April 19, 2023

2023 Nutrition Symposium

University of Illinois Urbana-Champaign | College of Agricultural, Consumer & Environmental Sciences | Division of Nutritional Sciences




College of Agricultural, Consumer & Environmental Sciences
Division of Nutritional Sciences

NSGSA
Nutritional Sciences
Graduate Student Association

On behalf of the Nutritional Sciences Graduate Student Association (NSGSA), the Division of Nutritional Sciences (DNS), and all participating presenters, we would like to welcome you to the 2023 Nutrition Symposium at the University of Illinois Urbana-Champaign!

The Nutrition Symposium is an important event for sharing ideas across disciplines and with the community. Started in 1994 by NSGSA, the symposium offers graduate students with nutrition-related research on campus an opportunity to present prior to annual national and international scientific meetings and conferences. This symposium offers a first glance at exciting research in areas including metabolic regulation, cancer, gastrointestinal physiology, immunology, physical activity, public health, and bioactive plant compounds. Students will be traveling to present their work at a variety of national and international conferences.

This year, we are honored to have Dr. Katherine Ryan Amato deliver the keynote address, "Exploring the potential of the gut microbiota to buffer hosts against nutritional stress". Additionally, NSGSA is proud to highlight the work of world-class faculty members through a mini-symposium. This year's presentations highlight research on environmental impacts on nutrition and health and will feature, Drs. Brett Loman, Melissa Pflugh Prescott, and Jacob Allen.

We are grateful to the many people involved with this meeting and program. We would first like to thank our keynote speaker, Dr. Katherine Ryan Amato. Thank you also to our sponsors – their support is essential to the success and quality of the program. We would also like to recognize the NSGSA Steering Committee and the symposium planning committee, whose members have worked long and hard to organize an excellent program. Most of all, we would like to thank our session chairs, judges, presenters, and attendees for participating in this year's event and making them a success.

The Nutritional Sciences Graduate Student Association Chair and Chair-Elect

nutrsci.illinois.edu

(Cover Image) Do you have to throw culture out the window when following a healthy dietary pattern? Absolutely not! Maribel Barragan is evaluating the impact of a culturally tailored dietary intervention on dietary adherence and metabolic health.

Research image by Maribel Barragan.

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8:15 a.m. – 8:50 a.m. ..... **Welcome Breakfast**
Sims Executive Conference Room, ACES Library
Sponsors, presenters, DNS students/faculty/staff invited

8:50 a.m. – 9:00 a.m. ..... **Break**

***9:00 a.m. – 10:00 a.m.** ..... **Oral Session 1: Nutrient Metabolism and Dietary Patterns**
Monsanto Room, ACES Library
9:00 - 9:15: Madelyn Bradley
9:15 - 9:30: Tori Holthaus
9:30 - 9:45: Megumi Hashida
9:45 - 10:00: Ayça N. Mogol

10:00 a.m. – 10:15 a.m. ..... **Break**

***10:15 a.m. – 11:15 a.m.** ..... **Oral Session 2: Community Nutrition**
Monsanto Room, ACES Library
10:15 - 10:30: Amirah Burton-Obanla
10:30 - 10:45: Ashleigh Oliveira
10:45 - 11:00: Christian Maino Vieytes
11:00 - 11:15: Emily Siebert

11:15 a.m. – 11:45 a.m. ..... **Outstanding Faculty Award Presentation**

11:45 a.m. – 12:45 p.m. ..... **Sponsor Network Lunch**
Heritage Room, ACES Library
Sponsors, presenters, DNS students invited; RSVP required

***12:45 p.m. – 2:45 p.m.** ..... **Faculty Symposium: Environmental Impacts on Nutrition and Health**
Monsanto Room, ACES Library
12:45 - 1:20: Dr. Brett Loman
1:20 - 1:55: Dr. Melissa Pflugh Prescott
1:55 - 2:10: Break
2:10 - 2:45: Dr. Jacob Allen

2:45 p.m. – 3:00 p.m. ..... **Break**

3:00 p.m. – 3:45 p.m. ..... **Sponsor Panel**
Bevier Commons, Bevier Hall
Sponsors, presenters, DNS students/faculty/staff invited

3:45 p.m. – 4:00 p.m. ..... **Break**

***4:00 p.m. – 5:00 p.m.** ..... **Keynote Address: Exploring the potential of the gut microbiota to buffer hosts against nutritional stress**
Katherine Ryan Amato, PhD, Northwestern University
150 Animal Sciences Labratory

5:00 p.m. – 5:15 p.m. ..... **Break**

***5:15 p.m. – 6:45 p.m.** ..... **Graduate Student Poster Session**
Heritage Room, ACES Library
Evening Reception; Award Announcements
Sponsors, presenters, DNS Students/faculty/staff invited

* Open to general public

The Nutritional Sciences Graduate Student Association (NSGSA) was founded in the spring of 1973 by students in the program. The mission of the organization is to provide a means of communication among graduate students, faculty, and alumni of the Division of Nutritional Sciences (DNS), which spans multiple colleges and departments.

NSGSA serves as a forum for student opinion and input and provides students the opportunity to expand their experiences as graduate students. Our activities reflect our desire to enrich our experiences as graduate students and promote the importance of the nutritional sciences discipline both within the university and among the surrounding communities of Champaign and Urbana.

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NSGSA Steering Committee photographs by Maegan Savage Photography

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Ayça N, Mogol
Breanna Nichole Metras

Session Judges

Oral Session 1: Nutrient Metabolism and Dietary Patterns

MC: Olufemi Fabusoro

Judge: Dr. Katherine Amato
Judge: Dr. Sharon Donovan
Judge: Dr. François Reichardt

Oral Session 2: Community Nutrition

MC: Hanchu Dai

Judge: Dr. Alicia Arredondo
Judge: Dr. Elvira de Mejia
Judge: Dr. Naiman Khan

Poster Session

Dietary Intervention

Judge: Dr. Elvira de Mejia
Judge: Dr. Diego Hernandez-Saavedra

Dietary Intervention Models I

Judge: Dr. Maria R. Cattai de Godoy
Judge: Dr. Brett Loman

Dietary Intervention Models II

Judge: Dr. Michael J. Miller
Judge: Dr. Weinan Zhou

Clinical and Preclinical Metabolism I

Judge: Dr. Yuan-Xiang Pan
Judge: Dr. Matthew Dean

Clinical and Preclinical Metabolism II

Judge: Dr. Jaume Amengual Terrasa
Judge: Dr. Elisa Caetano

Cardiovascular Health

Judge: Dr. John Erdman Jr.
Judge: Dr. Neda Seyedsadjadi

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Student Oral Session 1:
Nutrient Metabolism and Dietary Patterns

9:00 a.m. - 10:00 a.m.
Monsanto Room, ACES Library

Student Oral Session 2:
Community Nutrition

10:15 a.m. - 11:15 a.m.
Monsanto Room, ACES Library

Faculty Mini-Symposium:
"Environmental Impacts on Nutrition and Health"

12:45 p.m. - 2:45 p.m.
Monsanto Room, ACES Library

Graduate Student Poster Sessions

5:15 p.m. - 6:45 p.m.
Heritage Room, ACES Library
See poster session floor plan map on page 49

Dietary Intervention

Dietary Intervention Models I

Dietary Intervention Models II

Clinical & Preclinical Metabolism I

Clinical & Preclinical Metabolism II

Cardiovascular Health

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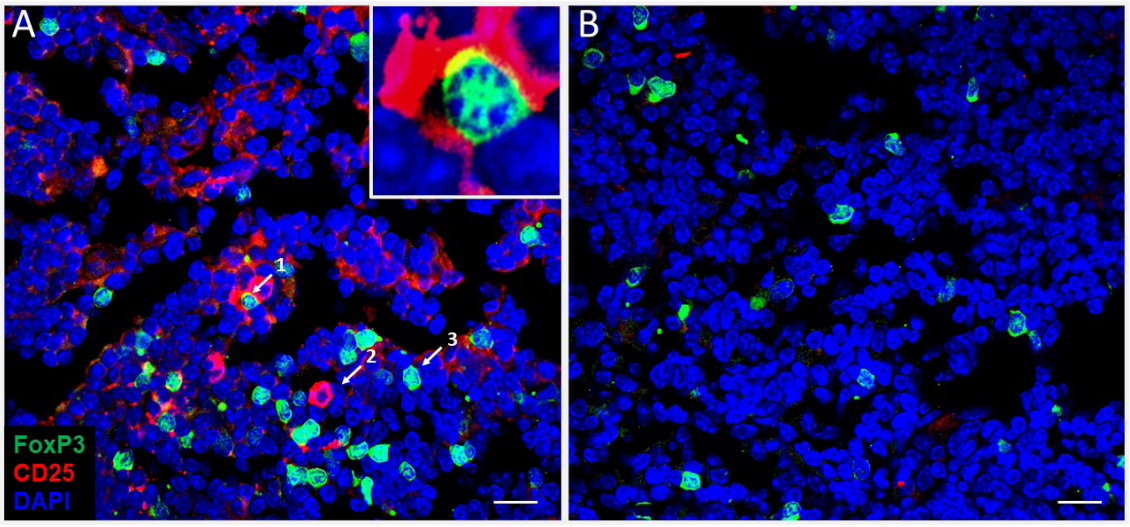

T regulatory cells (Tregs) immunofluorescent staining in lymph nodes before and after treated with anti-CD25 antibody. To evaluate the role of Tregs in atherosclerosis we used an anti-CD25 monoclonal antibody, a common marker for Tregs cells. Nonetheless, anti-CD25 did not completely eliminate Tregs. To assess this issue, we quantified for another Tregs marker, FoxP3, in the lymph nodes. We attributed this issue to the presence of a subpopulation of Tregs single positive for FoxP3 (green) and negative for CD25 (red). A, Before treatment with anti- CD25 antibody. 1) Double positive CD25+FoxP3+ T regs cells, 2) single positive CD25+ FoxP3-Treg, 3) single positive CD25-FoxP3+ Treg. B, After treatment with anti-CD25 antibody. Single positive CD25-FoxP3+ Tregs were still present as those were not depleted by the anti-CD25 antibody. Scale bar at 20 µm.

Research image by Amparo Blanco Cirer

The University of Illinois Division of Nutritional Sciences and the Nutritional Sciences Graduate Student Association would like to acknowledge the generosity of the sponsors and friends of our 2023 Nutrition Symposium.

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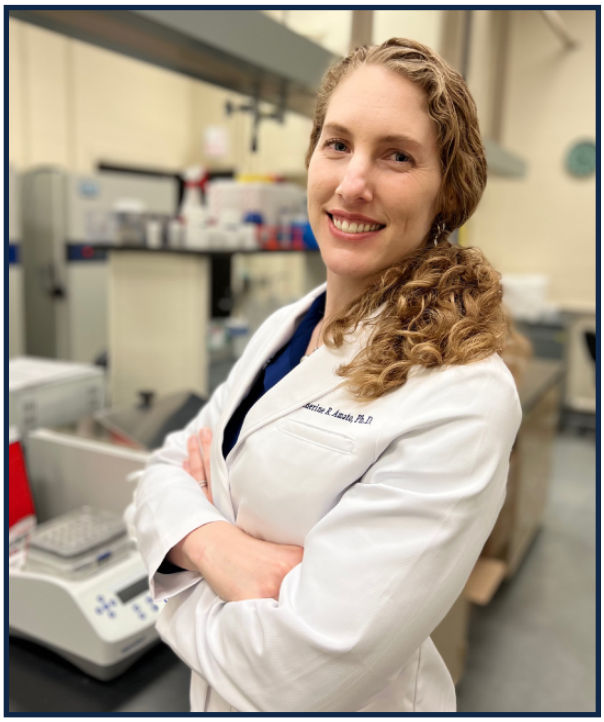
Keynote Speaker

Dr. Katherine Ryan Amato, PhD

Northwestern University
Associate Professor, Dept of Anthropology
Interdisciplinary Biological Sciences Graduate Program, Northwestern University

"Exploring the potential of the gut microbiota to buffer hosts against nutritional stress"



Dr. Amato is a biological anthropologist at Northwestern University studying the influence of gut microbes on host ecology and evolution. Her research examines how changes in the gut microbiota impact host nutrition, energetics, and health. She uses non-human primates as models for studying host-gut microbe interactions in selective environments and for providing comparative insight into the evolution of the human gut microbiota. Her main foci are understanding how the gut microbiome may buffer hosts during periods of nutritional stress and how the gut microbiome programs normal inter-specific differences in host metabolism. In this realm, she is also interested in global variation in the human gut microbiome and its implications for local human adaptation.

