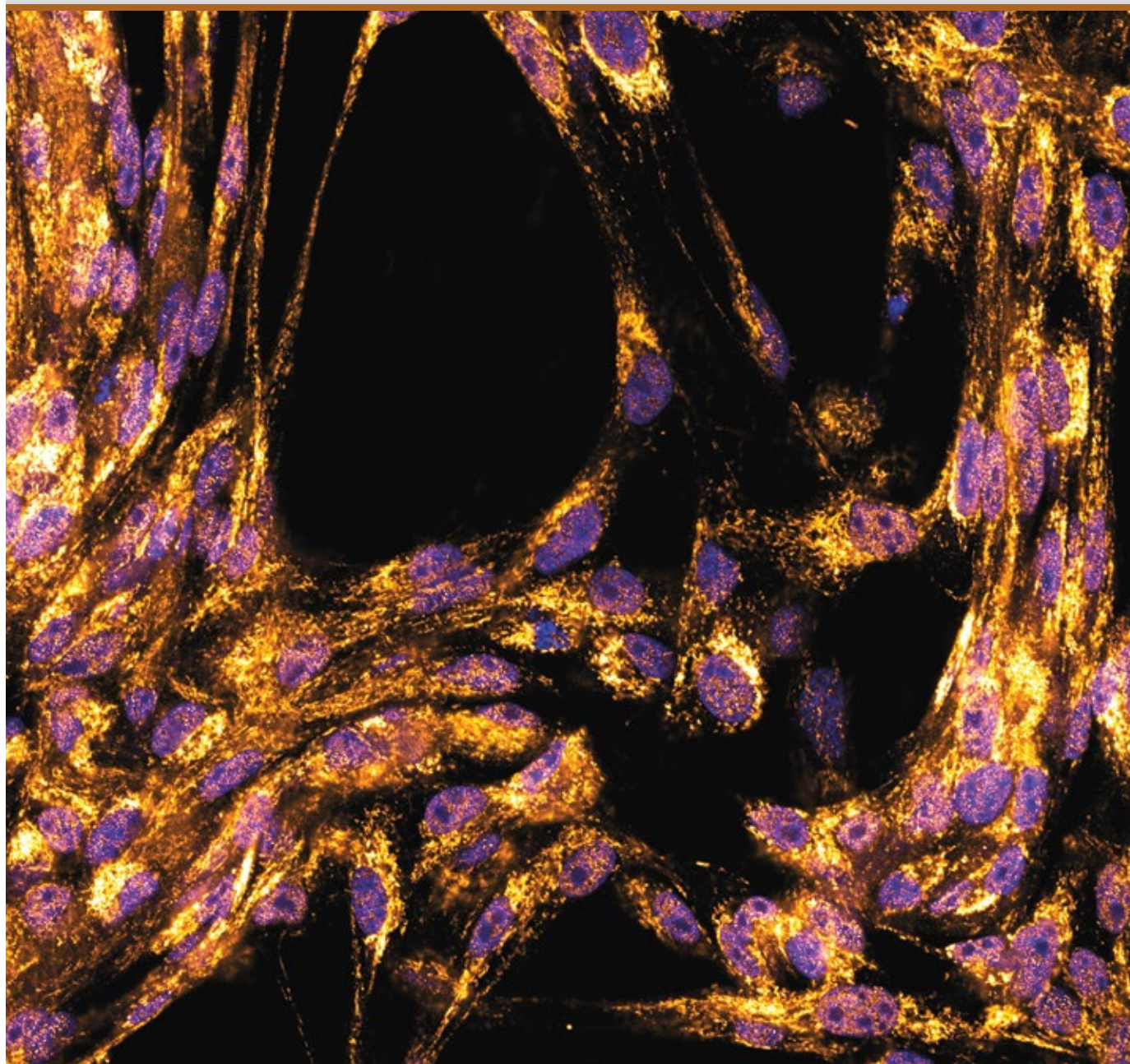


UNIVERSITY OF ILLINOIS AT URBANA-CHAMPAIGN | DIVISION OF NUTRITIONAL SCIENCES

2018 Nutrition Symposium

APRIL 18
2018

N•S•G•S•A Nutritional Sciences Graduate Student Association



50 *years*
DIVISION OF NUTRITIONAL SCIENCES
Preparing Transformative Leaders in Nutrition

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WELCOME

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 2018 Nutrition Symposium at the University of Illinois! The Nutrition Symposium is an important event for sharing ideas across disciplines and with the community.

Started in 1994 by NSGSA, the symposium offers students within DNS and related disciplines on campus an opportunity to present their nutrition research prior to the national meetings held annually in the spring. This symposium offers a first glance at exciting research in the areas of metabolic regulation, cancer, gastrointestinal physiology, immunology, physical activity, public health, and bioactive plant compounds. Students will be traveling and presenting at a variety of conferences, including the American Society for Nutrition Annual Meeting, American Society of Animal Sciences Joint Annual Meeting, and Experimental Biology.

This year, NSGSA is honored to have Dr. Thomas N. Seyfried deliver the keynote address, "Cancer as a mitochondrial metabolic disease: Implications for novel therapeutics." Dr. Seyfried will discuss the metabolic perturbations inherent

in cancer. Further, he will address novel therapeutic paradigms centered on leveraging these perturbations for management of the disease.

Additionally, NSGSA is proud to highlight the work of world-class faculty members through a mini-symposium. This year's presentations address metabolic alterations of disease, and will feature Drs. John Erdman, H. Rex Gaskins, and Hannah Holscher.

We are grateful to the many people involved with this meeting and program. We would like to first thank our keynote speaker, Dr. Thomas N. Seyfried. Thank you to our sponsors - their support is essential to the success and quality of the program. The NSGSA executive board and the symposium program committee have worked long and hard to organize an excellent program. We also thank the many others who contributed to this undertaking, including DNS staff and College of ACES Advancement Office staff. Most of all, we would like to thank our session chairs, judges, presenters and attendees for participating in this year's events and making them a success.

The Nutritional Sciences Graduate Student Association Board



Meal prep in the UIUC Metabolic Kitchen.

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COVER PHOTOGRAPH: “Glioblastoma in Hypoxia,” Submitted by J. Lumibao

Glioblastoma (GBM) is the most common and aggressive form of brain cancer in adults. The Gaskins Lab investigates mitochondrial responses to the GBM tumor microenvironment. Pictured are U87-EGFRvIII GBM cells incubated in hypoxia (1% O₂) and stained for nuclei (blue) and CHCHD2 (orange), a mitochondrial protein which participates in mitochondria-to-nucleus signaling and accumulates in the nucleus in response to hypoxia. We hypothesize that this phenomenon provides one mechanism by which GBM cells sense and adapt to a metabolically hostile tumor microenvironment, and could indicate a pathway to be targeted in future treatment regimens.

Photographs on pages 3, 7, 8, 12, and 13 submitted by Caitlyn Edwards

SCHEDULE OF EVENTS April 18, 2018

- *8:15 a.m. – 9:00 a.m.** **Breakfast**
Sims Executive Conference Room, ACES Library
 Sponsors, presenters, DNS students, faculty, and staff are invited
- *9:15 a.m. – 10:15 a.m.** **Graduate Student Oral Presentations 1**
Monsanto Room, ACES Library
 9:15 a.m. Brian J. Leyshon
 9:30 a.m. Yanling Wang
 9:45 a.m. Amanda N. Dainton
 10:00 a.m. Jan Lumibao
- 10:15 a.m. – 10:30 a.m.** **Break**
- *10:30 a.m. – 11:30 a.m.** **Graduate Student Oral Presentations 2**
Monsanto Room, ACES Library
 10:30 a.m. Alicia R. Jones
 10:45 a.m. Vanessa Lagos
 11:00 a.m. Katherine M. Ranard
 11:15 a.m. Diego Hernández-Saavedra
- 11:30 a.m. – 12:30 p.m.** **Lunch**
Heritage Room, ACES Library
 DNS students, presenters, and sponsors are invited, RSVP required
- 12:30 p.m. – 12:45 p.m.** **Break**
- *12:45 p.m. – 2:15 p.m.** **Faculty Mini-Symposium**
“Metabolic Alterations of Disease: What Role Can Nutrition Play?”
Monsanto Room, ACES Library
 12:45 p.m. Dr. John Erdman
 Tomato, Lycopene and Prostate Cancer: Molecular Targets
 1:15 p.m. Dr. H. Rex Gaskins
 Microbial Sulfur Metabolism and Colorectal Cancer Risk
 1:45 p.m. Dr. Hannah Holscher
 Diet, the Gastrointestinal Microbiota, and Human Health
- 2:15 p.m. – 2:20 p.m.** **Outstanding Faculty Award Presentation**
- 2:30 p.m. – 3:00 p.m.** **Data Blitz Session**
Monsanto Room, ACES Library
- 3:15 p.m. – 3:45 p.m.** **Industry Panel & Discussion**
Bevier Commons
 Sponsors, presenters, DNS students, faculty, and staff are invited
- *4:00 p.m. – 5:00 p.m.** **Keynote Address by Dr. Thomas N. Seyfried**
180 Bevier Hall
**“Cancer as a mitochondrial metabolic disease:
 Implications for novel therapeutics”**
- 5:00 p.m. – 5:15 p.m.** **Break**
- *5:15 p.m. – 6:40 p.m.** **Graduate Student Poster Session**
Heritage Room, ACES Library
 Evening Reception, Award Announcements
 Sponsors, presenters, DNS students, faculty, and staff are invited

* Open to the general public

NUTRITIONAL SCIENCES GRADUATE STUDENT ASSOCIATION

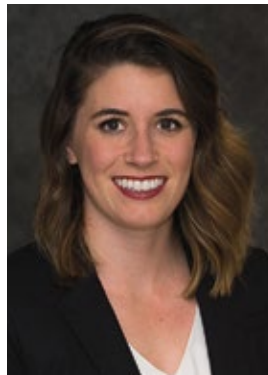
The Nutritional Sciences Graduate Student Association (NSGSA) was founded in the spring of 1973 by students in the program. The purpose of the organization is to provide a means of communication among graduate students, faculty, and alumni of the Division of Nutritional Sciences (DNS) which spans multiple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

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

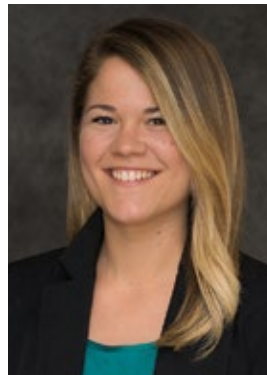
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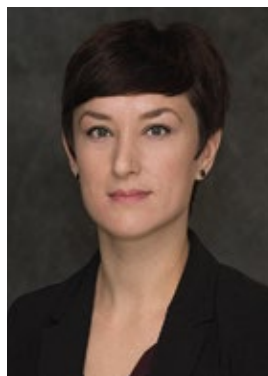
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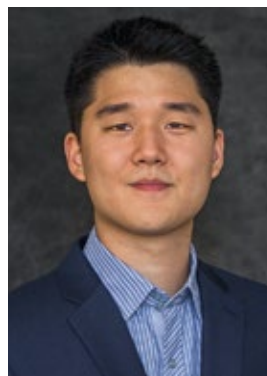
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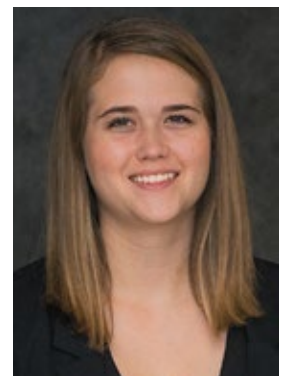
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SESSION JUDGES

Oral Session 1

Dr. Diana Grigsby-Toussaint
Dr. CheMyong Ko
Dr. Blair Rowitz

Oral Session 2

Dr. Maria Cattai de Godoy
Dr. Andrew J. Steelman
Dr. Kenneth Wilund

Poster Session

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Dr. Margarita Teran-Garcia
Dr. Jaime Amengual Terrasa



Samples labeled and prepped at the Nutrition Human and Microbiome Laboratory.

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Nutritional Sciences Graduate Student Association

<https://nutrsci.illinois.edu/students/gsa>

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Spanish Rice Bowl from the PATH Study and the UIUC Metabolic Kitchen.

SYMPOSIUM CONTRIBUTERS

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 2018 Nutrition Symposium.

