), to replicate cell-ECM interactions. Recently more advanced synthetic alternatives to the intrinsically variable and difficult to access Mg – such as PeptiGel (Manchester BIOGEL), a self assembling peptide matrix – have also been shown capable of reproducing cell-ECM interactions. My aim is to determine if the synthetic matrix PeptiGel is a viable ECM substitute for the previously established 3D co-culture system and is thus capable of facilitating a partial MET. Confocal Imaging and immunostaining will be used to determine whether PeptiGel facilitates a partial MET in TNBC cells when co-cultured with adipocytes. The partial MET described previously is marked by a distinct morphological change in TNBC cells, which shift from stellate colonies to more round/grape-like shaped colonies. As well, the increase in expression of epithelial markers while maintaining levels of mesenchymal markers, visualized through immunohistochemistry, indicates a partial transition into a hybrid mesenchymal/epithelial state. Should TNBCs co-cultured with adipocytes undergo a partial MET in the presence of PeptiGel as an ECM substitute, then it is a feasible replacement for Matrigel in the previously established 3D co-culture system.

ZOË
ROWE
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Fructose and Energy Balance

The intake of dietary fructose has increased significantly since the 1970s, as it is a commonly used sweetener in sugary drinks, sauces, and baked goods. There is overwhelming evidence from animal and human studies that chronically high fructose consumption is associated with cardiovascular diseases, obesity, and insulin resistance. The metabolic pathway of fructose once consumed remains understudied and many previous studies have investigated unrealistic circumstances of fructose intakes. To better understand the metabolic pathway of fructose in mammals, a group of adult C57BL/6J mice were separated by sex and randomly divided into three groups. For a period of 18 weeks, each group's diet consisted of either 0%, 10% or 20% of total caloric intake from fructose. The fatty acid composition of the kidney and the metabolic pathway of fructose was studied using lipid extraction and stable isotope techniques.

EMMA
JACOBS
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The Effects of Varying Parenteral Nutrition and Insulin Administration Regimens on the Risk of Hyperglycemia in Hospitalized Patients: A Systematic Review

Parenteral nutrition therapy involves administering essential nutrients intravenously. For individuals who are unable to consume food by mouth, this intervention can be lifesaving. However, parenteral nutrition therapy is associated with numerous risks and complications, including hyperglycemia, which can be dangerous in already-ill patients. Insulin is often administered to patients receiving parenteral nutrition therapy to both prevent and treat hyperglycemia. A vast array of administration regimens are used to administer insulin, including adding insulin to the nutrient admixture or delivery via subcutaneous injection. Further differences exist in the forms of insulin delivered and the dosing regimens. Despite a high prevalence of hyperglycemia amongst individuals receiving parenteral nutrition therapy, there is a lack of consensus surrounding which treatment regimens produce the most favourable outcomes for patients. This systematic review was conducted in an attempt to analyze available literature and observe patterns in treatments and results that may demonstrate differences in insulin regimen outcomes. Pubmed, EMBASE, Scopus, Web of Science and Cochrane databases were systematically searched, yielding 1173 publications. After two rounds of screening against specified criteria, 4 articles were selected for inclusion in the review. Detailed evaluation of articles revealed much heterogeneity within the literature on this topic. Nonetheless, the findings suggest that multiple insulin regimens can provide similar control of blood glucose levels in patients receiving parenteral nutrition therapy, though some regimens are accompanied by greater risks of complications, including hypoglycemia. More research is needed to assess the differences between insulin regimens to determine if one may be most advantageous.

AWARDS

L. M. Stead Award

Best M.Sc. student oral presentation

Best Ph.D. student oral presentation

Runner-up Prizes

2nd place M.Sc. student oral presentation

2nd place Ph.D. student oral presentation

Poster Prizes

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2nd place poster presentation

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