Dr. Amato obtained her A.B. in Biology from Dartmouth College and her Ph.D. in Ecology, Evolution and Conservation Biology from the University of Illinois Urbana-Champaign. She completed a postdoc at the University of Colorado Boulder. She is now an Associate Professor in the Department of Anthropology at Northwestern University. She is also affiliated with the Interdisciplinary Biological Sciences Graduate Program and sits on the Executive Committee of the Northwestern Institute on Complex Systems. Dr. Amato is the President of the Midwest Primate Interest Group, an Associate Editor at *Microbiome*, an Editorial Board member at *Folia Primatologica*, and a Fellow for the Canadian Institute of Advanced Research's 'Humans and the Microbiome' Program.

Keynote Address
4:00 p.m. – 5:00 p.m

Faculty Mini-Symposium:
Environmental Impacts on Nutrition and Health

Abstracts and Biographies

Impacts of psychological stress and nutrition on the microbiota-gut-brain axis

Dr. Brett Loman

Department of Animal Sciences, Division of Nutritional Sciences, University of Illinois Urbana-Champaign, Urbana, IL

ABSTRACT: Incidences of mental and gastrointestinal (GI) disorders are rising, while their etiologies and intersection remain poorly understood. The high co-occurrence of mental and GI disorders is unsurprising given the extensive bi-directional communication between the gut and the brain. Furthermore, similar microbial metabolite signatures are observed in individuals with conditions like depression and irritable bowel syndrome. Our research aims to understand the influence of environmental factors including stress and diet on enteric microbial metabolism, which can lead to the development of gut-brain disorders Gamma-aminobutyric acid (GABA) is a host- and microbe-derived neurotransmitter that modulates activities of the central and enteric nervous systems. Our experiments demonstrate that stress and microbial composition disrupt GABAergic signaling along the gut-brain axis. Using conventional and gnotobiotic mice, we demonstrate that stress alters expression of GABAergic genes in the intestine and brain, reduces intestinal GABA concentrations, and alters the microbiome structure. Both stress and microbial composition independently reduce concentrations of intestinal nutrients related to bacterial GABA synthesis and enhance measures of anxiety-like behavior. Conversely, we demonstrate that therapeutic interventions like dietary fiber intake enhance GABAergic signaling in the intestine. Compared to a fiber-free diet, mice consuming soluble, fermentable fiber have altered intestinal expression of GABAergic genes, enhanced luminal GABA concentration, and faster small intestinal transit. Finally, we demonstrate that intestinal Bifidobacterium isolates (diminished by stress and enhanced by fiber intake) are capable of synthesizing GABA, and that intestinal GABA exposure increases motility in the small intestine but decreases motility in the large intestine. Overall, while psychological stress disrupts GABAergic signaling that is associated with anxiety-like behavior and dysmotility, increasing fiber consumption represents a promising highly-translatable strategy to restore communication along the microbiota-gut-brain axis.

BIOGRAPHY: Dr. Brett Loman earned his PhD in Nutritional Sciences and RD at the University of Illinois Urbana-Champaign. He was a T32 Postdoctoral Fellow in the Comprehensive Training in Oral and Craniofacial Sciences program through the Ohio State University and Research Institute at Nationwide Children's Hospital in Columbus, OH. As a current Assistant Professor in the Department of Animal Sciences at the University of Illinois Urbana-Champaign, he is associated with the Division of Nutritional Sciences, Microbial Systems Initiative, and Personalized Nutrition Initiative. Dr. Loman's interdisciplinary research program strives to improve animal and human gastrointestinal and mental health. His team seeks to understand how environmental factors such as nutrition and stress alter communication between the resident microbiota, intestine, and brain, with the long-term goal of formulating dietary interventions that reduce gastrointestinal symptoms during functional gastrointestinal disorders, psychological stress, and cancer.

Disrupting inequitable food and health systems through food waste mitigation

Dr. Melissa Pflugh Prescott

Department of Nutrition, Case Western Reserve University School of Medicine, Cleveland, OH
Former: Department of Food Science and Human Nutrition, Division of Nutritional Sciences, University of Illinois Urbana-Champaign, Urbana, IL

ABSTRACT: Americans of all income levels waste food, averaging about 1 pound of food per American per day and resulting in 170 million metric tons of greenhouse gas emissions annually. At the same time, over 10% of Americans experienced food insecurity in 2021, and poor nutrition security has resulted in diet-related chronic disease being among the leading causes of deaths in the United States. The interrelated nature of food waste and nutrition security underscore the need for a systems approach to improving food and health environments. The objectives of this presentation are to 1) present evidence that food waste is a symptom of an inequitable food system, 2) identify drivers of health and food systems disparities, and 3) provide potential solutions to disrupt the current system. Disciplinary approaches, including behavioral economics, public health nutrition, and sociology, will be compared, as well as theory-based interventions to make the National School Lunch Program more equitable and households more resilient to rising food prices.

BIOGRAPHY: Melissa Pflugh Prescott is an Assistant Professor in the Department of Nutrition at Case Western Reserve University School of Medicine and is a former faculty member in FSHN and DNS at UIUC. Dr. Prescott completed her PhD in Public Health at New York University where she was a research assistant for the NYU Institute for Education and Social Policy. Dr. Prescott was awarded a Postdoctoral Fellowship from the USDA National Institute of Food and Agriculture, is a Fellow of the Academy of Nutrition and Dietetics, and completed her dietetic internship at the Bronx Veterans Affairs Medical Center. Dr. Prescott is a behavioral nutritionist and her research examines strategies to improve U.S. nutrition security and environmental stewardship, particularly among children and their families. Dr. Prescott's past experiences include managing a school-based obesity prevention program across seven elementary schools located in Harlem and Washington Heights, NYC and serving as a nutrition consultant to many public health organizations, including the NYC Coalition for the Homeless and United Way of NYC. Her research is funded by the United States Department of Agriculture and the National Science Foundation.

Exercise and fermented foods to promote immune-modifying microbial metabolites

Dr. Jacob M. Allen

Department of Kinesiology and Community Health, Division of Nutritional Sciences, University of Illinois Urbana-Champaign, Urbana, IL

ABSTRACT: Exercise modifies the gut microbiota and contributes significantly to metabolic and immune health. However, to what extent exercise-induce changes in the microbiota directly influences human physiology has not yet been thoroughly characterized. The microbiota produces and modifies a range of bioactive metabolites that may underly some of the benefits of exercise. Our laboratory has recently uncovered coordinated shifts in microbial metabolites in both the gut and serum in response to an exercise training intervention in humans. We have specifically focused on a shared class microbial-derived aromatic amino acid (ArAA) metabolites produced by the microbiota with potential immunomodulatory properties. Recently, we have begun to explore the potential that these metabolites impact the physiology of monocytes, vital innate immune cells that are critical mediators of early inflammatory responses to infection, wound repair, and exercise adaptation. Our preliminary analysis has revealed ArAA metabolites that tune both the metabolic and immune capacity of monocytes, highlighting the exciting possibility that exercise-induced shifts in monocyte function are supported by microbial metabolites. Our work has also uncovered a select group of fermented foods containing high levels of the same microbial-derived ArAA metabolites. These data led us to hypothesize that there is a common class of microbial products modifiable by exercise and fermented foods with immune- modifying potential. However, how fermented food and exercise interact to modify microbial metabolites and immune health has never been explored. To fill this gap in knowledge our lab is 1) Further testing the immune modulating potential of microbial ArAA metabolites and 2) Identifying exercise prescriptions and fermented food diets that most effectively increase immune-modifying microbial metabolites.

BIOGRAPHY: Dr. Allen received a master's degree from the University of North Carolina at Chapel Hill in 2013 and a PhD from the University of Illinois Urbana-Champaign in 2017. During his PhD, he studied the role of exercise in modifying the gut microbiota and its metabolites. After his PhD, Dr. Allen completed a postdoc at Nationwide Children's Hospital where he worked to understand how microbes interact with the immune system to predispose enteric infection and bowel disease. In August of 2020, Dr. Allen began a tenure-track Assistant Professor position at the University of Illinois hired under a campus wide Microbial Systems Initiative (MSI). His research program is focused on understanding how specific environmental interventions and conditions-- 1. Nutrition 2. Exercise and 3. Stress—influence gut microbial communities and metabolite production during both homeostatic and pathological disease states. Dr. Allen's lab utilizes a range of *in vitro* systems (organoids, bacterial culture), pre-clinical animal models, and clinical studies to test hypotheses into how gut microbes interact with the host immune system. In the nutrition space, his lab is currently focused on understanding how metabolites found in fermented foods (postbiotics) can impact innate immune function of humans.

Graduate Student Oral Session Abstracts

Oral Session 1: Nutrient Metabolism & Dietary Patterns

Lycopene bioaccumulation in transgenic mice lacking one or both carotenoid cleaving enzymes

Madelyn J. Bradley[1], M. Black[2], J. R. Arballo[1], J. Amengual[1,2], J. W. Erdman Jr.[1,2]

[1] Division of Nutritional Sciences, University of Illinois Urbana Champaign, Urbana, IL
[2] Food Science and Human Nutrition, University of Illinois Urbana Champaign, Urbana, IL

INTRODUCTION: β-carotene oxygenase 1 (BCO1) and β-carotene oxygenase 2 (BCO2) are responsible for the cleavage of carotenoids in mammals. The goal of this study was to establish the relative contribution of BCO1 and BCO2 to lycopene bioaccumulation in mice.

METHODS: We utilized male and female wild-type (WT), *Bco1*$^{-/-}$, *Bco2*$^{-/-}$, and *Bco1*$^{-/-}$ *Bco2*$^{-/-}$ double knockout (DKO) mice. All genotypes were gavaged daily with 1 mg of lycopene resuspended in cottonseed oil as a vehicle, or a control group (WT) with only vehicle, for two weeks. One day after the last dosing, mice were sacrificed and tissues were collected for HPLC and RT-PCR analyses. Hepatic mitochondria were isolated to determine lycopene content and isomer characterization.

RESULTS: Of the ten tissues measured, the liver contained 94-98% of the lycopene content across genotypes. Ablation of BCO1 resulted in halving the hepatic and total body lycopene compared to other genotypes (*Bco1*$^{-/-}$ verses *Bco2*$^{-/-}$ ($p < 0.0001$), DKO mice ($p < 0.001$), or WT (ns)) . However, in the serum the *Bco2*$^{-/-}$ mice accumulated less lycopene than the *Bco1*$^{-/-}$ ($p < 0.01$) and the DKO mice ($p<0.0001$), a trend also observed in most extrahepatic tissues. Ablation of one cleavage enzyme failed to upregulate the expression of the other enzyme. Mitochondria, as compared to the whole liver, were enriched 3 – 5 fold in lycopene (μg/mg protein) ($p<0.05$) in all genotypes and gender, showing comparable isomer content among genotypes. Vitamin A deficient mice (VAD) accumulated more lycopene than Vitamin A sufficient (VAS) mice in the liver. In comparison to control VAD mice, VAD mice dosed with lycopene upregulated ISX (intestine-specific homeobox) jejunal expression.

CONCLUSION: These findings suggest that BCO2 is the primary lycopene cleavage enzyme. The lycopene concentration was enriched in the mitochondria of hepatocytes independently of the presence of BCO2. Lycopene may act as retinoic acid to interact with the ISX/SRB1 axis.

Dietary patterns and carotenoid intake: Comparisons of MIND, Mediterranean, DASH, and Healthy Eating Index

Tori A. Holthaus[1], S. A. Keye[2], S. Verma[2], C. N. Cannavale[2], H. D. Holscher[1,2,3], N. A. Khan[1,2,4,5]

[1] Division of Nutritional Sciences, University of Illinois Urbana-Champaign, Urbana, IL
[2] Kinesiology and Community Health, University of Illinois Urbana-Champaign, Urbana, IL
[3] Department of Food Science and Human Nutrition, University of Illinois Urbana-Champaign, Urbana, IL
[4] Neuroscience Program, University of Illinois Urbana-Champaign, Urbana, IL
[5] Beckman Institute of Advanced Science and Technology, Urbana, IL

INTRODUCTION: Carotenoids have been linked to numerous health benefits including greater cognitive function. Likewise, adherence to the Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet is suggested to reduce cognitive decline. The degree to which the MIND diet index emphasizes carotenoids relative to other healthful dietary indices is not well defined. Thus, we investigated the relationship between degree of adherence to the MIND diet index and carotenoid intake relative to the Mediterranean, Dietary Approaches to Stop Hypertension (DASH), and Healthy Eating Index (HEI-2015).

METHODS: Adults aged 19-82 years (N = 392) completed the Dietary History Questionnaire (DHQ) food frequency questionnaire to assess carotenoid intake and adherence to each diet index. Linear regressions were used to assess the variance in carotenoid intake explained by each diet index following adjustment for energy, age, sex, income, and ethnicity. The predictive ability of each diet index for carotenoids was then compared using Meng's z-test.