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and Human Nutrition

University of Illinois
Department of Human Development
and Family Studies

Graduate Student Oral Presentations Session 1

9:15 a.m. - 10:15 a.m.
Monsanto Room, ACES Library

- Comorbid iron deficiency and respiratory infection interact to alter neurochemistry and neurodevelopment in piglets
Brian J. Leyshon..... 17
- Cooked and raw broccoli are both effective at protecting against dextran sulfate sodium-induced colon colitis in mice
Yanling Wang 17
- The use of avocado meal as a novel dietary fiber source in a canine diet
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- CHCHD2 knockout alters mitochondrial metabolism, increases sensitivity to sulfasalazine, and decreases proliferation and invasive potential of glioblastoma cells expressing EGFRvIII
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Graduate Student Oral Presentations Session 2

10:30 a.m. - 11:30 a.m.
Monsanto Room, ACES Library

- Retinal morphology and macular xanthophylls: Implications for intellectual ability
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- Requirement for digestible calcium at different dietary concentrations of digestible phosphorus indicated by growth performance and bone ash of 50- to 85-kg pigs
Vanessa Lagos 20
- Alpha-tocopherol transfer protein (α TTP) is differentially expressed in the cerebellum and occipital cortex of infant rhesus macaques
Katherine M. Ranard 21
- Epigenetic control of cortical estrogen receptor (Er)- α is altered by early-life BPA and high-fat diet exposure differentially by sex
Diego Hernández-Saavedra.....22

Faculty Mini-Symposium:

“Metabolic Alterations of Disease: What Role Can Nutrition Play?”

12:45 p.m. - 2:15 p.m.
Monsanto Room, ACES Library

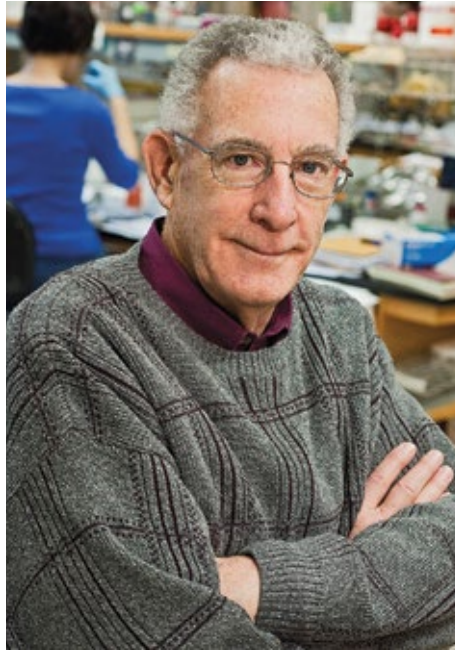
- Tomato, Lycopene and Prostate Cancer: Molecular Targets
Dr. John W. Erdman Jr. 14
- Microbial Sulfur Metabolism and Colorectal Cancer Risk
Dr. H. Rex Gaskins..... 15
- Diet, the Gastrointestinal Microbiota, and Human Health
Dr. Hannah Holscher 16

Graduate Student Poster Session

5:15 p.m. – 6:40 p.m.
Heritage Room, ACES Library

See poster session floor plan on page 37.

Development of a novel model of cholecystectomy in intact and ovariectomized mice and the impact on parameters of metabolism and gastrointestinal health – a pilot study Celeste Alexander	23	Irritable bowel syndrome and gastrointestinal issues in endurance athletes: prevalence and management strategies Lauren B. A. Killian	30
The impact of dietary tomato or lycopene on molecular outcomes of castration-resistant prostate cancer in the transgenic adenocarcinoma of the mouse prostate model Catherine C. Applegate	23	Anti-tumorigenic properties of omega-3 endocannabinoid epoxides Justin Kim	30
The presence of fiber-degrading bacteria in the gastrointestinal microbiome is associated with reduced whole-body inflammation Melisa A. Bailey	24	Evaluation of a community-based breastfeeding program in African American adolescent mothers on knowledge, attitudes, subjective norm, and perceived behavioral control Julia H. Kim	31
The relationship between adiposity and cognitive function in early childhood Corinne N. Cannavale	25	The effects of a yeast fermentation product on immune cell numbers and responsiveness of adult dogs Ching-Yen Lin	31
Herring roe oil alters circulating immune cell populations in a neonatal piglet model Megan P. Corbett	26	Hispanic mothers’ misperceptions of healthy weight: a barrier for childhood obesity prevention programs Viridiana Luna	32
Immune composition of mature human milk in exclusively breastfeeding and mixed-feeding mothers Erin C. Davis	26	An exploratory look at the role of childcare providers as a support and resource for breastfeeding mothers Alexandra Lundquist	33
You are what you didn’t eat: Cognitive processing in the fasted state amongst breakfast consumers and non-consumers Caitlyn G. Edwards	27	Six weeks of exercise improves markers of insulin sensitivity and metabolic endotoxemia: correlations with the gut microbiota Lucy J. Mailing	34
Polymorphisms in the reverse cholesterol transport pathway cumulatively affect triglyceride levels in young Mexican adults Bridget A. Hannon	28	Exploring the associations of non-osmotic tissue sodium accumulation to age and hypertension Luis Perez	34
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Dietary fatty acids and lipid profiles in a cohort of young adults Rebecca Jung	29	Microbial taxa differ by metabolic syndrome and hepatic health status among adults with overweight or obesity Sharon V. Thompson	36

**KEYNOTE SPEAKER:
Dr. Thomas N. Seyfried**

Thomas N. Seyfried is Professor of Biology at Boston College, and received his Ph.D. in Genetics and Biochemistry from the University of Illinois, Urbana, in 1976. He did his undergraduate work at the University of New England where he recently received the distinguished Alumni Achievement Award. He also holds a Master's degree in Genetics from Illinois State University, Normal, IL. Thomas Seyfried served with distinction in the United States Army's First Cavalry Division during the Vietnam War, and received numerous medals and commendations. He was a Postdoctoral Fellow in the Department of Neurology at the Yale University School of Medicine, and then served on the faculty as an Assistant Professor in Neurology. Other awards and honors have come from such diverse organizations as the American Oil Chemists Society, the National Institutes of Health, The American Society for Neurochemistry, and the Ketogenic Diet Special Interest Group of the American Epilepsy Society. Dr. Seyfried previously served as Chair, Scientific Advisory Committee for the National Tay-Sachs and Allied Diseases Association. He recently received a *Lifetime Achievement Award* from the Academy of



Complimentary and Integrative Medicine, and the *Uncompromising Science Award* from the American College of Nutrition for his work on cancer. He presently serves on several editorial boards, including those for *Nutrition & Metabolism*, *Neurochemical Research*, the *Journal of Lipid Research*, and *ASN Neuro*. Dr. Seyfried has over 180 peer reviewed publications and is author of the book, *Cancer as a Metabolic Disease: On the Origin, Management, and Prevention of Cancer* (Wiley Press). His full list of peer-reviewed publications can be found on PubMed (www.ncbi.nlm.nih.gov/pubmed/?term=Seyfried+TN).

“Cancer as a Mitochondrial Metabolic Disease: Implications for Novel Therapeutics”

Over 1,600 people die each day from cancer in the US according to recent data from the American Cancer Society. The failure to manage cancer has been due in large part to the dogmatic belief that cancer is a constellation of

genetic diseases. Accumulating evidence, however, indicates that cancer is primarily a mitochondrial metabolic disease involving disturbances in energy production through respiration and fermentation. The disturbances in tumor cell energy metabolism are linked to abnormalities in the structure and function of mitochondria that disrupt ATP synthesis through oxidative phosphorylation (OXPhos). Consequently, all cancer can be considered a single disease with a common pathophysiological mechanism involving dysfunction of mitochondrial OxPhos. The gene mutations observed in various cancers and all other recognized cancer hallmarks are considered downstream effects, and not causes, of the initial disturbance of cellular energy metabolism. Cancer growth and progression can be best managed following a whole-body transition from fermentable metabolites, primarily glucose and glutamine, to respiratory metabolites, primarily ketone bodies. Normal cells transition to ketone bodies for energy under low glucose conditions. Ketone body metabolism thus protects the brain against hypoglycemia. Tumor cells, on the other hand, cannot effectively use ketone bodies for energy due to their dysfunction in OxPhos. Therapeutic fasting and calorie restricted ketogenic diets lower cancer-provoking glucose and insulin-like growth factor (IGF-1) levels, while elevating ketone bodies. The metabolic transition from glucose to ketone bodies reduces tumor angiogenesis and inflammation while enhancing tumor cell apoptosis. The Press-Pulse therapeutic paradigm used with the Glucose/Ketone Index will facilitate the non-toxic management and prevention of cancer. As each individual is a unique metabolic entity, personalization of metabolic therapy as a broad-based cancer treatment and prevention strategy will require fine-tuning to match the therapy to an individual’s unique physiology. The efficacy of metabolic therapy for management of malignant cancer is seen in preclinical models and in humans with various cancers. It is anticipated that metabolic therapies targeting glucose and glutamine while increasing therapeutic ketosis will significantly improve quality of life and overall survival for most cancer patients.

Dr. Seyfried’s Keynote Address
4:00 – 5:00 p.m.
180 Bevier Hall

(Left) Study meals from a 7-week menu from the PATH Study and the UIUC Metabolic Kitchen.



FACULTY MINI-SYMPOSIUM:**“Metabolic Alterations of Disease: What Role Can Nutrition Play?”****ABSTRACTS AND BIOGRAPHIES****Tomato, Lycopene and Prostate Cancer: Molecular Targets****John W. Erdman Jr., Ph.D.***Department of Food Science and Human Nutrition; Division of Nutritional Sciences; Interdisciplinary Health Science Institute*

ABSTRACT: Prostate cancer (PCa) is the most diagnosed cancer and second cause of cancer-related deaths for American males. Our recent systematic review and meta-analyses of over 80 studies demonstrate that increased consumption of tomato and its primary bioactive, lycopene, are associated with reduced PCa incidence. A number of preclinical animal trials from our lab and others have consistently supported a protective role of dietary tomato, and often lycopene, in PCa disease occurrence or progression. Our recent studies have probed mechanisms whereby whole tomato or lycopene may reduce PCa risk. Our primary model is the TRAMP (transgenic adenocarcinoma of the mouse prostate) model. PCa is androgen dependent, especially at early stages of cancer. Our results suggest that dietary tomato (freeze-dried tomato paste) or supplemental lycopene reduces androgen status in the prostate. We also have evidence that lycopene metabolites (lycopenoids), rather than lycopene itself, might be most active against PCa. These compounds are similar in structure to retinoids, and may impact expression of numerous genes regulated by RARs and RXRs, including cell cycle, stress response, DNA repair and apoptosis genes. Recently, we have been evaluating the impact of tomato on castration-resistant PCa in the TRAMP model and have focused upon androgen receptor expression/localization, STAT3 phosphorylation, and their subsequent downstream activation of genes important for tumor invasion and metastasis. Overall, our evidence suggests that preventative effects of tomato and lycopene are partially mediated by their inhibition of androgen activity and co-regulators of androgen receptor activation.



BIOGRAPHY: Dr. Erdman is Emeritus Professor of Food Science and Human Nutrition at the University of Illinois at Urbana Champaign. He also is Deputy Director of the Interdisciplinary Health Sciences Institute on the Illinois campus. He has an active research program with 4 Ph.D. students. He has authored over 215 original research articles and over 350 total publications (H-Index is 52). He is a Fellow of the American Society for Nutrition (ASN), the Institute of Food Technologists (IFT) and the American Heart Association (AHA). He is past President of the American Society for Nutritional Sciences (now ASN). He has served on over two dozen committees for the Institute of Medicine, National Academy of Sciences (NAS). He chaired the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes (DRIs) and the Committee on Military Nutrition Research for NAS. He was elected as a Member of the Institute of Medicine (now National Academy of Medicine). He has received numerous honors for research, teaching and mentoring. His B.S., M.S., M.Phil. and Ph.D. are in Food Science from Rutgers University.

Microbial Sulfur Metabolism and Colorectal Cancer Risk

H. Rex Gaskins, Ph.D.

Departments of Animal Sciences and Pathobiology; Division of Nutritional Sciences

ABSTRACT: Despite the centrality of sulfur metabolism to most anaerobic microbial ecosystems, relatively little is known about the microbial assemblages and ecological constraints that contribute to microbial sulfur metabolism in the human colon. This gap in knowledge exists even with compelling evidence that bacterial-derived hydrogen sulfide is linked to prevalent human colonic disorders, namely inflammatory bowel disease (IBD) and colorectal cancer (CRC). Most of the attention given to the contribution of sulfidogenic microbes to colonic disorders has focused on sulfate-reducing bacteria (SRB), which are ubiquitously present in human colonic. Much less is known regarding the abundance of microbes capable of conserving energy through the utilization of organic sulfur sources in the human colon. We are examining the extent to which bacterial-derived hydrogen sulfide may serve as a proinflammatory and genotoxic insult that modifies colon cancer risk. Data will be presented which demonstrate that the human colonic mucosa is persistently colonized by bacteria capable of generating sulfide from both inorganic and organic sulfur sources along with evidence that sulfide activates molecular pathways that underlie epithelial inflammation and hyperplasia, a phenotype common to both ulcerative colitis and colorectal cancer. Published studies will also be summarized, which demonstrate direct free radical based genotoxicity by exogenous sulfide that is independent of host cell metabolism. As well, a recent study will be shared that implicates mucosal-resident sulfidogenic bacteria as an environmental risk factor contributing to CRC development particularly in African Americans.