RESULTS: Following adjustment, all diet indices were positively associated with dietary lutein + zeaxanthin, β-carotene, α-carotene, and β-cryptoxanthin (all β's ≥0.36, P's <0.01). Dietary lycopene was associated with MIND (β=0.16, P <0.01) and Mediterranean (β =0.18, P <0.01). Effect size comparisons revealed that MIND diet adherence predicted greater variance in dietary lutein + zeaxanthin (β =0.64, P <0.01) and β-carotene (β=0.63, P <0.01) relative to the Mediterranean, DASH, and HEI-2015 (all Z's ≥2.6, P's <0.01). The MIND index explained greater variance in α-carotene (β=0.48, P<0.01) and β-cryptoxanthin (β =0.38, P <0.01) relative to the HEI-2015 (all Z's ≥3.6, P's <0.01).

CONCLUSION: MIND diet adherence was disproportionately related to carotenoid intake, indicating the MIND index places greater emphasis on carotenoids relative to other diet indices. This was more evident in comparisons between the MIND and HEI-2015 indices. Future work is needed to test the role of dietary carotenoids in explaining the neurocognitive health effects of the MIND diet.

Influence of α-tocopherol depletion in murine brain affects lipopolysaccharide-induced muscle weakness

Megumi Hashida[1], K. M. Ranard[2], A. J. Steelman[1,3], J. W. Erdman Jr.[1,4]

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[2] Department of Pediatrics, University of Colorado Anschutz Medical Campus, Aurora, CO
[3] Department of Animal Sciences, University of Illinois Urbana-Champaign
[4] Department of Food Science and Human Nutrition, University of Illinois Urbana-Champaign

INTRODUCTION: Prior murine work from our laboratory showed that lipopolysaccharide (LPS) exposure caused systemic immune and neuroinflammatory responses, evidenced by decreased circulating immune cells and an increased interleukin-6 (IL-6) production in the brain. However, when fed vitamin E deficient diets, there were no significant additional effects seen when comparing α-tocopherol transfer protein null (*Ttpa*$^{-/-}$) to wild-type (*Ttpa*$^{+/+}$) mice on measured markers of the inflammatory response. There is a need to identify functional abnormalities related to αT deficiency and link those changes to neurobehavioral deficits that accompany αT deficiency in the brain in young adult *Ttpa*$^{-/-}$ mice with low vitamin E status. Our objective was to investigate if αT depletion, followed by exposure to LPS, altered an acute inflammatory response and muscle strength in *Ttpa*$^{-/-}$ and *Ttpa*$^{+/+}$ mice.

METHODS: After weaning (3 weeks of age), male *Ttpa*$^{+/+}$ and *Ttpa*$^{-/-}$ littermates (n=12-14/genotype) were fed αT deficient diet *ad libitum* for 9 weeks. The *Ttpa*$^{-/-}$ mouse model is useful to facilitate α-tocopherol (αT) depletion through genetic and dietary approaches. At 12 weeks of age, mice were injected with LPS (10 µg/mouse) or saline (control) intraperitoneally and sacrificed 4 hours later. αT concentrations in diet and tissues were confirmed via HPLC. Cerebellar and serum IL-6 levels, markers of acute inflammatory response, were measured via ELISA. Hippocampal *Il-6* gene expression was measured via RT-qPCR. Mouse grip strength was measured via a grip strength meter apparatus a day before termination and 3.5 hours after LPS injection.

RESULTS: αT concentrations in the liver, heart, and adipose tissue of *Ttpa*$^{+/+}$ mice were significantly higher than from *Ttpa*$^{-/-}$ mice ($P < 0.0001$). Brain and serum αT were not detected in *Ttpa*$^{-/-}$ mice. Hippocampal *Il-6* gene expression and cerebellar and serum IL-6 protein levels were enhanced in 10 µg LPS groups ($P < 0.05$), confirming acute neuro- and systemic-inflammatory response. Although muscle strength in *Ttpa*$^{+/+}$ mice was decreased by LPS ($P = 0.07$), only *Ttpa*$^{-/-}$ mice showed a statistically significant decrease in muscle strength following LPS exposure ($P < 0.0001$).

CONCLUSION: LPS administration resulted in a significant increase in acute inflammatory response and a decline in muscle strength, especially in *Ttpa*$^{-/-}$ mice. αT depletion may exacerbate neuromuscular function.

NAD+ metabolism generates a metabolic vulnerability in endocrine-resistant metastatic breast tumors

Ayca Nazli Mogol[1], Q. Zuo[2], J.Y. Yoo[2], A.Z. Kaminsky[2], O.B. Imir[1], Y. Landesman[3], C.J. Walker[3], Z. Madak-Erdogan[1,2,4,5,6]

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2 Department of Food Science and Human Nutrition, University of Illinois Urbana-Champaign, IL
3 Karyopharm Therapeutics, MA
4 Department of Biomedical and Translational Sciences, Carle Illinois College of Medicine, IL
5 Carl R. Woese Institute for Genomic Biology, University of Illinois Urbana-Champaign, IL
6 Cancer Center at Illinois, IL

INTRODUCTION: Approximately 70% of human breast cancers express estrogen receptor-α (ERα), providing a potential target for endocrine therapy. However, 30%–40% of patients with ER$^+$ breast cancer still experience recurrence and metastasis, with a 5-year relative overall survival rate of 24%.

METHODS: We used xenograft mouse model and cell culture assays to inhibit NAD$^+$ salvage pathway. Dose response assays, NAD/NADH Assay, RNA sequencing and metabolomics assays were performed to further investigate this mechanism.

RESULTS: In this study, we identified NAMPT, an important enzyme in nicotinamide adenine dinucleotide (NAD$^+$) metabolism, to be increased in metastatic breast cancer (MBC) cells treated with Fulv. We tested whether blockade of NAD$^+$ production via inhibition of nicotinamide phosphoribosyltransferase (NAMPT) synergizes with standard-of-care therapies for ER$^+$ metastatic breast cancer in vitro and in vivo. A synergistic effect was not observed when KPT-9274 was combined with palbociclib or tamoxifen or when Fulv was combined with other metabolic inhibitors.

We show that NAMPT inhibitor KPT-9274 and fulvestrant (Fulv) works synergistically to reduce metastatic tumor burden. RNA-sequencing analysis showed that NAMPT inhibition improved antiestrogenic activity of Fulv, and metabolomics analysis showed that NAMPT inhibition reduced the abundance of metabolites associated with several key tumor metabolic pathways.

CONCLUSION: Targeting metabolic adaptations in endocrine-resistant metastatic breast cancer is a novel strategy, and alternative approaches aimed at improving the therapeutic response of metastatic ER+ tumors are needed. Our findings uncover the role of ERα–NAMPT cross-talk in metastatic breast cancer and the utility of NAMPT inhibition and antiestrogen combination therapy in reducing tumor burden and metastasis, potentially leading to new avenues of metastatic breast cancer treatment.

Oral Session 2: Community Nutrition

Understanding the nutritional challenges of food insecure adults with a history of cancer

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INTRODUCTION: The economic burden associated with cancer care in the USA may intensify the risk of food insecurity among adults with a history of cancer. Diet quality and nutritional status play a critical role in determining cancer outcomes, but adults diagnosed with cancer are at risk of suboptimal nutrition due to the intensity and burden of the disease and associated treatments. Being food insecure may further exacerbate the nutritional risk of individuals with cancer. Therefore, the purpose of this study was to examine the associations of food insecurity with diet quality, malnutrition risk, and participation in food assistance programs in adults with a recent cancer diagnosis.

METHODS: This was a cross-sectional pilot study of a hospital-based sample of 220 adults diagnosed with cancer between 2018-2021 in the USA. Food insecurity was assessed using the United States Department of Agriculture Six-item Short Form Food Security Survey Model. Usual intake of total fruits and vegetables, percent kilocalories from fat, and dietary fiber were estimated using the National Cancer Institute Multifactor Screener. Malnutrition risk was measured using the Malnutrition Screening Tool. Two additional survey questions asked participants about their use of Supplemental Nutrition Assistance Program (SNAP) benefits and food banks or pantries. Associations between food security status and variables of interest were examined using chi-square or fisher exact tests.

RESULTS: The prevalence of food insecurity in the study sample was 10%. Food insecure participants were more likely to consume <5 servings of fruits and vegetables, ≥30% of their total kilocalories from fat, ≤15 grams of fiber per day, and be at risk for malnutrition compared to food secure participants. Food insecure participants also reported utilizing SNAP benefits and food assistance from a food bank/pantry at a higher rate compared to food secure participants.

CONCLUSION: Food insecure adults with a recent history of cancer may be more likely to experience nutritional deficits which may, in turn, lead to worse cancer outcomes. and use food assistance programs than those who are food secure. Future work should focus on building connections between cancer patients and charitable food assistance programs.

Feasibility and acceptability of an online weight loss program for obesity in rural Illinois counties

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INTRODUCTION: The prevalence of obesity continues to increase worldwide with disproportionately higher rates in rural populations. The factors contributing to higher rates of obesity in rural areas may include inadequate access to medical care, housing, income, and ethnic segregation. The EMPOWER program is an online weight loss program previously established online weight loss program, EMPOWER, that emphasizes protein and fiber with potential to reach populations in any geographic vicinity with internet. We hypothesized that 1) an online weight loss program was feasible and acceptable in rural populations, and 2) an average weight loss of 5% with most of the loss in fat mass.

METHODS: Sixteen adults with a body mass index (BMI) ≥ 25 kg/m^2 living in rural counties (population < 25,000) were recruited through collaboration with University of Illinois Extension to participate in feasibility trial of a previously established online weight loss program that emphasizes protein and fiber. The program consists of 17 online education sessions administered via an online platform (eText), teleconference nutrition and lifestyle coaching, and diet and weight monitoring via the web application MealPlot, a tool that allows participants to visualize progress toward meeting daily dietary targets for protein and fiber to total kcal ratios.

Anthropometric measurements, 24-hour records, and nutrition surveys were collected at baseline and 6- months. A food frequency questionnaire was completed at baseline. Weight was measured daily via Wi-Fi scale. Feasibility and acceptability of the program was measured at 3-months by qualitative and quantitative interview.

RESULTS: Participant retention was 62.5% (10/16) at 3-months. Level of satisfaction (5-point Likert) of all components of the program on average was 4.2 (n=10). The visualization tool, MealPlot, was significantly less satisfactory than other components of the program at 2.7 (n=10). At 6-months, intention to treat analysis (n=16), mean weight and fat mass loss was statistically significant ($p = 0.0029$) at -4.1 ± 6.1% and -7.7 ±13.2% respectively. Skeletal muscle mass was preserved with an average of -0.37 ± 0.96 kg. Although protein and fiber density changes were insignificant, both increased 0.35 ± 1.33 g/100 kcal and 0.02 ± 0.49 g/100 kcal, respectively.

CONCLUSION: An online weight loss program in rural populations resulted in significant weight loss. The majority of weight loss program components were ranked satisfactory. Attrition suggests that barriers to participation in rural communities were not yet sufficiently addressed. Future studies will address these barriers and establish liaisons within the rural community who act as community champions. The MealPlot web application will also be improved for increased satisfaction and usability.

County-level food insecurity and COVID-19 mortality in the United States: a spatial analysis

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INTRODUCTION: Food insecurity (FI) is a public health issue of concern and a sociodemographic factor that drives health inequities and affected approximately 13.5% of U.S. households in 2020. The COVID-19 pandemic exacerbated food insecurity in the United States, owing primarily to job and income loss. Previous work demonstrated the relationship between pre-pandemic estimates of food insecurity and COVID-19 infection during the early phases of the pandemic. However, we are unaware of any studies evaluating the relationship of pre-pandemic estimates of food insecurity to COVID-19 mortality.

METHODS: To examine associations between food insecurity and COVID-19 mortality in the United States, we employed 2019 estimates of county-level food insecurity from Feeding America's Map the Meal Gap and COVID-19 death counts from two sources between March 25, 2020, and December 25, 2021. Cumulative crude COVID-19 death counts were obtained from the Johns Hopkins University (JHU) Centers for Civic Impact for the Coronavirus Resource Center (CRC) and age-standardized mortality counts were computed using COVID-19 mortality data from the Centers for Disease Control and Prevention (CDC). We fit spatial negative binomial regression models that accounted for county centroid coordinates, using a Bayesian framework, to estimate posterior means and 95% credible intervals (CrI). These models adjusted for several county-level demographic, socioeconomic, and geographic confounders. Models using the JHU CRC crude mortality counts included county median age as an additional covariate.

RESULTS: The culmination of the analytic period included 3,131 counties reporting 774,231 COVID-19 deaths (crude deaths) with an observed mean mortality rate of 297.39 COVID-19 deaths per 100,000 inhabitants. Our analysis showed evidence of substantial underreporting of COVID-19 mortality counts to the CDC, and this was accentuated in non-metropolitan counties relative to the crude mortality counts reported to the CRC. Using the JHU CRC data, we found that a 4% increase in county-level food insecurity prevalence was associated with a 1.37-fold increased risk of county-level COVID-19 mortality (95% CrI: 1.34-1.41). When using the CDC's age-standardized counts, we found a weak inverse association between county-level food insecurity and COVID-19 mortality rate (MRR: 0.91, 95% CrI: 0.87-0.97).

CONCLUSION: Our findings highlight the implications of county-level food insecurity on COVID-19 mortality. Another theme that emerged from our analysis was how data quality and source affected the parameter estimates from the models constructed. Future analyses using data measured at the individual level are warranted to substantiate the associations described here.

Mixed methods study investigating the consumer acceptance and implementation of spicy vegetables into school lunch

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INTRODUCTION: By late adolescence, the average vegetable consumption is about half of the recommended intake range, and vegetables are the most wasted school meal component during lunch time. While vegetable consumption remains low, spicy food has grown in popularity. Globally, 1 in 4 people eat chilies daily. To increase vegetable palatability and cater to various cultural taste preferences, chili pepper can serve as a healthy flavor enhancer, but its implementation into school lunch has not been studied. This convergent parallel mixed method study has three objectives: To identify what degree of capsaicin pungency is the most liked among adolescents, to estimate the appropriateness and acceptability of spicy broccoli in school lunch, and to identify strategies to promote spicy vegetables in school lunch.

METHODS: 100 participants in central Illinois, ages 11-17 sampled 4 spice levels of steamed broccoli containing 0, 0.9, 2.0, and 4.0 grams of cayenne and red pepper blend. Participants also completed a survey, and a short audio-recorded interview. Participants rated their consumer acceptance on a 9-point hedonic scale for each broccoli sample. The survey assessed race, language, age, and gender, as well as validated chili liking, chili consumption patterns, spicy broccoli appropriateness, and spicy broccoli acceptability items on a 5 point agree/ disagree scale. The interview assessed perspectives on spicy vegetables and their implementation into school lunch.

RESULTS: Consumer acceptance ratings did not significantly differ between the four samples, however sample 3 had the highest overall mean rating (6.69 + 2.0). Participants rated the acceptability (4.0 + 1.0) of spicy broccoli in school lunch higher than the appropriateness (3.6 + 1.0). Thematic analysis results suggest most participants are in favor of incorporating spicy vegetables into school lunch and believe there is potential for spices to increase vegetable intake. Suggested promotion strategies included posters, announcements, take home menus, and parent emails.

CONCLUSION: Overall, spicy vegetables were liked among adolescents, but more research is needed to assess if spicy vegetables will successfully increase vegetable consumption and reduce waste during school lunch.

Graduate Student Poster Session Abstracts

Poster Section: Dietary Intervention

MIND Diet Pattern is selectively associated with attentional control in preadolescent children

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INTRODUCTION: Previous work has shown that specific dietary patterns contribute to cognitive function across the lifespan. The Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) – a dietary pattern that emphasizes specific foods associated with neuroprotective benefits – is related to reduced risk for cognitive decline in older adults. However, no previous study has examined the relationship between MIND diet pattern adherence and cognitive function in childhood. The purpose of this study was to investigate the relationship between the adherence of different dietary indices [i.e., Health Eating Index-2015 (HEI-2015) vs. MIND] and attentional inhibition in school-aged children.

METHODS: Children (n=85, 44 female) aged 9.6 ± 1.9 years participated in a cross-sectional study. HEI-2015 and MIND diet scores were derived from 7-day diet records. Participants completed a modified Eriksen flanker task to assess attentional inhibition. Step wise regressions were conducted to assess the variance in attentional inhibition explained by each diet pattern index (i.e., MIND vs. HEI-2015) following adjustment for age, sex, total caloric intake, and household income.

RESULTS: Average HEI-2015 score was 46.41 ± 10.12 out of 100 and average MIND diet score was 4.48 ± 1.77 out of a 14. MIND score was positively related to congruent accuracy (ΔR^2= 0.06, β=0.25, *P*= 0.02) and incongruent accuracy (ΔR^2= 0.06, β=0.24, *P*=0.02) on the flanker task. However, there was no relationship between HEI-2015 and congruent accuracy (ΔR^2=0.00, β=0.05, *P*=0.66) or incongruent accuracy (ΔR^2=0.00, β =0.01, *P*=0.89).

CONCLUSION: In preadolescent children, participants with higher adherence to the MIND diet exhibited greater accuracy on a task assessing attentional inhibition. These results indicate that the potential beneficial influence of the MIND diet pattern is evident in childhood. Future MIND diet interventions are needed to inform dietary recommendations for cognitive function and brain health in children.

Effects of lutein supplementation on carotenoid status and cognition among persons with multiple sclerosis

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INTRODUCTION: Multiple sclerosis (MS) is an immune-mediated, demyelinating disease of the central nervous system that is often accompanied by visual and cognitive impairment. Lutein supplementation has been shown to improve macular pigment optical density (MPOD) and cognitive function among healthy adults. However, no previous research has examined the effects of lutein supplementation in persons with MS. Thus, we investigated the effects of 4-month lutein supplementation on measures of carotenoid status and cognitive function in persons with MS.

METHODS: Participants (*N*=21) were randomly assigned to the placebo (safflower oil without lutein, n = 9) or treatment (20 mg/day lutein with safflower oil, n = 12) group for a 4-month period. Carotenoid status was assessed in the macula (MPOD), skin, and serum using heterochromatic flicker photometry, reflection spectroscopy, and high-performance liquid chromatography, respectively. Cognitive function was assessed via the Eriksen flanker task, the i-Position spatial memory task, and the symbol digit modalities test.

RESULTS: There was a significant group by time interaction whereby the treatment group exhibited a significant increase in MPOD ($\Delta\bar{x}$ = 0.24, p = 0.006), skin carotenoids ($\Delta\bar{x}$ = 141.97, p < 0.001), and serum lutein ($\Delta\bar{x}$ = 0.51, p < 0.001). There was no significant interaction effect for any of the cognitive outcomes. However, there was a significant positive association between change in MPOD with change in flanker incongruent accuracy (rho = 0.51, p = 0.044) and change in object location binding in the spatial memory task (rho = 0.55, p = 0.033) only among the treatment group participants. No significant associations were observed for changes in cognitive function and changes in serum lutein or skin carotenoids.

CONCLUSION: These findings are the first to demonstrate improved carotenoid status in the macula, skin, and serum among persons with MS following lutein supplementation. Additionally, change in macular carotenoids, but not skin carotenoids or serum lutein, was related to improved attention and memory, providing support for targeting neural carotenoids for cognitive benefits in persons with MS. Larger intervention trials are necessary to test lutein supplementation effects on cognitive function among persons with MS.

Effectiveness of short-term, home-delivered, low sodium meals to sustain long-term changes in dietary behavior in hemodialysis patients

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INTRODUCTION: Dietary sodium intake (DSI) influences chronic volume overload (VO) and interdialytic weight gain (IDWG), which are variables associated with poor cardiovascular outcomes. Reducing DSI through dietary education is typically ineffective; however, a 4-week pilot study in our lab found that short-term low-sodium home-delivered meals provision could reduce DSI resulting in lower IDWG and ultrafiltration rates (UF) in hemodialysis (HD) patients. It is unknown if reductions in DSI from short-term meal feeding can be sustained after meal provision ends. The purpose of this study is to determine if short-term feeding of low-sodium meals can lower IDWG in HD patients and maintain the effect 3 months after the intervention through enhanced low-sodium dietary education. As secondary outcomes, systolic arterial pressure and volume overload were assessed.

METHODS: HD patients were randomized to one of two groups: 1) a low sodium diet (LS); or 2) usual care/wait-list control (CON). LS received 2 low sodium meals/day (<600mg Na/meal) during month 1, 1 LS meal/day in month 2, as well as enhanced LS education for 5 months. CON received usual care for 5 months before receiving the LS meals in months 6 and 7.

RESULTS: LS had a reduction in IDWG after 1 month, but this was not sustained with continued feeding in month 2 or out to month 5 with continued low sodium education. BP, VO, and other secondary outcomes also were not improved in the LS group at any time point.

CONCLUSION: Providing LS meals may provide short-term reductions in IDWG, but this benefit may not be sustained with continued feeding. More comprehensive approaches to reducing sodium intake may be necessary to produce significant benefits in this population. This may include engaging HD patients' families/caregivers and the clinic staff to help support their nutrition behavior change. Future studies could incorporate culinary medicine, such as cooking classes, gardening, and virtual reality grocery shopping for an engaged nutrition education approach.

Persea americana for total health 2 (PATH-2): Effects of avocado intake on gastrointestinal and cognitive health

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INTRODUCTION: Dietary interventions can modify the intestinal microbiome and metabolome, which is linked to health benefits. Previously, we demonstrated that avocado consumption increased *Faecalibacterium* spp. and short chain fatty acid (SCFA) concentrations, decreased abdominal adiposity, and improved attention abilities in adults with overweight and obesity. Therefore, the proposed research aims to establish that avocado consumption positively affects the gastrointestinal microbiome and improves cognitive function. Our primary hypothesis is that consumption of an average American diet with fresh Hass avocado (AV) will increase *Faecalibacterium* spp. and SCFA concentrations and reduce secondary bile acid formation compared to the control groups (average American diet; AA) and high oleic oils + fiber group (OF) group. Our secondary hypotheses are that consuming an avocado daily will improve cognitive function relative to the control groups. The proposed work will also explore the potential mediation of benefits of avocado consumption on intestinal health and cognitive benefits via the gut microbiome.

METHODS: This study will use a randomized-controlled crossover complete feeding design with 3 dietary periods (AA, AV, and OF). Each 4-week dietary periods will be separated by a 2-week washout. Weight stable adults (25-74 y) without diabetes with overweight or obesity (BMI > 25 kg/m^2) will be eligible for enrollment. Fecal, blood and urine samples will be collected during week 4 of each condition. Fecal microbiome and metabolites will be assessed using metagenomic sequencing analysis and gas liquid chromatography, respectively. Circulating and fecal inflammatory markers will be quantified using ELISA. Gut permeability will be assessed by quantifying the urinary excretion of orally ingested sugar substitutes. Neuropsychological performance will be evaluated by measuring neuroelectric function during cognitive tasks.

CONCLUSION: We expect that avocado consumption will beneficially affect the intestinal microbiome and metabolome, thereby contributing to decreased systemic and intestinal inflammation and improve neuropsychological performance.

Preliminary effects of a carbohydrate-restricted, high-fat diet in head and neck cancer patients undergoing radiotherapy: A pilot randomized controlled trial

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INTRODUCTION: Research demonstrated the ketogenic diet benefits in cancer patients, but the extremity of this diet makes adherence difficult. Our previous observational studies showed that a self-reported diet of <50% carbohydrates and ≥23% unsaturated-fats is associated with more positive outcomes in head and neck cancer (HNC) patients. The purpose of this single-blinded randomized controlled trial (RCT) is to test the preliminary effects of implementing a carbohydrate-restricted, high-fat (CRHF) diet in HNC patients undergoing definitive radiotherapy (RT).

METHODS: From January-2020 to August-2021, thirteen newly-diagnosed HNC patients that planned to receive RT at Augusta Victoria Hospital in Palestine, were enrolled and randomized to one of two arms—a CRHF arm (N=6); received ~30% carbohydrates, ~45% fats, and ~25% proteins or a standard diet (SD) arm (N=7). Three meals per day were provided to all participants for 2-weeks before RT and 6-7 weeks during RT, with adequate calories estimated for weight maintenance. Per-protocol analysis was performed, including (N=4) participants in the CRHF arm and (N=5) participants in the SD arm. The CRHF and the SD arms were compared in terms of nutritional status, using patient-generated subjective global assessment (PG-SGA) tool; body composition, using bioelectrical impedance (BIA); symptom burden, using the Memorial Symptom Assessment Survey (MSAS); health-related quality of life (HRQOL), using EORTC QLQ-H&N43; and survival.Outcomes were assessed at T0 (baseline), T1 (2-weeks), and T2 (1.5-2 months post-RT).

CONCLUSIONS: Both arms showed improvements in the PG-SGA score at T1 and overall, the CRHF arm had non-significant, slightly lower PG-SGA score than the SD arm. At T2, 56% of participants were well-nourished and 44% were moderately malnourished. Both arms gained skeletal muscle mass. The CRHF arm lost more, non-significant fat mass and phase angle degree than the SD arm. None of the participants developed sarcopenia. Overall, the most common symptoms were pain, dry mouth, and feeling nervous, and the most common HRQOL symptoms were problems with teeth, anxiety, and opening mouth. The CRHF had lower MSAS and HRQOL scores than the SD arm. There was one recurrence in the CRHF arm and one mortality in the SD arm.