BIOGRAPHY: H. Rex Gaskins joined the faculty at the University of Illinois at Urbana-Champaign in 1992 and is a professor with appointments in the Departments of Animal Sciences and Pathobiology, the Division of Nutritional Sciences and the Carl R. Woese Institute for Genomic Biology. He obtained the Ph.D. degree in nutritional sciences with an emphasis on cell biology from The University of Georgia in 1989. From 1989-92, he completed postdoctoral studies in immunology and genetics at The Jackson Laboratory in Bar Harbor, Maine. Research in his laboratory focuses on host-intestinal microbe interactions relevant to colorectal cancer with a particular interest in microbial sulfur and hydrogen metabolism. Efforts to understand colonic mucosal responses to hydrogen sulfide led to further interest in redox regulation of tumorigenesis resulting in a long-time collaboration with bioengineering faculty at Illinois to create genetically-encoded biosensors and engineered platforms for the study of redox poise in subcellular compartments in live cells. Professor Gaskins has authored 156 peer-reviewed publications and book chapters, and obtained as principal or co-investigator over \$13 million in grants during his tenure at Illinois. He has won numerous awards including a Future Leaders Award from the International Life Sciences Institute, the Bio-Serv Award from the American Society of Nutrition, a Burroughs Wellcome Fund visiting scientist fellowship (University of Reading), and a Sir Frederick McMaster CSIRO Research Fellowship (University of Queensland). From 1999-2002, he was named a University Scholar at Illinois, received in 2012 the Paul A. Funk Award from Illinois, and in 2016 received the Distinguished Scientist Award from the Society for Experimental Biology and Medicine. Professor Gaskins serves as Deputy Director of the NIH-supported Tissue Microenvironment Training Program and chairs the Steering Committee and serves as Associate Director of Education for the Cancer Center at Illinois.

Diet, the Gastrointestinal Microbiota, and Human Health

Hannah Holscher, Ph.D.

Department of Food Science and Human Nutrition; Division of Nutritional Sciences

ABSTRACT: The composition and metabolic function of the gastrointestinal microbiome are increasingly linked to metabolic health, and there is keen interest in developing evidence-based strategies to modulate the microbiome for human health benefit. Dietary components, such as fiber, which are resistant to digestion by human alimentary enzymes can be fermented by microbes in the gastrointestinal tract. Importantly, dietary fiber consumption is also linked to reduced risk of diseases including obesity, nonalcoholic fatty liver disease, and type 2 diabetes. As dietary fibers are heterogeneous, their impact on the microbiome will vary depending on factors including the botanical origin of the fiber and its physicochemical properties, as well as the treatment dosage and the phenotypic responses related to the composition of the individual's gastrointestinal microbiota. Fruits, vegetables, whole grains, and nuts, provide sources of dietary fiber. Indeed, our work has demonstrated the differential effects of consuming foods that contain dietary fiber, including almonds, walnuts, broccoli, and whole grains, on human gastrointestinal microbes and microbially derived metabolites. Our work has demonstrated that it is not only what you eat, but also when you eat that impacts the microbiota—variations in the human gastrointestinal microbiota occur throughout the day and eating behaviors, such as eating frequency and early energy consumption, are also related to the composition and function of gastrointestinal microbes. In addition to advancing knowledge on the impact of diet and eating behaviors on the gastrointestinal microbiome, our work has revealed relations between markers of human health status, including metabolic syndrome, hepatic steatosis, and systemic inflammation, and gastrointestinal microbes and microbial-derived fermentation end products. Much work remains to delineate the interrelationships between the gastrointestinal microbiome and human health; however, it is clear that diet is a contributing factor to these relationships.



BIOGRAPHY: Dr. Hannah Holscher is an assistant professor of nutrition in the Department of Food Science and Human Nutrition, and a member of the Division of Nutritional Sciences and the Institute of Genomic Biology at the University of Illinois, where she has been a faculty member since 2015. Before joining the faculty, she completed postdoctoral training focused on the human microbiome, as well as a Ph.D. in Nutritional Sciences and a B.S. in Food Science and Human Nutrition at the University of Illinois. She is also a Registered Dietitian. Research in Dr. Holscher's laboratory, the Nutrition and Human Microbiome Laboratory, integrates the areas of nutrition, gastrointestinal physiology, and the microbiome. Her research focuses on the clinical application of nutritional sciences with an overarching goal of improving human health through dietary modulation of the gastrointestinal microbiome. In addition to publishing in top nutrition journals, she also actively disseminates research findings in formats ranging from scientific presentations and webinars to podcasts, Twitter chats, blogs, and popular press particles. Dr. Holscher was recently recognized as a 2017 New Innovator in Food and Agricultural Research. She has received grant funding from the United States Department of Agriculture, the Foundation for Food and Agriculture Research, and industry. She has also served in local and national leadership roles, including her current position as the chair-elect of the Nutrition Translation Research Interest Section of the American Society for Nutrition.

Graduate Student Oral Session Abstracts

Oral Session 1

Comorbid iron deficiency and respiratory infection interact to alter neurochemistry and neurodevelopment in piglets

Brian J. Leyshon¹, M.A. Lawson², R.W. Johnson^{1,2}

¹Division of Nutritional Sciences, University of Illinois at Urbana-Champaign, Urbana, IL; ²Department of Animal Sciences, University of Illinois at Urbana-Champaign, Urbana, IL

OBJECTIVE: Since iron deficiency (ID) often presents with immunodeficiency, this study used a neonatal piglet model to investigate negative impacts on neurodevelopment and neuroinflammation caused by concurrent postnatal ID and respiratory infection. We hypothesized that ID would reduce immune activation and cytokine expression, reduce expression of genes associated with neurodevelopment, and impair monoamine metabolism.

METHODS: On postnatal day 2 (PD 2) piglets were divided into four groups (N = 30), and fed either iron normal (N) or ID sow milk replacer. Piglets were inoculated with either vehicle or porcine reproductive and respiratory syndrome virus (PRRSV) on PD 8. Rectal temperatures, feeding score and sickness behaviors were measured daily until piglets were harvested on PD 28. Tissues were collected to measure monoamine levels, cell isolation, and gene expression.

RESULTS: Hematocrit, hemoglobin, and serum iron were reduced by ID ($p < 0.0001$) but not PRRSV infection. PRRSV-infected piglets displayed viremia on PD 14, though PRRSV-ID viremia remained consistent while PRRSV-N piglets had reduced viremia PD 28 ($p = 0.0074$). Expression of iron metabolism, cytokine, and anti-microbial genes in peripheral and brain tissue was altered differentially across tissues by main effects of diet and infection as well as interactions. Microglial activation was increased by infection ($p < 0.0001$) but unaffected by ID, as was microglial cytokine expression, though hippocampal TNF α expression was increased by infection and further by an interaction with ID diet ($p < 0.0001$, $p = 0.048$). Brain weight was reduced by PRRSV infection but not ID. Hippocampal brain-derived neurotrophic factor (BDNF) expression was reduced by ID ($p = 0.030$). Dopamine metabolism was profoundly altered in key brain regions including the hippocampus, medial prefrontal cortex, striatum and amygdala due to ID, infection, and interaction of ID with PRRSV infection.

CONCLUSIONS: ID altered cytokine expression and reduced anti-microbial gene expression, impairing infection clearance. Reduced hippocampal BDNF may contribute to impaired learning and memory observed in ID animals. Altered dopamine metabolism may contribute to increased fear and anxiety and reduced stress resilience reported with postnatal ID and infection.

Cooked and raw broccoli are both effective at protecting against dextran sulfate sodium-induced colon colitis in mice

Yanling Wang¹, Y. Wu², M.A. Wallig^{3,4}, E.H. Jeffery^{1,4}

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OBJECTIVES: Broccoli is reported to have anti-inflammation properties. Raw broccoli was used in most past studies, whereas most consumers prefer cooked broccoli. This study compares effectiveness of lightly cooked broccoli (CB) with greatly diminished myrosinase activity and raw broccoli (RB), in mitigating colitis in dextran sulfate sodium (DSS)-treated mice.

METHODS: Male C57BL/6 mice were fed for 2 weeks on a 10% RB, 10% CB or control diet, all based on the AIN-93M diet. On day 8, mice (n=9) from half of each group received drinking water or 2.5% DSS in water to induce colitis. The disease activity index (DAI), including weight loss, fecal bleeding and solid stool formation, was scored daily. On day 15 mice were killed and gut barrier function, pro-inflammatory cytokines and histopathology of middle colon were assessed.

RESULTS: Both RB and CB decreased the DAI, attenuating weight loss, reducing fecal bleeding and improving solid stool formation, compared to that of DSS-control mice. RB and CB also mitigated the shrinkage of colon length induced by DSS. Moreover, histological evaluation of colon from both RB and CB showed less erosion and active inflammation, with signs of regeneration. Plasma lipopolysaccharide (LPS), indicative of failed colonic barrier function, was lower in both RB and CB compared to DSS mice not receiving broccoli. Decreased urinary levels of orally ingested sucralose suggested a less compromised gut barrier in mice receiving RB, but not in those receiving CB. These data suggest RB and CB improved gut barrier function in DSS-treated mice, but that RB was more effective than CB. Expression of the pro-inflammatory cytokines IL-6 and CCR2 typically caused by DSS was diminished only in RB mice. Expression of VCAM-1, however, was decreased in both RB and CB mice. CCR2 and VCAM-1 are involved in the IL-6 trans-signaling pathway and transition from acute to chronic inflammation. These data are consistent with dietary broccoli decreasing the IL-6 trans-signaling pathway and halting transition to chronic inflammation.

CONCLUSIONS: Our data suggest that CB, while not as effective as RB, was able to mitigate the clinical symptoms of colonic colitis in a DSS mouse model.

The use of avocado meal as a novel dietary fiber source in a canine diet

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ABSTRACT: The growing human and pet populations frequently compete for food ingredients and has sparked interest in identifying new sustainable and healthy sources. Often, by-products of the human food industry find their way into pet foods. The objective of this experiment was to evaluate the effects of avocado meal (AMD), an underutilized by-product of the avocado oil processing industry, on macronutrient apparent total tract digestibility (ATTD) and fecal fermentative end-products when compared to beet pulp (BPD) and cellulose (CD), fiber sources commonly used in pet food formulations. Nine intact female beagles (4.9 ± 0.6 yr, 11.98 ± 1.76 kg) were randomly assigned in a replicated 3 x 3 Latin square design. Experimental periods were 14 days long with 10 days of diet adaptation followed by 4 days of total fecal and urine collection for macronutrient apparent total tract digestibility (ATTD) calculations. All fecal samples were scored on a 5-point scale (1 = hard, dry feces; 5 = pourable, liquid diarrhea), with the ideal score between 2.5 and 3. One fresh fecal sample was analyzed for fermentative end-products. Dogs were fed to maintain body weight, which was monitored weekly. All animal procedures were approved by the University of Illinois Institutional Animal Care and Use Committee. Beet pulp (87.0 g/d) had higher (P < 0.05) daily fecal output on an as-is basis than AMD (62.3 g/d) and CD (58.0 g/d), but not on a dry matter (DM) basis. Fecal score for BPD (3.1) was greater (P < 0.05) than AMD (2.8) and CD (2.6). Fecal pH was greater (P < 0.05) for CD (6.2) than AMD (5.9) and BPD (5.5). Dry matter, organic matter, and energy ATTD were not affected (P > 0.05) by the treatments. Acid-hydrolyzed fat ATTD was lower (P < 0.05) for BPD (94.1%) than AMD (95.5%) and CD (95.7%). A greater (P < 0.05) crude protein ATTD was seen for CD (88.5%) in contrast to AMD (82.2%) and BPD (83.7%). Total-short chain fatty acids, acetate, and propionate for AMD (351.0, 233.4, and 70.9 μmole/g DM, respectively) were similar (P > 0.05) to CD (219.0, 132.9, and 61.7 μmole/g DM, respectively), with BPD (672.7, 480.5, and 138.0 μmole/g DM, respectively) greater (P < 0.05) than both. Butyrate concentration for CD (24.7 μmole/g DM) was lower (P < 0.05) than AMD (70.9 μmole/g DM) and BPD (138.0 μmole/g DM). Levels of isovalerate, ammonia, and total phenols and indoles were lower (P < 0.05) for BPD (5.6, 90.7, and 2.9 μmole/g DM, respectively), but similar (P > 0.05) between AMD (8.8, 109.3, and 12.1

µmole/g DM, respectively) and CD (7.5, 132.9, and 9.9 µmole/g DM, respectively) not different. Differences across dietary treatments were seen in serum chemistry profiles; however, these results were within reference ranges for adult dogs and all animals remained healthy throughout the experimental study. Given these results, avocado meal appears to be an acceptable dietary fiber source for adult canine diets. Avocado meal exhibited comparable results to beet pulp and cellulose, which are both fiber sources commonly used in formulations for the pet food industry, in terms of ATTD and fermentation profile and showed no indications of poor health.