CONCLUSION: Overall, this pilot RCT shows better outcomes than the general HNC population. Both arms gained skeletal muscle mass by the end of the study. The CRHF arm had better PG-SGA, MSAS, and HR-QOL scores than the SD arm. Overall, the SD arm had better improvements in body composition. These preliminary results can be used to develop a larger sample size, with more statistical power to test the efficacy of implementing a CRHF diet on HNC outcomes.

Poster Section: Dietary Intervention Models I

Determination of honey varietals' impact on *Bifidobacterium animalis* ssp *lactis* survivability in commercial yogurt through simulated *in vitro* digestion

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INTRODUCTION: Consumption of yogurt containing the probiotic strain *Bifidobacterium animalis lactis* DN-173 010/CNCM I-2494 (*B. animalis*) has been shown to improve digestive health and improve quality of life in adults. To optimize the benefits of this probiotic, we aimed to test our hypothesis that adding honey to commercial yogurt would increase the survivability of *B. animalis* under simulated gastrointestinal tract digestion conditions.

METHODS: Yogurt samples were subjected to *in vitro* simulated oral, gastric, and intestinal phase digestion using simulated salivary, gastric, and intestinal fluids, respectively. At four time points—pre-digestion (baseline), and then after each phase of digestion (i.e., oral, gastric, and intestinal)—probiotic cells were enumerated first by spread plating on MRS agar and incubated for 5 h at 37℃ under anaerobic conditions to allow *B. animalis* cells to recover. Then, plates were overlaid with MRS supplemented with lithium chloride and sodium propionate and incubated at 37℃ for an additional 67 h prior to quantification of the probiotic colony forming units (CFU).

RESULTS: Significantly higher *B. animalis* survivability was observed in yogurt with clover honey after exposure to simulated intestinal fluids (~3.5 Log CFU/g reduction) compared to undiluted yogurt, sucrose added yogurt, and water added yogurt (~5.5 Log CFU/g reduction, $P < 0.05$). Phase 2 demonstrated significant *B. animalis* survivability after exposure to simulated intestinal fluids at new concentrations of clover honey, 14 and 10% w/w (~ 4.6 Log CFU/g reduction, $P < 0.05$) and verified the previously observed significance using 20% w/w (~3.9 Log CFU/g reduction, $P < 0.05$) compared to undiluted control (~5.6 Log CFU/g reduction).

CONCLUSION: These results demonstrated that clover honey increased *B. animalis* survivability in commercial yogurt during *in vitro* digestion.

Dietary β-carotene accelerates atherosclerotic resolution by promoting T reg expansion in the atherosclerosis lesion

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INTRODUCTION: Forkhead box P3 (*FoxP3*)$^{+}$ regulatory T cells (Tregs) mediate atherosclerosis resolution and regression by promoting an anti-inflammatory environment in the lesion. FoxP3 is upregulated by retinoic acid, which is the transcriptionally active form of vitamin A, but whether vitamin A status influences atherosclerosis resolution remains unexplored. We hypothesized that β-carotene, the main vitamin A precursor in human diet, accelerates atherosclerosis resolution by upregulating FoxP3 expression to promote Treg expansion.

METHODS: We induced atherosclerosis in *FoxP3*^{EGFP} mice fed Western diet control (without β-carotene) and injected with an anti-sense oligonucleotide targeting the LDLR (ASO-LDLR) for 16 weeks. After this period, we harvested a subset of mice as Baseline, while remaining mice underwent atherosclerosis resolution upon the interruption of ASO-LDLR infusions to recover LDLR expression and normalize plasma lipids for a period of three weeks. During these three weeks, mice either continued on the same Western diet (WD-Control) or were switched to Western diet supplemented with 50 mg of β-carotene/kg of diet (WD-β-carotene). To examine the implication of Tregs in our model, we injected a subset of WD-β-carotene with anti-CD25 (WD-β-carotene + anti-CD25), while WD-Control and a second subset of WD- β-carotene mice were injected with IgG control. We estimated atherosclerosis resolution by quantifying macrophage and collagen contents in the lesions present at the level of the aortic root. WD-β-carotene-fed mice injected with IgG showed a reduction in macrophage content accompanied by an increase in collagen in comparison to the all the remaining groups, while WD-β- carotene + anti-CD25 lesions resembled those present in WD-Control mice. We examined Treg the relative number of $CD25^{+}FoxP3^{+}$ and $CD25^{-}FoxP3^{+}$ cells in the spleen, blood, and the lesion. $CD25^{+}FoxP3^{+}$ numbers decreased in response to anti-CD25 infusions in all the tissues examined.

RESULTS: Dietary β-carotene, independently of the injection with either IgG or anti-CD25, favored an increase of 269% and 653% in $CD25^{-}FoxP3^{+}$ Tregs in comparison to the Baseline of WD-Control groups, respectively. These effects occurred independently of changes in plasma lipids or changes in body weight.

CONCLUSION: Our data show that β-carotene supplementation during atherosclerosis resolution results in Treg expansion and an acceleration of atherosclerosis resolution by increasing Treg number.

Concentrations of digestible and metabolizable energy, ileal digestibility of amino acids, and digestibility of phosphorus in a new variety of soybeans fed to growing pigs

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INTRODUCTION: The objective was to test the hypothesis that digestible energy (**DE**), metabolizable energy (**ME**), standardized ileal digestibility (**SID**) of amino acids (**AA**), and standardized total tract digestibility (**STTD**) of P in soybean meal produced from a new variety of soybeans (**Photoseed**) are not different from conventional soybean meal (**SBM-CV**).

METHODS: In Exp. 1, 30 pigs (18.3 ± 1.3 kg) were randomly allotted to a corn diet or 2 diets containing corn and Photoseed or corn and SBM-CV. Pigs were housed in metabolism crates and feces and urine were collected for 4 d after 5 d of adaptation. Feces and urine were analyzed for gross energy and DE and ME were calculated for each ingredient. The statistical model included ingredient as fixed effect and replicate as random effect and pig was the experimental unit. Results indicated that DE and ME of Photoseed were not different from DE and ME in SBM-CV. In Exp. 2, nine barrows (30.0 ± 1.5 kg) with a T-cannula in the distal ileum were allotted to a triplicated 3 × 3 Latin Square design with 3 diets and 3 periods in each square. AN N-free diet and diets containing SBM-CV or Photoseed were used. Pigs were housed individually in fully slatted pens and ileal digesta were collected on d 6 and 7 of each period. Digesta were lyophilized, ground, and analyzed for AA, and SID of AA was calculated. The statistical model included diet as fixed effect and square, period, and pig as random effects and pig was the experimental unit. Results indicated that the SID of Arg, Ile, and Lys were not different between the two ingredients, but the SID of other indispensable AA were greater ($P < 0.05$) in SBM- CV than in Photoseed. In Exp. 3, forty-eight barrows (12.0 ± 1.6 kg) were allotted to 6 diets with 8 pigs per diet. The SBM-CV or Photoseed were included in diets with 3 levels of microbial phytase (0, 500, or 1,000 units/kg). Pigs were housed in metabolism crates and feces were collected quantitatively for 4 d after 5 d of adaptation. Fecal samples were dried and analyzed for P and the STTD of P was calculated. The statistical model included ingredient, phytase, and the interaction between ingredient and phytase as fixed effects and replicate as the random effect. The pig was the experimental unit.

RESULTS: Results indicated that inclusion of phytase in the diets increased ($P < 0.05$) the STTD of P, but STTD of P in Photoseed was not different from the STTD in SBM-CV.

CONCLUSION: In conclusion, no differences in DE and ME, STTD of P, and SID of some indispensable AA between Photoseed and SBM-CV were observed.

Effects of commercial and traditional kefirs on apparent total tract macronutrient digestibility and fecal characteristics, metabolites, and microbiota of healthy adult dogs

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INTRODUCTION: Our objectives were to determine the effects of commercial or traditional kefirs on apparent total tract macronutrient digestibility (ATTD) of a diet when fed to healthy adult dogs, and determine the fecal characteristics, microbiota populations, and metabolite and immunoglobulin (Ig) A concentrations of healthy adult dogs fed commercial or traditional kefirs.

METHODS: Twelve healthy adult dogs (mean age: 5.67±1.72 y, mean BW: 7.27±1.15 kg) were used in a replicated 3x3 Latin square design (n=12/group). All dogs were fed a commercial diet and allotted to 1 of 3 treatments: 2% reduced-fat milk treated with lactase (CNTL), commercial kefir (Champions Choice; C-Kefir), or traditional kefir brewed daily from 2% reduced-fat milk and kefir grains (Kefir Garden; T-Kefir). The experiment was composed of three 28-d periods, with each consisting of a 22-d transition phase, a 5-d fecal collection phase, and 1 d for blood collection. Dogs were weighed and body condition was assessed weekly before feeding. Blood samples were collected for serum chemistry and hematology measurements. Fecal samples were collected for the determination of pH, dry matter (DM), microbiota populations (16S rRNA gene amplicons), ATTD, and metabolite and IgA concentrations. Data were analyzed using Mixed Models procedure of SAS 9.4. The main effects of treatment were tested, with significance set at $p<0.05$ and trends set at $p<0.10$.

RESULTS: T-Kefir had a higher ($p<0.0001$) microbial count (1.79E+09 CFU/mL) than CNTL (4.57E+03 CFU/mL) and C-Kefir (6.95E+04 CFU/mL). Fecal microbiota populations were weakly impacted by kefir consumption. Bacterial alpha diversity tended to be greater ($p=0.10$; Faith's PD) in dogs fed T-Kefir than those fed CNTL. Beta diversity analysis (unweighted principal coordinates analysis) identified a difference ($p<0.0004$) between dogs fed CNTL and those fed C-Kefir. At the phylum level, dogs fed C-Kefir tended to have greater ($p=0.06$) relative abundance of *Fusobacteriota* than those fed CNTL or T-Kefir. Dogs fed T-Kefir had a greater ($p<0.0001$) relative abundance of *Lactococcus* than those fed CNTL or C-Kefir. Dogs fed T-Kefir also tended to have lower ($p=0.09$) relative abundance of *Escherichia-Shigella* and greater ($p=0.09$) relative abundance of *Candidatus stoquefichus* than dogs fed CNTL or C-Kefir. Dogs fed C-Kefir tended to have lower ($p=0.08$) fecal valerate concentrations than those fed CNTL or T-Kefir. Fresh fecal pH, DM, IgA and metabolite concentrations, and blood biomarkers were not affected by treatment.

CONCLUSION: The supplementation of commercial or traditional kefir to healthy adult dogs had minor effects on fecal microbiota populations and fecal metabolite concentrations. Stool quality, fecal IgA concentrations, and blood metabolites were unaffected.

Poster Section: Dietary Intervention Models II

Concentration of digestible energy in corn-based diets fed to gestating and lactating sows is increased by adding xylanase to the diets

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INTRODUCTION: The hypothesis that exogenous xylanase added to diets for gestating and lactating sows will increase the apparent total tract digestibility (**ATTD**) of gross energy (**GE**) and total dietary fiber (**TDF**), and digestible energy (**DE**) and metabolizable energy (**ME**) was tested.

METHODS: Two diets for gestating and two diets for lactating sows containing corn, soybean meal, distillers dried grains with solubles, wheat middlings, and soybean hulls were formulated without or with 16,000 BXU per kg of exogenous xylanase (Econase XT; AB Vista, Marlborough, UK). Lactation diets contained an indigestible marker. Diets were fed to gestating and lactating sows in two reproductive cycles. A total of 106 gilts and sows were randomly allotted to the two gestation diets 7 d after breeding in a randomized complete block design with 4 blocks. On d 30 post-breeding, 8 non-pregnant animals were removed. From the remaining 98 animals, 48 sows (24 replicates per treatment, 12 sows per block) were placed in metabolism crates on d 35 (mid-gestation) for 10 d with feces and urine collected for 5 d. The same 48 sows were placed in metabolism crates again on d 95 (late-gestation). All sows were moved to the lactation unit on d 106 and lactation diet feeding was initiated. Fecal samples were collected (grab-sampling) for 5 d starting on d 10 post-farrowing. Sows were weaned on d 20 and 63 sows were rebred. Of these sows, 46 sows were placed in metabolism crates on d 35 and 95 as in the first cycle, and treatments in the farrowing unit were also as in the first cycle. Data were analyzed using the MIXED procedure of SAS with sow as the experimental unit, diet as fixed effect, and block and replicate as random effects.

RESULTS: Results indicated that in the first cycle, the ATTD of TDF in late-gestation were greater ($P < 0.05$) in sows fed the xylanase-diet compared with the control diet. During the first lactation period, sows fed the xylanase-diet had greater ($P < 0.05$) ATTD of TDF and GE and greater ($P < 0.05$) DE than sows fed the control diet. During the second gestation period, sows fed the xylanase-diet had greater ($P < 0.05$) ATTD of GE, and DE in mid-gestation and tended to have greater ($P < 0.05$) DE in late-gestation, but no differences were observed in ATTD of TDF. During the second lactation period, sows fed the xylanase-diet had greater ($P < 0.05$) ATTD of TDF and GE and greater ($P < 0.05$) DE than sows fed the control diet.

CONCLUSION: In conclusion, DE was greater in the xylanase-diet than the control diets during two reproductive cycles, and sows fed lactation diets with xylanase had greater digestibility of fiber.

Digestible indispensable amino acid score (DIAAS) in animal-based burgers versus plant-based burgers, as well as additivity in mixed meals

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INTRODUCTION: Animal-based proteins constitute a significant portion of the human diet in most developed countries. However, predictions for animal protein indicate that consumption will begin to decline because plant-based proteins have become accepted as having an appearance and taste similar to animal products. The digestible indispensable amino acid score (DIAAS) is recommended by FAO as the best method for determining protein quality in human foods. This method allows for calculation of the protein value of individual ingredients and combined meals consisting of several proteins. Therefore, the objectives of this study were to determine DIAAS values for animal- and plant-based burgers and test the hypothesis that DIAAS calculated for a combination of burger and a burger bun is additive in a combined meal.

METHODS: Ten ileal cannulated gilts (initial body weight: 24.6 ± 1.3 kg) were fed experimental diets for six 9-day periods with ileal digesta being collected on days 8 and 9 of each period, with the first 7 days serving as an adaptation period to the diet. Six diets contained a burger (i.e., 80% lean beef, 93% lean beef, 80% lean pork, Impossible Burger, or Beyond Burger) or a burger bun as the sole source of amino acids (AA). Three additional diets were based on a combination of the bun and 80% beef, pork, or Impossible Burger. A nitrogen-free diet was also used to measure basal endogenous losses of AA. The standardized ileal digestibility (SID) of each AA was calculated for all foods and combined meals (measured and predicted). In addition, DIAAS values were calculated for children from 6 months to 3 years and for individuals older than 3 years, and DIAAS in combined meals was predicted from individual ingredient DIAAS values.

RESULTS: For individuals older than 6 months, the 93% lean beef and pork burgers had the greatest ($P < 0.05$) DIAAS, whereas the Beyond Burger had the lowest ($P < 0.05$) DIAAS when comparing all burgers. The 80% lean beef burger had greater ($P < 0.05$) DIAAS than the plant-based burgers for children from 6 months to 3 years. For individuals older than 3 years, no differences were observed between the 80% lean beef and Impossible Burger, but the 80% lean beef had greater ($P < 0.05$) DIAAS compared with Beyond Burger. Regardless of age group and burger-bun combination, there were no differences between the measured and the predicted DIAAS.

CONCLUSION: The protein quality of animal-based burgers is greater than that of plant-based burgers for individuals older than 6 months. However, for individuals older than 3 years, the Impossible Burger has comparable protein quality to the 80% lean beef burger. The high DIAAS in beef and pork burgers allowed for the low protein quality in burger buns to be compensated for. However, the combination of the Impossible Burger and bun does not provide enough digestible AA to meet the requirements. The DIAAS in mixed meals is additive and can be predicted from the individual ingredient DIAAS values.

Using yellow mealworm (*Tenebrio molitor*) and lesser mealworm (*Alphitobius diaperinus*) as alternative protein sources reduced weight gain, improved blood lipid profiles, and altered adipose and hepatic gene expression of diet-induced obesity mice

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INTRODUCTION: Insect-based proteins have been identified as sustainable, high-quality protein sources that may be used as alternatives to conventional proteins. Studies have also shown that a high-protein diet exhibits beneficial effects on obesity by modulating satiety and energy metabolism. Although mealworms have gained increasing attention recently, limited information is known about different mealworm species and their ability to impact metabolism and obesity. We aimed to determine the effects of yellow mealworm (*Tenebrio molitor*)- or lesser worm (*Alphitobius diaperinus*)-based proteins on the body weight (BW), serum metabolites, and liver and adipose tissue histology and gene expression profiles of diet-induced obesity mice.

METHODS: Male C57BL/6J mice were fed a high-fat diet (HFD; 45% kcal from fat) for 8 wk to induce obesity and metabolic syndrome that was confirmed by intraperitoneal glucose tolerance tests (IPGTT) at 20 wk of age. Obese mice were then randomly assigned to 1 of 5 treatments (n=10/group) for another 8 wk following a 1-wk diet transition: HFD: high-fat controls continued to receive HFD based on casein protein; B50: HFD where 50% protein derived from lesser mealworm; B100: HFD where lesser mealworm was the sole protein source; Y50: HFD where 50% protein derived from yellow mealworm; Y100: HFD where yellow mealworm was the sole protein source. Lean mice (n=10) fed a low-fat-diet (LFD; 10% kcal from fat) were also included. Food intake was recorded every 2 d, BW, body composition, and IPGTT were measured every 2 wk. After 8 wk, serum was collected, and perirenal adipose depot and liver tissue samples were collected for gene expression, histopathology, and triglyceride analysis.

RESULTS: After 8 wk, mice fed HFD, B50 and B100 had greater ($P<0.05$) weight gain than mice fed LFD, whereas mice fed Y50 and Y100 did not differ from those fed LFD ($P>0.05$). Mice fed Y50, B100 and Y100 had a lower ($P<0.05$) BW change rate than mice fed HFD. Obese mice fed mealworm-based diets had increased ($P<0.05$) serum HDL and reduced ($P<0.05$) serum LDL concentrations, and reduced ($P<0.05$) LDL/HDL ratio. Mice fed mealworm-based diets had increased ($P<0.05$) expression of genes related to immune response and antioxidants in the liver, and reduced ($P<0.05$) expression of genes associated with chemoattractants, inflammation, and apoptosis in adipose tissue. Mealworm-based diets also altered ($P<0.05$) glucose and lipid metabolism genes in the liver, and to a lesser extent, in adipose tissue.

CONCLUSION: Mealworms may not only serve as an alternative protein source, but may confer beneficial health effects in diet-induced obesity mice.

Fructan chain-length influences enteric microbiota-host GABAergic signaling

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INTRODUCTION: Nearly half of the adult population suffers from a functional gastrointestinal disorders (FGID), which are characterized by severe intestinal dysmotility. While FODMAP (fermentable oligo-, di-, mono- saccharides and polyols) elimination diets are a promising clinical treatment for FGIDs, they fail in around half of patients. Those who do benefit tend to implement the diet long-term, thus lowering long-term diet quality and fiber intake. Fructooligosaccharides, or fructans, are a fiber and FODMAP subtype that varies in chain-length. Long-chain fructans are more slowly fermented, produce gradual decreases in luminal pH, and are reported to cause fewer intestinal symptoms compared to short-chain fructans. Lower luminal pH enhances enteric microbial synthesis of gamma-aminobutyric acid (GABA) (an inhibitory neurotransmitter of the enteric nervous system). Notably, patients with FGIDs have lower colonic mucosal GABA. Therefore, this study investigated whether fructan length plays a role in enteric microbiota-host GABAergic signaling.

METHODS: Female and male C57BL/6 mice (N=120) were randomly assigned to one of four experimental diets for two weeks: fiber-free diet (FFD), FFD + 20% cellulose (CELL) (inert fiber control), FFD + 10% cellulose + 10% short-chain fructooligosaccharide (scFOS), or FFD + 10% cellulose + 10% inulin (INU) (a long- chain fructooligosaccharide). Expression of 94 motility genes from ileum (ILE) and proximal colon (PC) tissue was collected using Fluidigm qPCR. GABA concentration of intestinal contents was quantified using ELISA.

RESULTS: Compared to FFD, both fructan diets (scFOS & INU) raised pH of ILE digesta. By contrast, both fructan diets reduced pH in PC digesta. All fiber diets increased ileal and colonic content GABA vs FFD. Fructan chain-length influenced motility gene expression in a segment-dependent manner. Fructans influenced gene expression inconsistently in ILE. In ILE, INU influenced expression of 50 motility genes, while scFOS influenced only 14 compared to FFD. INU enhanced expression of 12 ILE GABAergic genes including 9 GABA receptor subunits (*Gabbr1*, *Gabbr2*, *Gabra1, Gabrb1, Gabrb2, Gabrb3, Gabrg1, Gabrg2, Gabrg3*), and GABA synthesis and degradation enzymes (*Gad1, Gad2, Abat*) compared to FFD. Notably, expression of only 3 GABAergic genes (*Gabbr1, Gabbr2, Abat*) were enhanced by scFOS in ILE. Fructans influenced gene expression more consistently in PC. In PC, INU influenced expression of 52 motility genes, while scFOS influenced 60 compared to FFD. Both fructan diets diminished expression of 8 GABA receptor subunits (*Gabbr1, Gabra1, Gabra2, Gabra5, Gabrb3, Gabrg1, Gabrg2, Gabrg3*), but enhanced expression of Abat compared to FFD.

CONCLUSION: Overall, these data indicate that fructan chain-length influences host-microbiota enteric GABAergic signaling in a segment-dependent manner. This suggests that certain FODMAP subtypes may impact symptoms of FGIDs differently, thus increasing fiber intake options. Ongoing studies are investigating how these changes in GABAergic signaling contribute to intestinal motility phenotypes.

Poster Section: Clinical and Preclinical Metabolism I

Chemotherapy-induced changes in gut microbial composition disrupt entero-hepatic bile acid metabolism.

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INTRODUCTION: Chemotherapy causes gastrointestinal side effects such as bile acid (BA) malabsorption leading to diarrhea, bloating, malnutrition, and treatment failure. BA malabsorption triggers gastrointestinal symptoms by stimulating colonic motility and fluid secretion. While enterotoxicity, chemotherapy-induced death of absorptive intestinal epithelial cells, is the presumed basis of BA malabsorption in chemotherapy-treated patients, other key players in BA metabolism like the microbiome remain under investigated. BA signaling is balanced by both host synthesis of primary BA and microbial synthesis of secondary BA. However, the impact of chemotherapy on microbial BA metabolism remains unknown. Therefore, we hypothesize that chemotherapy-induced changes in enteric microbial composition provoke disruptions in enterohepatic BA metabolism and, thus, gastrointestinal side effects.

METHODS: Adult female mice (N=18) were randomized to receive intraperitoneal injections of either the vehicle control (n=10) (Veh) or the chemotherapy paclitaxel (n=8) (30mg/kg; Chemo) every other day for a total of 6 injections. Animals were sacrificed three days after the final injection. Germ-free adult female mice (N=18) received a gut-microbial transplant (GMT) (ileal, cecal, and colon contents) from a cohort of mice treated with Veh (n=9) or Chemo (n=9) as explained above. Mice were sacrificed one week following GMT. Expression of genes central to BA synthesis, transport, and signaling pathways was measured in liver and intestinal tissues via qPCR.

RESULTS: Chemo altered expression of genes relating to BA metabolism in the liver, ileum, and colon. In the liver, BA synthesizing enzymes (Cyp7a1 and Cyp7b1) were reduced in Chemo vs Veh and in Chemo-GMT vs Veh-GMT. BA transporters (Abcc1, Abcg8, Abcb1a, Abcb1b, and Abcb4) were reduced in Chemo vs Veh group. However, only Abcb1b was reduced in Chemo-GMT vs Veh-GMT. Additionally, BA receptors (Gpbar1, Nr1h4, Nr1h3, Vdr, Nr1i2, and Klb) were reduced in Chemo vs Veh. However, only Nr1h4 was reduced in Chemo-GMT vs Veh-GMT. In the ileum, BA transporter (Slc10a2), BA signaling protein (Fgf15), and BA receptors (Nr1h4, Nr1i2, and Nr1h3) were reduced in Chemo vs Veh. Fgf15, Nr1h4, and Nr1i2 but not Slc10a2 or Nr1h3 were reduced in Chemo-GMT vs Veh-GMT. In the colon, BA receptors (Vdr and Nr1i2) were reduced in Chemo vs Veh, but no changes were elicited in Chemo-GMT vs Veh-GMT. BA receptor (Gpbar1) was increased in both Chemo vs Veh and Chemo-GMT vs Veh-GMT.

CONCLUSION: The microbiome plays a role in BA metabolism by affecting transcription across the enterohepatic axis. While many effects of Chemo were recapitulated by transplanting only the microbiota into Chemo-GMT animals, incomplete transfer of all effects indicates that the microbiome is not the only contributor to chemotherapy-induced disruptions in BA metabolism. Future studies are required to investigate how microbial metabolism of BA is modified by chemotherapy.

Bile acid modulation of macrophage phenotype in colorectal cancer

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INTRODUCTION: Cyclooxygenase-2 (COX-2) is overexpressed in CRC, which leads to more PGE2 production and higher proliferation and survival of cancer cell. Bile acids can affect host immune responses. As LCA isomers, isoalloLCA enhances Treg differentiation. IsoLCA suppressed TH17 cell differentiation by inhibiting retinoic acid receptor-related orphan nuclear receptor-gt, a key TH17-cell-promoting transcription factor, similar to its isomer 3-oxo-LCA. PGE2 was shown by the literature to differentiate naïve T cells into TH17 instead of Tregs.