CHCHD2 knockout alters mitochondrial metabolism, increases sensitivity to sulfasalazine, and decreases proliferation and invasive potential of glioblastoma cells expressing EGFRvIII

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ABSTRACT: Glioblastoma (GBM) is the most common and malignant form of brain cancer in adults, with a poor median survival time of 14.6 months driven by hyperproliferative zones and diffusively invasive cells. Tumors are heterogeneous and characterized by hypoxic foci. Amplification, over-expression, and mutation of the receptor tyrosine kinase (RTK) EGFR is prevalent in GBM, and the constitutively active EGFRvIII mutant shows the poorest clinical outcomes. However, RTK inhibitors have failed clinically against GBM. We are investigating compensatory signaling pathways active within GBM tumor margins as a possible source of resistance to molecular subtype therapies. Previous studies have reported coiled-coil-helix-coiled-coil-helix domain-containing protein 2 (CHCHD2) as a bi-organellar protein localized predominantly in the mitochondrial inter-membrane space but also present in the nucleus, where it acts as a transcription factor. We hypothesized that this co-localization enables GBM cells to adapt to hypoxic stress in the tumor microenvironment, particularly via mitochondrial retrograde signaling. Our previous work demonstrated that U87 GBM cells harboring EGFRvIII (U87vIII) displayed nuclei enriched with CHCHD2 compared to isogenic U87 cells not expressing EGFRvIII. The objective of this study was to characterize the effect of CHCHD2 knockout (KO) on mitochondrial metabolism, therapeutic sensitivity, and proliferation and invasion of U87vIII cells. Stable U87vIII CHCHD2^{KO} cells were derived using CRISPR-Cas9 genome engineering, and knockout was verified at the gene and protein level. CHCHD2^{KO} cells displayed decreased basal and maximal oxygen consumption rate and decreased spare respiratory capacity compared to U87vIII CHCHD2^{WT}, determined using the Seahorse XFp Extracellular Flux Analyzer. U87vIII CHCHD2^{KO} cell growth was significantly decreased over 72 h in both normoxia (21% O₂) and hypoxia (1% O₂). Mitochondrially targeted, genetically encoded fluorescent redox biosensors revealed decreased levels of oxidized GSSG in the mitochondrial matrix of CHCHD2^{KO} cells. Further, CHCHD2^{KO} cells were more sensitive to treatment with the drug sulfasalazine, an inhibitor of the glutamate-cystine antiport system x_c⁻. We next used engineered gliomas (EG) formed by microfluidic templating as a model of the GBM microenvironment. A three-dimensional invasion assay within these EGs revealed significantly increased invasion of U87vIII cells in hypoxia compared to normoxia, but this effect was abrogated in CHCHD2^{KO} cells, which displayed decreased invasion compared to U87vIII under both oxygen tensions. Collectively, these data demonstrate the multifaceted nature of CHCHD2 in the context of cell metabolism, proliferation, invasion, and therapeutic resistance, all outcomes which promote the malignancy and recurrence of GBM. Future work will further interrogate the function of CHCHD2 in the nucleus and the gene expression signature it may regulate in GBM.

Graduate Student Oral Session Abstracts

Oral Session 2

Retinal morphology and macular xanthophylls: implications for intellectual ability

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OBJECTIVES: Macular xanthophyll (i.e., lutein and zeaxanthin) accumulation in the retina – measured as macular pigment optical density (MPOD) – is recognized as a biomarker for multiple domains of cognitive function. On the other hand, neuroimaging studies have established that retinal structure or morphology corresponds to brain white matter and gray matter, and exhibits susceptibility to neurodegeneration. However, MPOD and retinal morphometric measures are seldom examined together. Accordingly, this study aimed to delineate the relationships between MPOD and retinal morphometric measures and intellectual ability (IQ) among adults with overweight and obesity.

METHODS: Adults between 25-45 years ($N=53$, 17 males) with overweight or obesity (≥ 25.0 kg/m²) underwent Optical Coherence Tomography to assess retinal morphometric measures including total macular (TMV) volume, retinal nerve fiber layer (RNFL) volume, ganglion cell layer (GCL) volume, and central foveal thickness (CFT). MPOD was assessed via customized hetero-flicker photometry. Dual Energy X-Ray Absorptiometry and 7-d diet records were used to assess whole body adiposity (%Fat), and dietary lutein and zeaxanthin intake (LZ), respectively.

RESULTS: Initial bivariate correlations indicated that IQ was associated with %Fat ($r=-0.25$, $p=0.04$), and positively with sex ($r=0.30$, $p\leq 0.01$), MPOD ($r=0.26$, $p=0.03$), TMV ($r=0.23$, $p\leq 0.05$), RNFL volume ($r=0.35$, $p\leq 0.01$), GCL volume ($r=0.26$, $p=0.03$), and CFT ($r=0.33$, $p\leq 0.01$). However, LZ intake was not significantly related to IQ ($r=0.20$, $p=0.16$). Using regression modeling, following adjustment for sex and %Fat, only CFT ($\beta=0.28$, $R^2=0.17$, $p=0.03$) and RNFL ($\beta=0.33$, $R^2=0.20$, $p\leq 0.01$) were independent predictors of IQ. MPOD ($\beta=0.21$, $R^2=0.14$, $p=0.06$) and GCL ($\beta=0.23$, $R^2=0.14$, $p=0.06$) approached statistical significance.

CONCLUSIONS: Our data suggest that macular xanthophylls and retinal morphology are markers of intellectual ability in adults. Future work is necessary to continue bridging the gap in the literature relating retinal structural and pigmentation measures to cognitive health.

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Requirement for digestible calcium at different dietary concentrations of digestible phosphorus indicated by growth performance and bone ash of 50- to 85-kg pigs

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ABSTRACT: An experiment was conducted to determine the requirement for standardized total tract digestible (STTD) Ca by 50 to 85 kg pigs and to test the hypothesis that the requirement to maximize growth performance expressed as STTD Ca:STTD P ratio is less than 1.35:1. Fifteen

corn-soybean meal based diets were formulated using a 3 × 5 factorial design. Diets contained 0.14, 0.27, or 0.41% STTD P and 0.13, 0.25, 0.38, 0.50, or 0.63% STTD Ca. Ninety barrows (initial BW: 50.2 ± 2.1 kg) were individually housed and randomly allotted to the 15 diets. Pigs were fed experimental diets for 30 d and the amount of feed offered was recorded. At the conclusion of the experiment, pig weights were recorded and average daily gain (ADG), average daily feed intake (ADFI), and gain to feed (G:F) were calculated for each diet. On d 31, pigs were euthanized and the right femur was removed. The concentrations and percentage of ash, Ca, and P were determined in the dried-defatted femurs. Data were analyzed using the second-order response surface model in NLREG by removing the terms in the model that were not significant ($P > 0.10$). Results indicated that there was an interaction ($P < 0.05$) between dietary concentrations of STTD Ca and STTD P for ADG, G:F, and the concentration (g per femur) and percentage of bone ash, bone Ca, and bone P. The model to predict ADG was reduced because the quadratic terms for STTD Ca and STTD P were not significant and for G:F, the model only included the linear terms for STTD Ca and STTD P, and the interaction between the linear terms. Both models contained a negative linear effect of STTD Ca ($P < 0.05$). The predicted maximum ADG at STTD P concentrations of 0.14 and 0.27% were 1.07 and 1.19 kg. These values were obtained at STTD Ca:STTD P ratios of 1.64:1 and 1.15:1, respectively. However, a predicted maximum G:F was not obtained because of the linear nature of the equation. A decrease in ADFI (main effect of Ca, $P < 0.05$) regardless of the concentration of STTD P in the diets was observed as the concentration of STTD Ca increased. In the model to predict bone ash (g) the quadratic term of STTD P was removed and the estimated maximum value at STTD P concentrations of 0.27 and 0.41% were 54.7 and 59.8 g. These values were obtained at STTD Ca:STTD P ratios of 1.98:1 and 1.50:1, respectively. Maximum ADG and bone ash in g per femur values at STTD P concentration of 0.41 and 0.14%, respectively, were not estimated because of the nature of the responses. Only small differences among treatments were observed for bone ash as a percent of the dried defatted bone, which indicates that bone tissue synthesis primarily is regulated by changing the size of the bone. In conclusion, excess Ca is detrimental to growth performance of 50- to 85-kg pigs if the concentration of P is at or below the requirement, but if the concentration of dietary P is above the requirement, increasing concentration of Ca increases growth performance. The amount of STTD Ca needed to maximize bone ash is greater than the amount needed to maximize growth performance and the STTD Ca to STTD P ratio needed to assure adequate bone mineralization without affecting growth performance is around 1.20:1 if the concentration of P is at the requirement.

Alpha-tocopherol transfer protein (α TTP) is differentially expressed in the cerebellum and occipital cortex of infant rhesus macaques

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BACKGROUND: Alpha-tocopherol (α T) is critical for an infant's developing central nervous system. Most infant formulas are fortified with synthetic (all-racemic) α T, but naturally-sourced (RRR) α T preferentially accumulates in human breast milk and infant brain tissues. Hepatic α T transfer protein (α TTP) facilitates secretion of α T into lipoproteins and discriminates in favor of RRR- α T. This partially explains the selective accumulation of RRR- α T in tissues, but tissue-specific expression of binding proteins like α TTP may also affect α T deposition. The expression of α TTP was previously reported in the adult cerebellum, but expression in regions of the infant brain is unknown.

OBJECTIVES: To compare α TTP expression in the cerebellum (CB) and occipital cortex (OC) of infant rhesus macaques. Secondly, to determine the effect of dietary α T source (all-racemic or RRR) on α TTP expression and α T accumulation.

METHODS: Infant rhesus macaques were fed one of two formulas (containing either RRR- α T or all-racemic- α T) or were breastfed for 6 months (n = 8, 7, 8). α TTP protein expression and α TTP mRNA expression were determined by Western blots and qPCR, respectively. Total α T concentrations and α T stereoisomer profiles were measured via HPLC.

RESULTS: α TTP protein expression was significantly higher in the CB than the OC ($p=0.019$); this was supported by mRNA expression results ($p=0.0069$). α TTP expression was not significantly modulated by dietary α T source. Across all diet groups, total α T accumulation was significantly higher in the OC than the CB ($p=0.020$), with no significant differences between groups. RRR- α T was consistently the predominant stereoisomer across brain regions and diet groups, constituting ~92%, ~71%, and ~38% of the α T in RRR- α T-fed, all-racemic- α T-fed, and breastfed monkey brains, respectively.

CONCLUSIONS: α TTP expression, but not total α T levels, were higher in the CB than the OC of infant rhesus macaques. Dietary α T source did not affect α TTP expression. There is a biological preference for the RRR stereoisomer of α T in the infant brain.

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Epigenetic control of cortical estrogen receptor (*Er*)- α is altered by early-life BPA and high-fat diet exposure differentially by sex

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BACKGROUND: Early-life exposure to high-fat diet (HF) and endocrine disruptors such as Bisphenol A (BPA) directly impact behavior and metabolism partly by altering the epigenetic landscape. The prefrontal cortex (PFC) is of importance given its vital role in executive function, memory and intelligence. Behavioral alterations seen in animals exposed to BPA or HF at early developmental stages might be explained by changes in Estrogen Receptor (*Er*)- α in PFC, which could impact estrogen sensitivity later in life. Objective: To evaluate the genetic and epigenetic effect of early-life BPA and/or HF exposure on *Er*- α in PFC of male and female rats.

METHODS: From gestational day 2 through parturition, female Long Evans hooded dams were assigned to either a control (CON, 15.8% Kcal fat) or HF diet (HF, 45% Kcal fat). Concurrently, BPA doses were administered at 0, 40, 400 μ g BPA/kg BW in a cookie to the dams, or via oral dosing to the pups until postnatal day (P) 10. After P10, animals consumed CON diet with no additional BPA dosing. At P10 and P90, offspring animals were sacrificed and the prefrontal cortex was collected for mRNA, DNA methylation and microRNA analysis.

RESULTS: Overall, *Er*- α expression in PFC was increased by HF diet ($p=0.03$) and BPA (400 μ g, $p=0.04$); at P10, *Er*- α was increased in females ($p=0.05$) and 400 BPA dose ($p=0.001$), whereas at P90, HF diet increased *Er*- α ($p=0.03$) and males with 400 BPA dose decreased *Er*- α ($p=0.03$). Analysis of DNA methylation patterns of four regions (1-4) of *Er*- α promoter revealed a decrease in region 1 for females consuming HF diet ($p=0.014$), and a decrease in region 4 by HF diet ($p=0.049$); However, no active transcription changes were detected by *Er*- α premRNA expression. Identification of microRNA through database mining retrieved 3 conserved miRNAs that target *Er*- α (mir-19b-3p, mir-221/222-3p, mir-22-3p), which showed significant Sex*BPA and BPA*miRNA interactions with *Er*- α expression.

CONCLUSION: Early-life exposure to BPA or HF diet regulated gene expression of *Er*- α in PFC in a sex-dependent manner, not by altering its DNA methylation pattern, but by inducing the expression of small microRNAs that target *Er*- α for degradation that could alter executive function and cognition later in life.

Graduate Student Poster Session Abstracts

Development of a novel model of cholecystectomy in intact and ovariectomized mice and the impact on parameters of metabolism and gastrointestinal health - a pilot study

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OBJECTIVES: Cholecystectomy (XGB), the removal of the gallbladder due to disease, is the most common abdominal surgery performed in the United States. Individuals can survive without a gallbladder, but have an increased prevalence of metabolic syndrome, non-alcoholic fatty liver disease, bile reflux, and insulin resistance. Age (40-50 years old) and sex (female) are two main risks factors for XGB, corresponding with the average onset of menopause in women. Since it is known that post-menopausal estrogen loss exacerbates metabolic dysfunction in women without XGB, the objectives of this pilot study were to: 1) establish a novel model of XGB in intact (SHM) and ovariectomized (OVX) mice, and 2) assess longitudinal effects of loss of ovarian function on metabolism, inflammation, and gastrointestinal (GI) health in XGB mice.

METHODS: Eight-wk-old female C57BL/6J mice (N=48) were fed a high-fat (45% kcal), low-sucrose (7% kcal) diet for the entire study (24 wk). BW and food intake were measured weekly. All mice were XGB at wk 0 and either OVX or SHM at wk 6. Body composition was analyzed every 6 wk with EchoMRI. Every 6 wk, 4-8 mice were sacrificed from each group. At sacrifice, serum was collected for lipid profiling and the GI tract, liver, and adipose depots were collected for histomorphology, histopathology, and gene expression analysis.

RESULTS: Preliminary analysis revealed a significant ($p < 0.01$) treatment x wk interaction for BW, body composition, and food intake, with XGB/OVX mice having higher BW at wk 11-23 and increased % fat mass and decreased % lean mass compared to XGB/SHM mice. XGB/OVX mice had higher ($p < 0.05$) hepatic triglyceride (TG) content at wk 24, however blind hepatic lipodosis scoring revealed no significant differences between groups. At wk 12, XGB/OVX mice had greater ($p < 0.05$) subcutaneous and mesenteric fat mass than XGB/SHM mice. Gonadal fat mass tended to be higher ($p < 0.10$) in XGB/OVX mice at wk 12 and 24. Serum total cholesterol tended to be higher ($p < 0.10$) in XGB/OVX mice at wk 12 and serum TG tended to be higher ($p < 0.10$) in XGB/SHM mice at wk 24.

SUMMARY: Findings suggest that loss of ovarian function following XGB results in an increase in fat mass, hepatic steatosis, and serum total cholesterol.

The impact of dietary tomato or lycopene on molecular outcomes of castration-resistant prostate cancer in the transgenic adenocarcinoma of the mouse prostate model

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OBJECTIVE: The objective of this study is to determine the effects of dietary tomato or lycopene on the tumor microenvironment of castration-resistant prostate cancer (CRPC). We hypothesize that both tomato and lycopene will result in less aggressive tumor phenotypes as defined

by reduced expression of the neuroendocrine (NE) phenotype, lower expression of the androgen receptor (AR) and its variants, decreased activation of AR-associated STAT3, and a resultant decrease in the expression of several downstream growth and metastasis-related proteins.

METHODS: The transgenic adenocarcinoma of the mouse prostate (TRAMP) model was used to obtain tissues for this study. Mice were separated into three dietary groups (n=30/group) and fed powdered, modified AIN-93G diets containing 10% tomato powder (TP), lycopene beadlets matched for lycopene content of TP (LYCO), or placebo beadlets (CON). Mice were castrated at 12 weeks of age to mimic the effects of androgen deprivation therapy (ADT) leading to CRPC. Tumor growth was monitored by ultrasound scan bi-weekly until tumor detection. At tumor detection, mice were scanned weekly for four additional weeks (total of five consecutive scans) and euthanized. Prostate and tumor tissues were collected and prepared for histology or snap frozen for analysis. Tumor tissues will be analyzed via histology for the presence of the NE phenotype and AR localization. Tumor DNA will be extracted and sequenced for detection of AR variants; mRNA and protein will be extracted for measurement of STAT3 and downstream targets of both AR and STAT3.

RESULTS: Previous data have shown decreased expression of NE-related genes in TRAMP mice after lycopene supplementation. Preliminary data from our laboratory have shown that tomato feeding slowed the growth of CRPC tumors, reduced expression of the AR, and inhibited STAT3 activation in tumor tissues. We expect that TP and LYCO will result in reduced expression of tumor biomarkers, with TP having greater effects on inhibition of these aggressive molecular indicators than LYCO.

CONCLUSIONS: This work will determine whether tomato and/or lycopene will demonstrate growth inhibitory effects and whether these effects are the result of inhibition of tumor phenotypic changes, AR-mediated signaling, or pathways related to growth and metastasis.

The presence of fiber-degrading bacteria in the gastrointestinal microbiome is associated with reduced whole-body inflammation

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BACKGROUND: High-fiber diets have been associated with decreased systemic inflammation and decreased risk of metabolic disease. This reduction in inflammation may be due to mechanisms which involve the gastrointestinal (GI) microbiota. Diets high in dietary fiber promote the growth of fiber-degrading bacteria, like *Rumminococcus* and *Prevotella*, that ferment dietary fiber and produce short chain fatty acids, which have systemic anti-inflammatory effects.

OBJECTIVE: We assessed cross-sectional relationships between diet, the GI microbiota, and markers of inflammation in healthy adults of varying BMI classes.

METHODS: Blood and fecal samples were collected from adults (n=56, 27 males; 25-45 years of age; BMI \geq 18.5) without physician-diagnosed metabolic or GI diseases. Dietary intake patterns were assessed using the Diet History Questionnaire (DHQII, Past Year and Past Month with Portion Size) and Healthy Eating Index (HEI-2010) scores were calculated to assess diet quality. Following DNA isolation, bacterial (V4 region of 16S rRNA) amplicons were generated using a Fluidigm Access Array followed by high-throughput sequencing on an Illumina MiSeq. Sequences were quality-filtered then operational taxonomic units were picked against the Greengenes 13-8 references database using QIIME version 2.0. Plasma C-reactive Protein (CRP) and interleukin (IL)-6 levels were measured by enzyme-linked immunosorbent assay. Data were analyzed using SAS 9.4.

RESULTS: Bivariate correlations revealed a negative association between the genera *Rumminococcus* and plasma CRP ($r=-0.49$, $p<0.01$) and a trending negative association between *Rumminococcus* and plasma IL-6 ($r=-0.22$, $p=0.09$). However, partial correlations revealed that these relationships were mediated by BMI. *Rumminococcus bromii*, a keystone

species for the degradation of resistant starch, trended toward a negative association with plasma CRP ($r=0.36$, $p=0.05$). *Prevotella spp.* were negatively associated with plasma IL-6 ($r=-0.29$, $p=0.03$) and positively associated with whole fruit ($r=0.23$, $p=0.03$) and whole grain ($r=0.22$, $p=0.03$) HEI-2010 components. There were no associations between diet quality scores and inflammatory markers.

CONCLUSIONS: Whole fruit and whole grain consumption is associated with the presence of fiber-degrading bacteria in the GI tract and these bacteria are associated with reduced whole-body inflammation as measured by plasma CRP and IL-6 concentrations. Positive correlations between BMI and inflammatory markers indicate the importance of research of dietary strategies that may reduce inflammation via modulation of the GI microbiota. Future directions include assessment of short-chain fatty acids, additional inflammatory cytokines, and pro-inflammatory monocyte activity to better understand the interconnections between the GI microbiota, habitual diet, and human health.

The relationship between adiposity and cognitive function in early childhood

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OBJECTIVE: Converging evidence indicates that excess adiposity during preadolescence has negative implications for children's cognitive function. However, there is limited data on whether or not this relationship is evident earlier in childhood. Further, patterns of adipose tissue accumulation differ based on sex and emerge early in life. Therefore, variances in adipose tissue accumulation may differentially influence physical and cognitive health throughout development. Accordingly, this study aimed to investigate relationships between sex, whole body adiposity and fat distribution, and cognitive function among 4 and 5-year-olds.

METHODS: Four and five-year-olds ($N=39$, 21 females) were recruited from the East-Central Illinois area. Participants underwent adiposity measures and cognitive assessment through the use of anthropometrics, dual-energy X-ray absorptiometry, and Woodcock-Johnson IV Test of Early Cognitive and Academic Development (WCJ) respectively. Cognitive function was evaluated using the WCJ subscales of expressive language and early academic achievement.

RESULTS: While there was no significant difference between sexes for BMI ($p=0.67$), females were found to have greater whole-body %fat ($p=0.032$) and abdominal subcutaneous fat ($p<0.01$), while males had more visceral fat ($p<0.01$), consistent with patterns of fat distribution observed in adulthood. Negative correlations (all r 's between -0.67 and -0.44) were found in females between early academic skills and expressive language assessments and all aforementioned adiposity measures (all p 's <0.03). In contrast, there were no significant adiposity-cognition relationships among males (all p 's >0.13).

CONCLUSION: Our findings indicate that, in females, adiposity is inversely related to cognitive function in early childhood. Given the previous literature in older children indicating similar results in both sexes, these data suggest that the negative implications of adiposity for cognitive function may emerge earlier in females. Future longitudinal and interventional studies are necessary to confirm this relationship.

Herring roe oil alters circulating immune cell populations in a neonatal piglet model

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OBJECTIVES: The objective of this study was to determine if dietary supplementation with omega-3 fatty acid DHA in the neonatal period could reduce neuroinflammation during an inflammatory immune response. **Methods:** Full term (114 days), average birth weight (1.2-1.8 kg), naturally delivered piglets (N = 14) were obtained from the University of Illinois Swine Farms on postnatal day (PD) 2 and weaned onto a liquid milk replacer diet containing herring roe oil (HRO) as a source of DHA (2 g/kg diet providing 40 mg/kg body weight/d DHA) or an isocaloric control (CON). On PD14, sample collection for complete blood cell counts, cell culture, gene expression, and fatty acid analysis occurred. CD11b+ microglial cells from the brain were isolated and stimulated ex vivo with 10 ng/mL lipopolysaccharide (LPS).

RESULTS: Piglets fed the HRO diet had a lower (p = 0.0256) white blood cell count on PD14 than CON piglets. Piglets on HRO diet also had shifted populations of neutrophils and lymphocytes, while monocyte populations were not different between groups (p = 0.16). HRO piglets had a lower number of neutrophils (p = 0.0114), a higher number of lymphocytes (p = 0.0483), and a lower neutrophil/lymphocyte ratio (NLCR; p = 0.0107). The brain tissue of piglets fed HRO contained more DHA than CON piglets (p = 0.0292). Gene expression analysis of the LPS stimulated CD11b+ cells showed no differences in the inflammatory response of the cells from HRO diet piglets versus the control diet. LPS stimulation increased gene expression of TNF, IL-6, and IL-1 β (p < 0.0001). There was no effect of dietary treatment on TNF (p = 0.52), IL-6 (p = 0.84), or IL-1 β (p = 0.93) gene expression. There was no effect of diet on the expression of anti-inflammatory markers IL-10 (p = 0.29) and IL1-RN (p = 0.16).