METHODS: Clinical samples were collected from colonoscopy in whom tumors were not identified at the Rush University Medical Center, Chicago. Based on the current literature, we know African Americans are more prone to colorectal cancer than non-Hispanic whites. To further investigate the effects of allo bile acids on macrophages, we isolated macrophages from mouse for testing. Mice from M-Cre, myeloid specific IDO1-KO strains were dissected, and the peritoneal macrophages were isolated and treated with LPS for 24 hours then 20 μM methanol (vehicle), LCA, and IALCA for 24 hours. The conditioned media are collected after 48hrs and used to run PGE_2 enzyme-linked immunoassay (ELISA).

RESULTS: The Nanostring RNA analysis showed multiple immune checkpoint expression aligns with expression of FoxP3 (the master TF of Tregs) and sex hormone receptors, mainly within females. We compared RNA expression levels to fecal bile acid concentrations; intriguingly, the IALCA and isoLCA concentrations were significantly higher in female NHW than female AA. In IALCA, $p=0.032$; in isoLCA, $p = 0.017$. The same effect is not observed within the male population. As NHWs are known to have lower CRC incidence rate than AAs, this observation show that the IALCA and isoLCA have anti-inflammatory effects in the GI tract, which lowers inflammation within the local intestinal microenvironment. The heatmap from our AA/NHW human data on IDO related genes showed PGTS2 expression (COX-2) aligns with female and low risk groups the most, which is intriguing considering the gender differences on immune responses.

CONCLUSION: Increasing evidence indicates that the allo forms of bile acids may activate distinct signaling functions in immune cells compared to their “traditional” isomers.

Mining the scientific literature to support personalized nutrition applications

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INTRODUCTION: Nutrition plays a fundamental role in maintaining health and preventing disease. The goal of personalized nutrition is to develop tailored dietary approaches to prevent or treat disease at the individual level. Much scientific knowledge about the interconnections among diet, the gut microbiome and metabolome, and human health remains buried in the scientific literature. Thus, research is needed to enhance literature mining on this topic. Our objectives include developing NLP (Natural Language Processing) models to standardize and organize relevant scientific evidence in the published literature to support personalized nutrition applications and construct a comprehensive literature-based knowledge graph (a network of entities and their relationships extracted using NLP).

METHODS: Annotation guidelines were developed, and an inter-annotator agreement was established to generate a ground truth dataset. A data model was created in the Brat annotation tool representing the entities and relationships used throughout the annotation process. The model was used to annotate nutrition/microbiome-related publications in PubMed with relevant entities (e.g., foods, microbes, metabolites, diseases) and their relationships (e.g., food-microbe interactions).

RESULTS: We annotated 165 total publications (32 with result sections), including 8 *in vitro*, 33 *in vivo*, and 124 human studies relevant to the nutrition-microbiome field. The annotations included approximately 11,000 entities and 3,000 relationships. The information present in the publications were rich and contained complex sentence structures.

CONCLUSION: We annotated a sufficiently large literature mining dataset that will allow for the development of NLP and machine learning models. The data set is noteworthy for the complex structures of sentences and relationships, which will likely present NLP challenges.

Vitamin A secretion in macrophages

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INTRODUCTION: Vitamin A plays a crucial role in modulating macrophage function. Retinoic acid, the transcriptionally active form of vitamin A, is released by macrophages to promote T-cell differentiation. Our laboratory is interested in the mechanisms that regulate vitamin A secretion.

METHODS: Using a method developed in our lab that allows us to load cells with vitamin A, we compared the release of retinoids between macrophages and hepatocytes by performing kinetic studies. Next, we focused on the mechanisms that regulate vitamin A secretion in the macrophage. Fully differentiated primary murine macrophages were exposed to pig serum containing chylomicrons loaded with vitamin A (primarily RE). After 24 h, we harvested a subset of cells (baseline), and maintained the remaining cells 1, 3, 6, or 24 h on a vitamin A-free media to study the amounts of retinoids released from the cell. We measured intracellular and extracellular (media) vitamin A by high-performance liquid chromatography. Cells were exposed to different experimental conditions such as chloroquine (to block lysosomal degradation). We also utilized Apoe (Apolipoprotein E) knockout and RBP4 (Retinol Binding protein 4) knockout macrophages to test out the role of these lipid transporters on macrophage vitamin A release.

RESULTS: The results demonstrated that the absence of RBP4 in macrophages did not reduce vitamin A secretion. Blocking of the lysosomal activity to prevented hydrolyzation of retinyl ester to retinol and high accumulation of retinyl ester did not affect the extracellular levels of vitamin A.

CONCLUSION: Our data show that multiple pathways regulate vitamin A release from the macrophage.

Poster Section: Clinical and Preclinical Metabolism II

Activation of membrane progesterone receptors induce glycogenolysis in uterine epithelial cells

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INTRODUCTION: In humans and cattle, 40-60% of pregnancies fail, with most losses occurring prior to or during implantation. Throughout this time, the developing embryo needs glucose to survive. The demand for glucose increases dramatically at the morula stage as the embryo enters the uterus. Yet, how glucose is regulated to match the changing needs of the embryo is unclear. Glycogen is composed of thousands of glucose molecules and is present in the uterine epithelium of many species. Our lab has shown that the glycogen content of the bovine uterine epithelium was lower during the luteal phase than at estrus. Therefore, our objective was to elucidate the role of progesterone in glycogenolysis of uterine epithelial cells.

METHODS: Bovine uterine epithelial (BUTE) and Ishikawa cells were treated as indicated. Glycogen was isolated with KOH, hydrolyzed to glucose, and then measured. cAMP was measured via ELISA, and pAMK was measured via western blot. Immunohistochemistry was conducted on bovine uteri collected on days 1 and 11 of the estrus cycle.

RESULTS: Progesterone decreased glycogen levels in BUTE cells by 99% ($P=0.0002$). RU486 did not block progesterone's effect, indicating that the effect of progesterone was not mediated by nuclear progesterone receptors (nPR). Thus, we hypothesized that the effect of progesterone is mediated by membrane progesterone receptors (mPRs). RT-PCR confirmed that BUTE cells expressed all five mPRs (α, β, γ, δ, and ε). Like progesterone, a specific mPR agonist (Org OD 02-0) decreased glycogen levels in BUTE cells by 99% ($P<0.0001$). Indicating a potential for mPRs to regulate glycogen in vivo, immunohistochemistry showed that the bovine uterine epithelium expressed high levels of mPRs. These results validate that progesterone is acting through mPRs to stimulate glycogen breakdown in BUTE cells. Once progesterone binds to the mPR, two signaling pathways can be activated, cAMP and AMPK. Neither progesterone nor Org OD 02-0 changed intracellular cAMP concentrations. In agreement, the adenylyl cyclase activator forskolin increased cAMP concentration 25-fold but did not decrease glycogen levels. Progesterone treatment increased pAMPK levels by 87% in BUTE at 24 hours compared to the control ($P=0.0001$). BUTE cells treated with an AMPK activator (D982) had a decrease in glycogen ($P=0.0051$). Supporting these results in vivo, pAMPK levels in the uterine epithelium were high in the bovine uterine epithelium during the luteal phase when glycogen levels were low. In a human model, progesterone and Org OD 02-0 decreased glycogen levels to a similar extent in Ishikawa cells that express mPRs but do not express nPRs ($P=0.0027$).

CONCLUSION: Progesterone acting through the mPR and AMPK to stimulate glycogenolysis may play a crucial role in providing glucose to endometrial tissue or the growing embryo. We are currently working to elucidate the pathway downstream of AMPK that lead to the breakdown of glycogen in BUTE cells.

SEC16B modulates high-fat diet induced obesity in female but not male mice

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INTRODUCTION: As obesity and its associated complications continue to rise in the US, it is crucial to gain a better understanding of the complex cellular processes involved in lipid metabolism. Genome-wide association studies (GWAS) have identified single nucleotide polymorphisms (SNPs) in *SEC16B* gene to be associated with obesity in a variety of populations. Previous studies have shown that SEC16 is involved in COPII coat assembly that allows for the packaging and delivery of proteins and lipids from the ER to the Golgi. While the function of SEC16B in ER transport is being defined, its potential roles in obesity have not been investigated. In this study, we aim to characterize the phenotype of a *Sec16b* knockout mouse model to further our knowledge of SEC16B in fat metabolism.

METHODS: We generated adipose-specific *Sec16b* knockout (AKO) mice by crossing *Sec16b* floxed mice with *Adiponectin-Cre* mice. Wild-type (WT) and AKO mice were challenged with a high-fat diet (HFD) and some females underwent ovariectomy surgery (OV). All mice groups were subjected to a glucose and insulin tolerance tests. Metabolic rate was measured with indirect calorimetry using the Comprehensive Lab Animal Monitoring System (CLAMS). Body compositions were measured with Echo MRI. Fat depots and the livers were collected for histology and gene expression analysis. Stromal vascular fraction (SVF) cells were harvested and differentiated *in vitro*. RNA sequencing was performed with epididymal white adipose tissues isolated from control and AKO female mice fed HFD.

RESULTS: When challenged with a HFD, female AKO mice gained more body weight and showed less energy expenditure compared to their WT littermates. RNA seq analysis revealed a decrease in the expression of *Ucp-1* and *Cidea*, two genes involved in thermogenesis, in AKO mice. In contrast, loss of *Sec16b* had no effect on HFD-induced obesity or energy expenditure in male mice. The AKO females also showed increased lipid accumulation in the liver and brown adipose tissue (BAT). Due to significant differences were only seen in females, we then performed OV to examine if estrogen contributes to the different body weight gain in female AKO mice. Interestingly, the ovariectomized AKO mice had significantly less body weight gain compared to their WT counterparts upon HFD challenge. Furthermore, the ovariectomized AKO mice showed improved insulin sensitivity, increased metabolic rate, decreased body fat percentage and reduced liver to body weight ratio compared to WT controls. Histology analysis revealed less lipid accumulation in the BAT and livers of ovariectomized AKO mice. Surprisingly, the *in vitro* differentiation of SVF cells isolated from whole body *Sec16b* knockout and WT mice showed no differences in lipid droplet accumulation. Thus, estrogen likely contributes to the effect of *Sec16b* deficiency on obesity in females.

CONCLUSION: Our studies revealed a novel regulatory mechanism of adiposity in females that involves the cooperation between SEC16B and estrogen. While further studies are be needed to elucidate the underlying mechanisms, these findings suggest that modulating *SEC16B* expression and estrogen may be beneficial for those with increased body fat and postmenopausal women.

Comparison of microbiota analytic techniques

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INTRODUCTION: Increasingly, there are many bioinformatic and statistical programs available to analyze microbiota data. We aimed to compare analytic techniques.

METHODS: We utilized data from a crossover trial in adults (n=24) who consumed prebiotics (5 g/d fructooligosaccharides + 5 g/d galactooligosaccharides; PRE) and no-fiber control (CON) for 4 weeks each. The 16S rRNA V4 region was amplified from extracted fecal DNA with a Fluidigm Access Array prior to high-throughput sequencing on Illumina HiSeq. FASTX-Toolkit, DADA2, and QIIME2 were used to process sequence data. Taxonomy was assigned with SILVA 138. Microbiota data were analyzed with differential abundances and taxa rankings. Differential abundance analyses were conducted via Wilcoxon signed-rank tests, Analysis of Compositions of Microbiomes with Bias Correction (ANCOMBC), and Linear Discriminant Analysis Effect Size (LEfSe). Compositional taxa rankings were created with DEICODE and Songbird. Qurro (Quantitative Rank/Ratio Observations) was used to visualize taxa rankings and sample log-ratio plots. Wilcoxon rank-sum tests or Welch's t-tests quantified Qurro findings.

RESULTS: Using Wilcoxon signed-rank tests, Actinobacteriota increased in PRE compared to CON (P=0.004, q=0.02). *Bifidobacterium* (P=0.001, q=0.03) and *Anaerostipes* (P=0.02, q=0.16) increased, while *Roseburia* (P=0.03, q=0.16) and *Ruminococcaceae* CAG352 (P=0.02, q=0.16) decreased in PRE. From LEfSe, *Bifidobacterium* increased (d=4.35, P=0.03), while *Dielma* (d=1.95, P=0.03) and *Eubacterium brachy* group (d=2.08, P=0.04) decreased in PRE. With Qurro, ratio of top to bottom ranked taxa from DEICODE principal component axis 3 increased in PRE (P=0.05), with *Bifidobacterium* and *Anaerostipes* among top taxa. From the Songbird treatment differential, ratio of top to bottom ranked taxa increased in PRE (P=0.001), with *Bifidobacterium* among top taxa.