CONCLUSIONS: Based on the data above, it does not appear that twice the WHO recommended dosage of dietary DHA for infants (40 mg/kg/day) can attenuate the inflammatory response when microglia from the brain are isolated and stimulated ex vivo, however, results are pending for a mild in vivo immune stimulation. The lower NLCR of the HRO group suggests that HRO supplementation may have had a positive effect on piglet health.

Immune composition of mature human milk in exclusively breastfeeding and mixed-feeding mothers

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OBJECTIVE: Human milk (HM) is composed of a diverse profile of immune components, which are believed to influence infant innate and adaptive immunity. Factors such as maternal age, geography, gestational age, and lactation stage influence immune composition. However, the breadth of cytokines, chemokines, and growth factors (CCGFs) present in HM is not well-characterized, and whether breastfeeding exclusivity affects CCGF composition is unknown. The objective was to measure CCGFs in mature HM and assess differences in composition between exclusively breastfeeding (EBF) and mixed-feeding (MF) mothers.

METHODS: Milk samples (EBF=44; MF=25) collected at 6 weeks postpartum from healthy mothers enrolled in the STRONG Kids 2 birth cohort study were utilized for analysis. All mothers vaginally-delivered their infants at term. Samples were analyzed for 41 CCGFs using a human cytokine/chemokine multiplex magnetic bead panel. **Results:** Forty components were detectable in the milk samples. Twelve CCGFs (EGF, TGF- α , GRO, MDC, PDGF-AA, IL-15, IL-4, IL-7, IL-8, IP-10, MCP-1, VEGF) were identified in more than 94% of samples. TNF- α , Fractalkine, and IL-1 α were detected in more than 78% of samples. Among this subset of 15 analytes, IL-8 was the only

CCGF, within limits of detection (LOD), which differed between groups, with greater concentrations in the milk of MF relative to EBF mothers ($p=0.002$). EGF and GRO were above the LOD (10,000 pg/ml) in most samples. Fisher exact tests were used to determine differences in frequency of detection for all CCGFs between EBF and MF mothers. The probability of detecting Fractalkine was greater among EBF compared to MF mothers ($p=0.04$). The probability of detecting IL-3 ($p=0.009$), IL-6 ($p=0.02$), IL-9 ($p=0.02$), and MIP-1 β ($p=0.006$) was greater among MF compared to EBF mothers.

CONCLUSION: While levels of many CCGFs have been shown to be higher in early lactation, results demonstrate that a wide range of immune components are present at detectable levels in mature HM. The high frequency of detection of a subset of CCGFs may point to a core immune composition in mature HM. Differences in CCGF components in HM of EBF and MF mothers suggests that breastfeeding exclusivity impacts the immune profile of HM, which may be related to differences in oral microbiota of EBF and MF infants.

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You are what you didn't eat: Cognitive processing in the fasted state amongst breakfast consumers and non-consumers

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OBJECTIVES: Cognitive control comprises a set of mental operations vital for the regulation of goal-directed behaviors. The use of event-related potentials (ERPs) to assess neurophysiologic activity provides insight into the effects of nutrition on the neural underpinnings of cognitive control. Through acute feeding studies, ERPs have been shown to be susceptible to the fasted vs. non-fasted metabolic state. Despite these findings, metabolic state is seldom accounted for. This is concerning, as acute meal consumption may account for differences observed in studies using the ERP technique. Recording ERPs in the fasted state has the potential to moderate these confounds. However, the acute effects of fasting on ERPs are not well known. Accordingly, the current study aimed to delineate differences in neuroelectric function between breakfast consumers vs. non-consumers following an overnight fast.

METHODS: Habitual meal patterns were assessed using 7-d food records from adults ($N=62$) between 25-45 years. Individuals who did not report any intake between 4am-10am for at least 6 of the 7 days were categorized as breakfast non-consumers ($n=28$). A comparator group of breakfast consumers ($n=28$) was matched for age, sex, and body mass index. Attentional and inhibitory control were assessed using two-stimulus visual Oddball and Nogo tasks, respectively. ERPs were recorded to examine amplitude and latency of the N2 and P3 waveforms using sensor FCZ and a central-parietal region of interest.

RESULTS: Kruskal-Wallis analyses revealed no behavioral (accuracy and reaction time) differences between groups in the Oddball, nor Nogo task (all p 's >0.17). In the Oddball task, no group differences were observed in N2 or P3 peak amplitude ($H=0.02$, $p=0.90$; $H=1.03$, $p=0.31$) nor latency ($H=1.32$, $p=0.25$; $H=0.78$, $p=0.38$). Similarly, in the Nogo Task no group differences were observed in the N2 or P3 peak amplitude ($H=2.79$, $p=0.10$; $H=1.24$, $p=0.27$) nor latency ($H=1.08$, $p=0.30$; $H=0.20$, $p=0.66$).

CONCLUSIONS: These results indicate that regular breakfast consumers and non-consumers exhibit similar behavioral and neuroelectric patterns under fasted states during morning testing appointments. These results provide preliminary support for a methodological alternative to the confounding use of non-fasted ERP testing sessions.

Polymorphisms in the reverse cholesterol transport pathway cumulatively affect triglyceride levels in young mexican adults

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ATP-binding cassette 1 (ABCA1) and cholesterol-ester transfer protein (CETP) are proteins in the reverse cholesterol transport (RCT) pathway, which facilitates cholesterol transport to the liver via high-density lipoprotein cholesterol (HDL) particles. Genetic variants, in the form of single-nucleotide polymorphisms (SNPs) may affect the efficiency of this process, resulting in low HDL and high triglyceride (TG) levels. Diet also influences these biomarkers, specifically carbohydrates (CHO) and saturated fat (SFA). Nutrigenetic interactions between diet and variants in RCT may contribute to the development of dyslipidemia. We aim to determine how cumulative risk of multiple SNPs in this pathway relates to diet and lipoprotein levels.

Subjects were Mexican young adults (ages 18-25, n=649, 53% female) from the UPAMIGOS cohort study. Fasting blood was collected for genetic and lipoprotein analysis. Dietary intake was also collected, and CHO and SFA were expressed as percentage of total calories. SNPs from *ABCA1* (rs9282541 and rs4149310) and *CETP* (rs1532624, rs289714, and rs5882) were selected based on their classification as functional variants. Genotypes were assigned 0, 1, or 2, corresponding with the number of risk alleles for each SNP. Cumulative risk was calculated from the sum of all alleles. Generalized linear models were adjusted for age, sex, and BMI.

SNPs were not in linkage disequilibrium and had a minor allele frequency greater than 10%. Cumulative genetic risk was associated with higher TG (P=0.001, R²=0.20). The mean number of risk alleles was 3.9. Individuals with 4 or more risk alleles had greater levels of TG compared to those with lower risk (115.1 vs. 105.6 mg/dL; P=0.001). CHO intake, as a percentage of calories, was associated with high TG levels in the cumulative risk model (β = 0.19, P=0.025). SFA intake was not found to be significantly predictive of TG levels.

In this cohort, common variants involved in RCT cumulatively influenced TG levels. TG levels were also affected by CHO, but not SFA intake. CHO promote high TG levels by increasing hepatic lipogenesis, and multiple variants in RCT may result in accumulation of TG-rich lipoproteins. This study provides novel information on the aggregate effects of pathway-specific variants and diet on TG in an understudied population.

Distribution of an oral dose of ¹³C-lutein into tissues of an adult rhesus macaque: a pilot study

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OBJECTIVE: Lutein selectively accumulates in primate retina and brain where it may have an essential role in ocular and cognitive health. To date, no isotopic tracer studies of lutein pharmacokinetics have been performed in a nonhuman primate model. The objective of this pilot study was to assess the distribution of lutein into tissues of an adult rhesus macaque (*Macaca mulatta*) after a single oral dose of isotopically-labeled lutein.

METHODS: One 19-year-old female macaque was supplemented daily for a year with 1 μ mol/kg/

day of unlabeled lutein and subsequently provided a single dose of 1.92 mg of ^{13}C -lutein. 65% of lutein was uniformly labeled while 35% remained unlabeled at 1 or 2 carbons. Plasma, six brain regions, retina, and other tissues were collected on the third day post-dose. Tissues from an undosed macaque were used as negative controls. Lutein accumulation was quantified by HPLC-PDA and ^{13}C enrichment was evaluated by LC-QTOF-MS.

RESULTS: In the control monkey, ^{12}C -lutein, but not ^{13}C -lutein, was detectable in serum and all tissues examined. In the ^{13}C -dosed macaque, the highest ratio of ^{13}C -lutein/ ^{12}C -lutein ($^{13}\text{C}/^{12}\text{C}$) was found in the plasma (11.3%), followed by liver (10.6%), heart (6.2%), kidney (6.1%), adrenal gland (3.5%), and quadriceps (0.7%). ^{13}C -lutein was detected in the brain and accumulated differentially across regions. The $^{13}\text{C}/^{12}\text{C}$ ratio was highest in the temporal cortex (2.3%) and lowest in the subcortical white matter (0.6%). However, ^{13}C -lutein was undetectable in macular and peripheral retina, despite high concentrations of ^{12}C -lutein. Among adipose depots, ^{13}C -lutein was detectable in abdominal subcutaneous and axillary brown adipose tissues ($^{13}\text{C}/^{12}\text{C}$ = 0.1% and 0.5%, respectively), but not in the thigh subcutaneous and mesenteric adipose tissues.

CONCLUSION: Three days following dosing, ^{13}C -lutein was differentially distributed across various tissues, including multiple brain regions, but undetectable in the retina. This pilot study demonstrates that distribution of lutein in the macaque is substantially dependent on tissue type. Studies with additional time points are needed to determine if these observations indicate differences in the kinetics of tissue uptake, metabolism, or recycling into circulation.

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Dietary fatty acids and lipid profiles in a cohort of young adults

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BACKGROUND: Current dietary recommendations from organizations such as the American Heart Association focus on limiting intake of saturated (SFA) and trans fatty acids, and concurrently increasing intake of monounsaturated (MUFA) and polyunsaturated (PUFA) fat. This is due to substantial evidence linking high SFA intake with increased risk of obesity and cardiovascular disease (CVD). Additionally, the associations between dietary fat intake and cholesterol levels, and major CVD risk factors in younger adults without CVD are less studied. Therefore, the objective of this study was to determine associations between dietary fat intake and cholesterol levels in a sample of young adults (ages 18-35).

METHODS: Adults were recruited to participate in a cross-sectional study. Participants provided a fasting blood sample for lipid analysis and reported dietary intake via food-frequency questionnaire. Dietary fat intake was expressed as a percentage of total calories. Regression analysis was used to determine associations between different types of fatty acids and lipid cholesterol levels, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL), and high-density lipoprotein cholesterol (HDL).

RESULTS: Seventy individuals (35 females), with an average age of 25.6 (SD 5.1) participated in this study. Saturated fat intake was significantly associated with TC levels ($P=0.02$, $\beta=0.04$), and LDL levels ($P=0.04$, $\beta=0.05$). The average SFA intake was 10.4% of total calories (SD 3.4). PUFA intake was significantly associated with higher HDL levels ($P=0.03$, $\beta=0.07$). MUFA intake was not associated with any outcomes.

CONCLUSION: Changes in modern diet has drastically increased the prevalence of obesity, CVD, and related comorbidities, highlighting the crucial need for both preventative and treatment interventions. Subsequent findings in this project will aid in developing preventative practices. In our sample of young adults, increase in SFA intake was associated with elevated TC and LDL levels. Additional work is warranted in this population to determine causative effects of specific fatty acids on CVD risk factors.

Irritable bowel syndrome and gastrointestinal issues in endurance athletes: prevalence and management strategies

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BACKGROUND: Gastrointestinal (GI) issues are known to be common among endurance athletes and can impair performance in training and competition. Symptom characteristics, particularly those of the lower GI, are similar to irritable bowel syndrome (IBS). No previous research has examined IBS diagnosis or fit to IBS diagnostic criteria within this population.

PURPOSE: To determine the prevalence of IBS among endurance athletes as well as their GI symptom management strategies.

METHODS: A 93-item online questionnaire was previously validated for the purpose of assessing IBS diagnosis, fit to IBS diagnostic criteria (Rome III or Manning), general GI symptoms, and symptom mitigation strategies of endurance athletes. The questionnaire was distributed between December 2015 and January 2017 to the athletes in the U.S. completing a marathon, ultra-marathon, half-distance triathlon, or full-distance triathlon within that calendar year.

RESULTS: The total prevalence of irritable bowel syndrome among 430 endurance athletes who completed the questionnaire was 22.8% using both Rome III and ≥ 2 Manning criteria, with only 2.8% medically diagnosed. Only 13.5% of athletes reported seeing a medical professional due to GI issues, while 18.6% had issues which sometimes or often interrupted or prevented their training. Additionally, 68.1% and 55.8% experienced at least one lower GI symptom at a frequency of sometimes or more during training and competition, respectively. Almost half (45.8%) of the athletes tried nutritional modifications to help ease their symptoms and 21.6% used over-the-counter medications.

CONCLUSION: Most endurance athletes that may suffer from IBS are undiagnosed, while even more have GI issues but do not fit IBS diagnostic criteria. Despite using various methods to manage their symptoms, endurance athletes are still experiencing issues and could potentially benefit from current IBS-mitigating strategies.

Anti-tumorigenic properties of omega-3 endocannabinoid epoxides

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ABSTRACT: Dietary omega-3 fatty acids such as docosahexaenoic acid (DHA) have demonstrated to suppress tumor growth due to its downstream lipid metabolites. DHA is converted to DHA endocannabinoids such as docosahexaenoyl ethanolamides (DHEA) that elicit similar effects as $\Delta 9$ -tetrahydrocannabinol (THC), the principal bioactive component of cannabis. DHEA possess anti-proliferative activity. DHA is also converted by CYP epoxygenases to produce DHA epoxides. DHA epoxides have shown to inhibit metastasis and tumor growth. As DHEA and DHA epoxides separately show anti-tumor activity, we report a novel class of dual functional compounds, DHEA-epoxide (EDP-EA), that contain both ethanolamide and epoxide moiety. As both pathways have been implicated in cancer, we evaluate the anti-tumorigenic properties of dual functional DHA derivatives in an osteosarcoma model. Thus, we investigate and report a novel class of endocannabinoid epoxides that exhibit anti-inflammatory, anti-angiogenic, anti-tumorigenic and anti-migratory properties. We also report the synthesis of stable analogs of these endogenous lipid metabolites that show enhances anti-metastatic properties.

Evaluation of a community-based breastfeeding program in African American adolescent mothers on knowledge, attitudes, subjective norm, and perceived behavioral control

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OBJECTIVE: To assess the impact of a newly-developed breastfeeding curriculum called Healthy And Proper Parenting for Youth (HAPPY).

METHODS: African American, adolescent mothers (n=7, M=18.9±1.1 years) who were mostly postpartum (children's age: M=2.4±1.3 years) and their mentors (n=12 M=38.4±9.4 years, children's age: M=10.6±8.2) were exposed to three one-hour breastfeeding education sessions. Using a pretest, posttest study design, knowledge was measured using the Infant Feeding Test, and attitudes, subjective norm and perceived behavioral control were measured using the modified Breastfeeding Attrition Prediction Tool. Significant differences between pretest and posttest were analyzed using paired t-test and log and square root transformation of non-normal variables and multiple imputation for missing data using SAS 9.4. Significance was determined at p value of 0.05.

RESULTS: Comparing those who attended all three workshops (n=14) to those who did not (n=5), there were no significant differences in knowledge, positive breastfeeding sentiment, negative breastfeeding sentiment, subjective norm, control, or intention to breastfeed (p>0.05). Participants who attended the SMART Goals Matter and You Matter workshop (n=10) had significant changes knowledge compared to participants (n=9) who did not attend both workshops (Mean change: 1.8 vs. -0.22; p=0.02). Participants who attended the Breastfeeding Matters workshop (n=7) did not have different scores in knowledge, positive breastfeeding sentiment, negative breastfeeding sentiment, subjective norm, or control compared to participants who did not attend (p>0.05). There was a trend for an increased odds of intention to breastfeed after exposure to the SMART Goals Matter and Breastfeeding Matters (p=0.059, 95% CI 0.016-1.085).

CONCLUSIONS: Attending two of three workshops increased breastfeeding knowledge, while there was a trend for increased intention to breastfeed their next child after attending the Breastfeeding Matters workshop. African American, adolescent mothers and their mentors may need more exposure to breastfeeding promotional efforts to observe significant changes in psychosocial antecedents to breastfeeding behavior.

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The effects of a yeast fermentation product on immune cell numbers and responsiveness of adult dogs

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ABSTRACT: Yeast products are commonly used in the pet food industry due to their nutraceutical properties. However, studies in yeast product supplementation and canine immunity are still limited. Thus, the aim of this study is to evaluate the effect of a yeast fermentation product on immune cell numbers and responsiveness of adult dogs. Twelve adult female beagles (age: 3.3 ± 0.8 yr, BW: 10.3 ± 0.68 kg) were fed the same diet but supplemented a yeast fermentation product with 3 levels (125, 250 and 500 mg) or a placebo via gelatin capsules in a replicated 4 x 4 Latin square design. Blood samples were collected after a 26-d yeast supplementation. Peripheral blood mononuclear cells (PBMC) were separated using Histopaque. One aliquot of PBMC (1x10⁶ cell/tube) was used for surface staining with cluster of differentiation marker antibodies for T cell, natural killer (NK) cell and antigen presenting (AP) cell (B cell and

monocyte) as well as major histocompatibility complex class II (MHC-II) antibodies for AP cells. Intracellular staining with INF- γ antibodies were conducted to investigate the population of INF- γ -secreting T cell. Cell populations were then determined using a flow cytometer. Another aliquot of PBMC was seeded into 96-well plates and incubated for 24 h with/without agonists of toll-like receptor (TLR) 2, TLR3, TLR4 and TLR7/8. Supernatant was collected for evaluating TNF- α concentration using an ELISA kit in order to study the responsiveness of lymphocytes to TLR agonists. Concentrations of serum oxidative stress markers (superoxide dismutase and malondialdehyde) and serum immunoglobulins (IgA, IgG, IgE, and IgM) were evaluated using ELISA kits. All data were analyzed using the Mixed Models procedure of SAS 9.4. Linear and quadratic effects as well as control vs. treatment effects were tested using contrasts. Cell populations, including T cell, NK cell and AP cell were not altered by treatment. However, when comparing control vs. all yeast treatments, dogs supplemented with yeast product had greater ($p < 0.05$) MHC-II presenting B cell and monocyte population than control dogs. TNF- α concentrations of control wells were not different among treatment groups. In cells stimulated with TLR2, TLR3, TLR4, and TLR7/8 agonists, an overall effect of controls vs. all yeast treatments was observed, with cells collected from dogs supplemented with yeast product having a lower ($p < 0.05$) TNF- α secretion than cells collected from control dogs. For cells stimulated with TLR4 agonist, dogs supplemented with 125 mg yeast product had lower ($P < 0.05$) TNF- α concentrations than control dogs, with dogs supplemented with 250 mg or 500 mg yeast product being intermediate. For cells stimulated with TLR2 and TLR7/8 agonists, dogs supplemented with 125 mg and 500 mg yeast product had lower ($P < 0.05$) TNF- α concentrations than control dogs, with dogs supplemented with 250 mg yeast product being intermediate. A linear effect was also observed for cells stimulated with TLR2 and TLR7/8 agonist, with TNF- α concentrations being reduced ($p < 0.05$) with increasing yeast dosage. Serum oxidative stress markers and immunoglobulins were not affected by treatment. A linear effect ($p < 0.05$) was observed in IgE, with higher yeast dosages leading to higher circulating IgE concentration. In conclusion, yeast supplementation decreased responsiveness of lymphocytes to TLR agonists but did not change immune cell numbers.

Hispanic mothers' misperceptions of healthy weight: A barrier for childhood obesity prevention programs

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ABSTRACT: Parents play an integral role in childhood obesity (OB) prevention. Hispanic parents' perceptions of child weight may be impacted by unique cultural beliefs regarding health. These beliefs may impact their recognition of an unhealthy weight. This study aimed to determine the percentage of Hispanic mothers that underestimate (UE) child weight and assess whether acculturation is associated with misperception and concern for child weight. Mother-child dyads ($n=109$) of Hispanic origin were recruited as part of the *Abriendo Caminos* workshop intervention. Cross-sectional data from participants in the Illinois and California sites were used. Acculturation was assessed with the Brief Acculturation Rating Scale for Mexican Americans-II (ARSMA-II). Level of concern for child weight was measured by Likert-scale. Anthropometric data were also collected. Mothers who accurately perceived child weight in a Likert-scale response question received a score of 0. Scores of -1 and +1 were assigned to mothers who UE and overestimated (OE) child weight, respectively. One and two-way ANOVA were used to compare differences in child weight misperception and the interaction between misperception and acculturation, respectively. Over half of children had overweight (OW) or OB (58.7%) based on measured BMI percentile. Only 36.1% of mothers expressed concern for their child's weight.

Twice as many mothers of children with OW as mothers of children with OB UE child weight (80.0% vs. 38.6%). Acculturation was not significantly associated with misperception or concern for child weight ($p>0.05$). Mothers who UE child weight and had low acculturation reported the lowest levels of concern compared to other groups ($p<0.05$). Mothers with low acculturation and reported not knowing child weight had the highest level of concern ($p<0.05$). There is a high level of child weight misperception among Hispanic mothers, especially among those with children with OW. Educating parents of children with OW about achieving a healthy weight can prompt them to make healthy changes. Given the high rate of obesity, an understanding of how perception and concern for child weight may influence health behaviors can aid professionals in providing culturally sensitive recommendations to Hispanic parents.

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An exploratory look at the role of childcare providers as a support and resource for breastfeeding mothers

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ABSTRACT: Research has examined workplace, healthcare, and family factors in support for the breastfeeding mother, whereas few studies have considered the available support in childcare settings. We hypothesized that the transition to non-parental care represents a vulnerable change for first-time mothers where increased support may be needed to facilitate the continuation of breastfeeding, and that mothers overlook childcare as a potential source of influence and support. Utilizing Bronfenbrenner's bioecological model as a theoretical framework for the development and evolution of the breastfeeding experience, a semi-structured interview was developed to guide data collection. First-time breastfeeding mothers (n=25) were recruited for interviews. Concepts and themes that comprise mothers' experiences with, and perceptions of, navigating the transition to childcare while breastfeeding were identified using thematic analysis. The resultant four key themes suggested that although mothers intended to continue breastfeeding beyond the transition to childcare, mothers did not consider the childcare setting to be either a barrier to or a source of support for breastfeeding continuation. Often, mothers responded in ways that indicated they had not previously given much consideration to the child care setting beyond that of a service provider. Mothers did not critically evaluate or prioritize childcare providers' ability to accommodate breastfeeding. Mothers introduced the idea that the primary way they felt supported by their provider was the provider doing what the mother wanted or needed them to do. Mothers conveyed that the pressure of trying to merge breastfeeding into the workplace and childcare settings within the constraints of societal values and ideologies was an overarching barrier they felt to breastfeeding continuation. Findings from this study offer new insight into how mothers perceive and interact with the childcare environment in regards to breastfeeding. The results strongly suggest that future policy initiatives should consider breastfeeding support within the childcare setting from a systems perspective - focusing on relationships or linkages between childcare settings and breastfeeding mothers rather than taking a top-down approach.

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Six weeks of exercise improves markers of insulin sensitivity and metabolic endotoxemia: correlations with the gut microbiota

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INTRODUCTION: Research from our laboratory indicates that six weeks of aerobic exercise alters the gut microbiota and microbial-derived short chain fatty acids (SCFAs) in both lean and obese humans. SCFAs directly modulate inflammation, insulin sensitivity and gut barrier function. Thus, the objectives of the present study were to (1) determine the effects of aerobic exercise training on circulating metabolic and inflammatory parameters indicative of inflammation, insulin sensitivity, and gut barrier function and (2) determine whether changes in these parameters paralleled shifts in the microbiota and its metabolites.