CONCLUSION: *Bifidobacterium* enrichment was consistently detected using various analytic techniques. However, microorganisms affected to a lesser degree were not consistent across platforms.

Greater increases in skin carotenoids are related to greater improvements in cognition among toddlers across six months

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INTRODUCTION: Carotenoids are antioxidant plant pigments, found in fruits, vegetables, and eggs, that accumulate in the human body (i.e., skin, adipose, and brain) and can serve as biomarkers for higher diet quality. Although carotenoid status has been associated with cognitive benefits in older children and adults, the relation between carotenoids and cognition during early life has received little attention. The Early Life Influences on Attention Study (ELIAS) is an on-going longitudinal study examining the influence of nutrition on visual and cognitive development among toddlers. We aimed to understand the longitudinal relationship between changes in skin carotenoids and cognitive, language, and motor abilities among toddlers, with the hypothesis that greater improvement in carotenoid status would be positively related to changes in these abilities.

METHODS: Preliminary data from ELIAS were utilized to conduct longitudinal analyses among toddlers across a six-month period (n = 31), with the first timepoint between 12-18 months ($\bar{x}$ = 14.3) and the second timepoint between 18-24 months ($\bar{x}$ = 20.5). Skin carotenoids were measured using reflection spectroscopy (i.e., VEGGIE METER®). Cognition, receptive and expressive communication, and fine and gross motor skills were measured using the Bayley Scales of Infant and Toddler Development IV Screening Test (BSID-IV). Partial correlations were conducted to assess relationships between changes in skin carotenoids and BSID-IV subsets, adjusting for age.

RESULTS: Skin carotenoid change scores (6-month follow-up - Baseline) were positively related to BSID-IV cognitive ability change scores (r = 0.548, p < 0.001). No significant associations were observed between skin carotenoid change scores and receptive communication (r = 0.147, p = 0.211), expressive communication (r = -0.119, p = 0.258), fine motor (r = -0.048, p = 0.397), or gross motor (r = -0.027, p = 0.442) change scores.

CONCLUSION: Greater increase in skin carotenoids was positively associated with greater improvements in cognitive abilities among toddlers over a 6-month longitudinal follow-up. The domain-specificity of the findings points to the importance of carotenoid status for early cognitive development.

Poster Section: Cardiovascular Health

Statin-dependent suppression of chemotaxis occurs in a geranylgeranyl pyrophosphate-dependent manner independent of both cholesterol and farnesyl pyrophosphate

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INTRODUCTION: A class of cholesterol-lowering medications, statins, increase sepsis-survival but only when taken prophylactically, which limits their clinical utility. Identifying how statins promote sepsis survival may reveal novel statin-inspired therapeutic targets that do not require prophylactic treatment. Our recent work showed that increased survival of prophylactically-treated mice was linked to cell-intrinsic inhibition of immune cell chemotaxis.

METHODS: A series of *in vitro* chemotaxis experiments were used to identify which metabolites downstream of statins were required for chemotaxis, including cholesterol and the other two non-sterol terminal metabolites farnesyl pyrophosphate (FPP) and geranylgeranyl pyrophosphate (GGPP).

RESULTS: Statin-treatment inhibited chemotactic responses in a dose-dependent manner. Restoring cholesterol or FPP in statin-treated both failed to restore chemotaxis, while supplementing with GGPP fully restored chemotactic function in a dose-dependent manner. Thus, inhibition of chemotaxis by statins depended on GGPP but not FPP or cholesterol. However, since FPP is the metabolic precursor in the synthesis of GGPP, it was expected that FPP supplementation should have restored both FPP- and GGPP-dependent functions. Similar unexplained findings have been prevalent in the broader literature. We hypothesized that the synthesis of GGPP from FPP required upstream metabolites that were also depleted by statin treatment. To test this, cells were treated with an FPP synthase inhibitor that blocked FPP synthesis without depleting other upstream metabolites. FPP synthase inhibitor suppressed chemotaxis, this was restored by FPP supplementation, and reversed by a GGPP synthase inhibitor. Thus, statin-dependent chemotaxis was indeed GGPP-dependent, and FPP-supplementation is only capable of restoring chemotaxis in the presence of metabolic precursors required for synthesis of GGPP.

CONCLUSION: There were two key findings. First, statin-dependent suppression of chemotaxis occurred in a GGPP-dependent manner independent of cholesterol and FPP. Second, a persistent unexplained phenomenon was regarding FPP to GGPP synthesis was solved by showing that FPP cannot be converted into GGPP in the absence of upstream metabolites that are also depleted by statins.

Exploring the role of HDL in carotenoid efflux and delivery to tissues

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INTRODUCTION: The high-density lipoprotein (HDL) eliminates excess cholesterol accumulated in tissues by transporting it to the liver. HDL also transports carotenoids, and previous data suggest that HDL can deliver carotenoids to tissues. The overall objective of this study is to examine whether HDL can mediate the efflux of carotenoids from tissues. We hypothesized that, as it occurs with cholesterol, HDL transports carotenoids from tissues to the liver.

METHODS: To examine the role of HDL in carotenoid distribution, we cross-bred β-carotene oxygenase 1-deficient (*Bco1*$^{-/-}$) mice, which mediates the cleavage of β-carotene to vitamin A, with mice over-expressing (*Apoal*$^{tg/tg}$) apolipoprotein A I (ApoA-I), the main protein component in HDL. We fed our mice a standard diet containing 50 mg/kg β-carotene for four or ten weeks. The concentration of β-carotene in plasma and tissues was measured by HPLC, and plasma ApoA-I levels were quantified by Western blot.

To determine the effect of HDL on carotenoid efflux from tissues, we fed *Bco1*$^{-/-}$ mice a standard diet containing 50 mg/kg β-carotene for four weeks. After this period, we infused them with either isolated human HDL or the same volume of saline. Carotenoids in tissues and plasma were collected for HPLC analyses and circulating ApoA-I levels were measured by Western blot.

RESULTS: *Bco1*$^{-/-}$*Apoal*$^{tg/tg}$ mice presented less β-carotene in the blood, adipose tissue, and eyes than *Bco1*$^{-/-}$ mice, while hepatic β-carotene levels followed the opposite trend. We did not observe changes in food intake or fecal carotenoid elimination. HDL infusions depleted plasma and adipose tissue β-carotene pools and increased hepatic β-carotene stores.

CONCLUSION: Overall, these data suggest that HDL promotes carotenoid efflux from tissues, which opens new avenues in our understanding of the role of lipoproteins in the biodistribution of carotenoids in mammals.

Finasteride treatment delays atherosclerosis progression and is associated with a reduction in plasma cholesterol

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INTRODUCTION: Finasteride is commonly prescribed to treat benign prostate hyperplasia and male-pattern baldness in cis men and, more recently, trans individuals. However, the effect of finasteride on cardiovascular disease remains elusive.

METHODS: We evaluated the role of finasteride on atherosclerosis using low-density lipoprotein (LDL) receptor-deficient (*Ldlr*$^{-/-}$) mice. Next, we examined the relevance to humans by analyzing the data deposited between 2009 and 2016 in the National Health and Nutrition Examination Survey (NHANES).

RESULTS: We show that finasteride reduces total plasma cholesterol and delays the development of atherosclerosis in *Ldlr*$^{-/-}$ mice. Finasteride reduced monocytosis, monocyte recruitment to the lesion, macrophage lesion content, and necrotic core area, the latter of which is an indicator of plaque vulnerability in humans. RNA sequencing analysis revealed a downregulation of inflammatory pathways and an upregulation of bile acid metabolism, oxidative phosphorylation, and cholesterol pathways in the liver of mice taking finasteride. Men reporting the use of finasteride showed lower plasma levels of cholesterol and LDL-cholesterol than those not taking the drug.

CONCLUSION: Our data unveil finasteride as a potential treatment to delay cardiovascular disease in people by improving plasma lipid profile.

Skin carotenoids in relation to cardiovascular health in adults

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INTRODUCTION: Carotenoids are plant pigments that may play a role in reducing arterial stiffness and improving cardiovascular health. The focus of this study was to investigate the relationship between skin carotenoids and markers of cardiovascular health (i.e., arterial stiffness and carotid artery wall thickness). The hypothesis was that measures of vascular health – such as pulse wave velocity (PWV), augmentation pressure and index, central (c) and peripheral (p) systolic and diastolic blood pressure (SBP and DBP respectively) – will be lower for individuals with greater skin carotenoids as compared to those with lower skin carotenoids.

METHODS: 39 adults aged 18-75 years participated in a cross-sectional study. Skin carotenoids were measured non-invasively using reflection spectroscopy (Veggie Meter®) and cardiovascular outcomes were assessed using the SphygmoCor® XCEL device. The relationship between skin carotenoids and cardiovascular health markers was analyzed using linear regression models after controlling for covariates (i.e., age, sex, income, BMI).

RESULTS: Skin carotenoids were inversely associated with augmentation pressure (β= -0.33, $p<0.05$). The effect remained significant after adjusting for age (β= -0.25, $p<0.05$), but not after adjusting for all covariates (β= -0.21, p= 0.13). While there was an inverse association with cSBP (β= -0.31, p=0.055), pSBP (β= -0.31, p=0.056) and central pulse pressure (β= -0.28, p=0.078), it did not reach statistical significance. Skin carotenoids showed no significant relationship with PWV (β= -0.02, p=0.91).

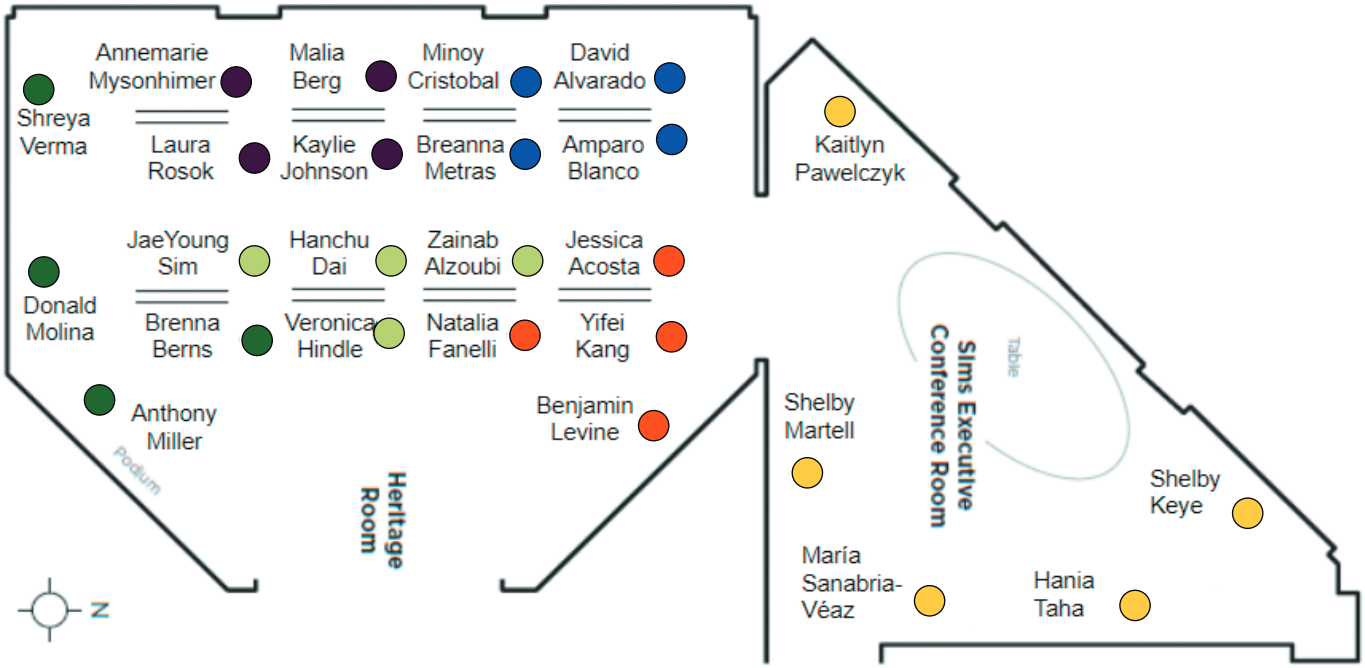
CONCLUSION: The findings of this preliminary study suggest a potential protective role of skin carotenoids against arterial stiffness, as indicated by the inverse relation with augmentation pressure. However, further research is needed in larger cohorts to confirm the association of skin carotenoids with other markers of cardiovascular health and investigate the potential mechanism underlying these relationships.

Location:

ACES Library, 1st floor

Heritage Room

5:15 – 6:45 pm



- Dietary Intervention
- Dietary Intervention Models I
- Dietary Intervention Models II
- Clinical & Preclinical Metabolism I
- Clinical & Preclinical Metabolism II
- Cardiovascular Health

Notes