METHODS: Previously sedentary but otherwise healthy adults (n=16 lean; n=11 obese) underwent a six-week aerobic exercise intervention. Blood samples collected before and after the intervention were analyzed for C-reactive protein (CRP), lipopolysaccharide binding protein (LBP), and insulin resistance by the homeostatic model assessment (HOMA-IR). Fecal samples were analyzed for microbiota composition (16S rRNA gene sequencing) and SCFA concentrations (gas chromatography)

RESULTS: At baseline, obese individuals had significantly higher CRP, LBP, insulin, and HOMA-IR compared to lean individuals (p < 0.05). There were no changes in CRP as a result of exercise training. However, LBP and HOMA-IR were significantly reduced by exercise in the obese group (p < 0.05). Change in CRP over the 6-week intervention positively correlated with change in abundance of *Erysipelotrichaceae* (r = 0.610, p = 0.009), a microbe previously shown to be associated with metabolic syndrome. Change in abundance of *Anaerostipes*, a genus of known butyrate-producers, negatively correlated with change in LBP (r = -0.727, p = 0.007) and HOMA-IR (r = -0.471, p = 0.036). Both CRP and LBP levels after the intervention were negatively correlated with post fecal acetate, butyrate, and propionate levels (p < 0.01).

CONCLUSION: Six weeks of aerobic exercise improved markers of insulin sensitivity and metabolic endotoxemia in obese individuals. These improvements were related to changes in the gut microbiota, as metabolic and inflammatory markers correlated with changes in several important microbial genera and post-intervention SCFAs.

Exploring the associations of non-osmotic tissue sodium accumulation to age and hypertension

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BACKGROUND: Recent research utilizing sodium magnetic resonance imaging (MRI) in healthy individuals has demonstrated that dietary sodium (Na) may be transiently held non-osmotically in interstitial spaces of tissues before excretion several days or weeks later. Accumulation of this Na is not accompanied by commensurate changes in water, so it is likely that non-osmotic storage of Na plays a role in whole body salt-loading and buffering. This non-osmotic Na may also be implicated in the pathogenesis of hypertension (HTN) through inflammation and vasoconstriction pathways. Preliminary studies are also beginning to indicate that tissue Na is positively associated with age and hypertension. The purpose of this pilot study was to quantify tissue Na levels in healthy individuals and in HTN individuals.

METHODS: We recruited 21 subjects (47±19 years, 25.7±3.9 kg/m², 0% male, 0% white) from the University of Illinois. Six subjects were diagnosed with primary hypertension. Each subject arrived at the Beckman Biomedical Imaging Center for Na MRI scans and collection of the following: height, weight, standardized blood pressure, and a 48-hour dietary recall.

RESULTS: All subjects had a mean SBP of 125±18 and tissue Na of 24.4±0.52. The mean difference in SBP between healthy subjects and HTN individuals was significant (27±7, p<0.01). The mean difference in tissue Na between groups was also significant (5.4±2.2, p=0.03). There was a strong significant correlation of tissue Na by age (R=0.81, p<0.01) for all subjects. Follow-up regression of tissue Na by age for all subjects was also significant (R²=0.66, p<0.01) as well as for healthy subjects or HTN alone (p<0.01). However, multiple regression including categorical HTN status (healthy or HTN) was not significant (p=0.56). There was no significant correlations or relationships (p<0.05) of dietary Na intake to any parameter measured. A strong significant correlation was found between age and blood pressure (R=0.61, p<0.01) as well as between blood pressure and tissue Na (R=0.70, p<0.01) among all subjects.

CONCLUSIONS: Subjects diagnosed with HTN had a significantly higher standardized SBP compared to healthy individuals as expected, while HTN subjects had significantly elevated tissue Na. Age appears to be a good predictor of tissue Na storage for all individuals. In this preliminary data, it is still not yet clear that HTN status is valuable predictor of tissue Na. It will be important to continually incorporate more subjects in this preliminary research to develop stronger associations and conclusions. Further research is important to replicate these findings and expand upon additional health endpoints and biomarkers.

Dietary tomato or lycopene intake and the emergence of castration-resistant prostate cancer in the transgenic adenocarcinoma of the mouse prostate (TRAMP) cancer model

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OBJECTIVE: Epidemiological evidence suggests that tomato products or lycopene are associated with reduced prostate cancer (PCa) incidence, yet little information exists regarding the ability of tomatoes or its bioactive components to impact PCa therapy. Castration-resistant prostate cancer (CRPC) is the result of progression following androgen deprivation therapy (ADT). CRPC is an advanced phase of PCa in humans with a poor prognosis. In our present study, we hypothesized that dietary tomato or an equivalent concentration of lycopene following orchiectomy would reduce tumor burden and progression in transgenic adenocarcinoma of the mouse prostate (TRAMP) mice compared controls diet.

METHODS: To test this hypothesis, male TRAMP mice (n=90) were provided a powdered AIN-93G diet (BASE) until 12 weeks of age. To model the effects of ADT (reducing serum testosterone), mice were castrated at 12 weeks. After castration, animals consumed either BASE with placebo beadlets (Placebo, n=30), an AIN-93G diet modified to contain 10% lyophilized tomato paste (TP; n=30), or BASE fed lycopene at a concentration matched to TP (LYCO, n=30). Prostates of TRAMP mice were monitored by ultrasound for *in vivo* tumor detection and 3-D volumetric growth measurement. Following euthanasia, tissues are collected, and carotenoids are measured by high performance liquid chromatography (HPLC) in the tumor, serum, liver, and epididymal adipose tissue. Prostate tissue and all suspected metastatic sites are harvested, processed, stained and evaluated by histopathologic criteria.

RESULTS: This study will be the first to compare the effects of tomato powder and lycopene on PCa progression in the TRAMP model following castration. In a preliminary study we have observed that tomato powder following orchiectomy (ADT) was sufficient to reduce CRPC growth

rate and volume in this model. Accordingly, we hypothesize that TP and LYCO will both reduce CRPC tumor recurrence, CRPC tumor growth and extend tumor-free survival compared to Placebo, and that TP will have a greater reduction than LYCO due to the additional carotenoids that may accumulate in the tumor to protect against progression.

CONCLUSIONS: This study may provide preclinical data to support future studies of specific dietary interventions to enhance the efficacy of androgen deprivation in men with PCa.

Microbial taxa differ by metabolic syndrome and hepatic health status among adults with overweight or obesity

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BACKGROUND: Nonalcoholic fatty liver disease (NAFLD), a progressive condition typified by elevated hepatic lipid deposition, is a common co-morbidity of metabolic syndrome (MetS) despite its lack of inclusion in MetS diagnostic criteria. Emerging research has implicated microbial dysbiosis and dysfunction of the gut-microbiota-liver axis, the direct conduit for microbial influence on hepatic function via portal circulation, in NAFLD development.

OBJECTIVE: To concomitantly assess microbial taxa, MetS status, and hepatic lipid deposition among adults with overweight or obesity.

METHODS: We conducted a cross-sectional study among 92 adults between 25-45 years of age with BMI values ≥ 25.0 kg/m². MetS was evaluated as the presence of ≥ 3 risk factors using the International Diabetes Federation guidelines. Hepatic fat fraction was assessed using quantitative ultrasound; hepatic steatosis was defined as fat fraction $\geq 5\%$. Microbial analyses were conducted using 16S rRNA sequencing (V4 region) and QIIME version 2. Bivariate correlations were conducted to assess relationships between microbial taxa and health outcomes. Mann-Whitney U tests were performed to identify differences in microbial taxa abundances by presence or absence of MetS and hepatic steatosis.

RESULTS: Thirty-eight percent of participants presented with MetS, 18% with hepatic steatosis, and 11% with both conditions. At the phyla level, hepatic fat fraction was positively correlated with Proteobacteria ($\rho=0.32$, $p=0.01$) and the relative abundance of this phylum was over-represented among participants with hepatic steatosis ($p=0.02$), as were the genera *Proteus* ($p=0.03$) and bile tolerant *Sutterella* ($p=0.03$). Individuals with MetS had greater relative abundances of *Bilophila* ($p=0.04$).

CONCLUSIONS: The present study identified differences in microbial taxa abundances, including elevated representation of bacteria involved in fat and protein metabolism among adults with MetS and hepatic steatosis, providing evidence for a connection between the gut-microbiota-liver axis, diet, and chronic disease risk.

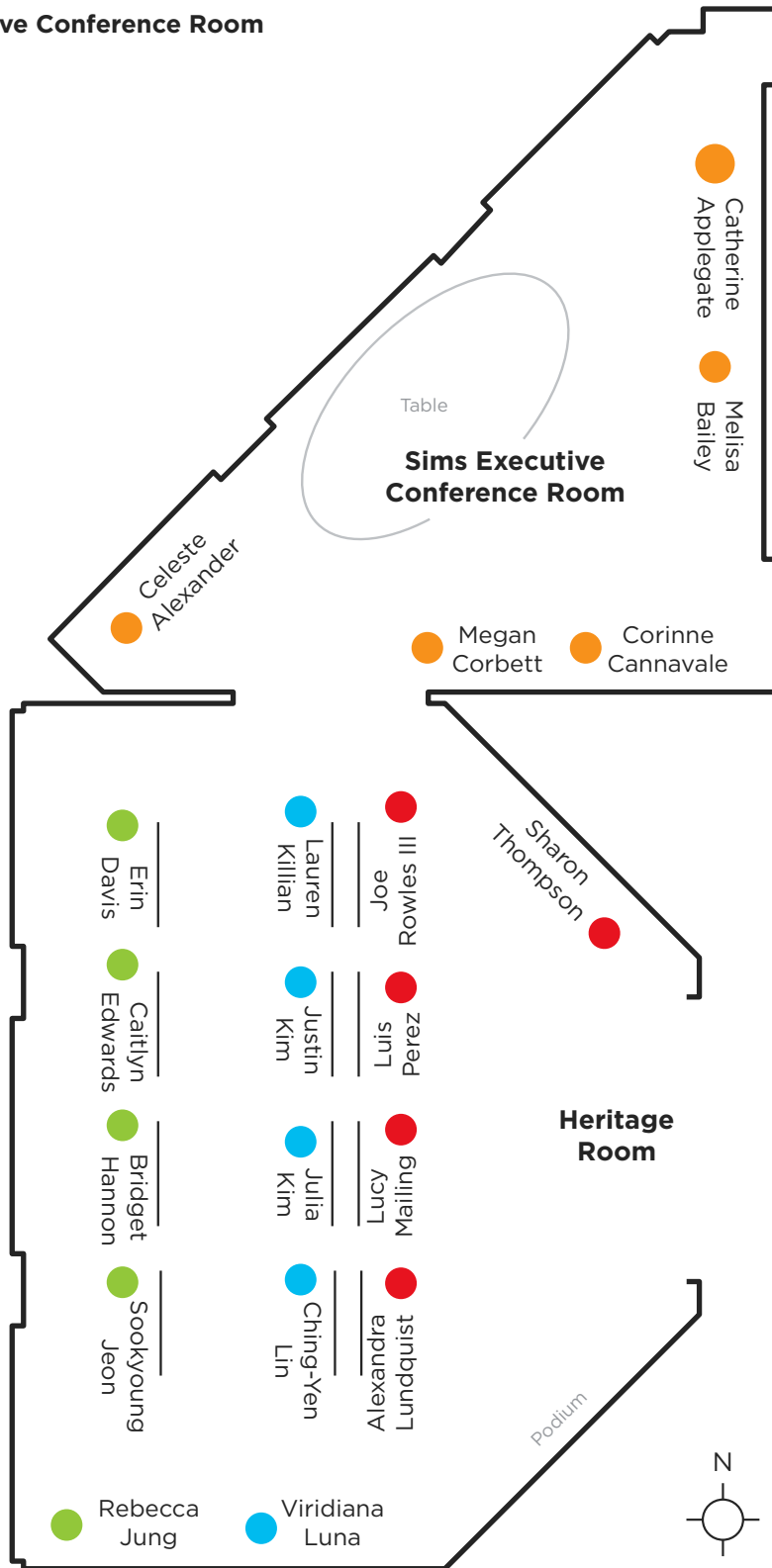
Graduate Student Poster Session Floor Plan

ACES Library, 1st Floor
Heritage Room and Sims Executive Conference Room
5:15 p.m. - 6:40 p.m.

Poster Judges

- **Orange Team**
Dr. Juan Loor
Dr. Marcia Siegel
Dr. Hans Stein*
- **Green Team**
Dr. Nu-Chu Liang
Dr. Kelly Swanson*
Dr. Jaime Amengual
Terrasa
- **Blue Team**
Dr. Anna Dilger*
Dr. Anna Keck
Dr. Elvira de Mejia
- **Red Team**
Dr. Sayeepriyadarshini
"Sayee" Anaak
Dr. Patricia Massae Oba
Dr. Margarita
Teran-Garcia*

* Team captain



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Winners of the 2017 University of Illinois Nutrition Symposium poster and oral competitions with keynote speaker, Dr. Teresa A. Davis. Photograph by L. Brian Stauffer.

