Obesity Research Cluster

5th Annual Meeting & Oral and Poster Competition

TTU Innovation Hub
8 May 2019
3:00-8:00 PM

Acknowledgments:
College of Human Sciences
Presidential Cluster Hire
OR&I and ORDC
ORC Advisory Committee
Dr. Debra Reed
BSI

For More Information About ORC Visit:
https://www.depts.ttu.edu/hs/obesityresearch/
5th Annual
Obesity Research Cluster (ORC) Meeting & Poster Competition

Obesity Science: An Integrative Look at Rural Health

3:00 - 3:20 Welcome & Introduction
Dr. Naima Moustaid-Moussa, Professor, Nutritional Sciences & Founding Director, ORC
Dr. Joseph A. Heppert, Vice President for Research

3:20 - 3:30 Dr. Naima Moustaid-Moussa
“ORC: Our Path Forward”

3:30 - 3:40 Speaker I: Introduced by: Dr. Sara Dodd, Associate Professor, HDFS
Dr. Angela Burkham, Interim State Program Leader & Regional Program Leader
Family Community Health at Texas A&M AgriLife Extension Service
“Growing a Healthy Texas”

3:40 - 3:50 Speaker II: Introduced by: Dr. Oak-Hee Park, Research Assistant Professor, COHS
Dr. Debra Flores, Managing Director-West Texas AHEC
TTUHSC, Center F. Marie Hall Institute for Rural & Community Health
“Understanding Obesity-Urban vs. Rural”

3:50 - 4:00 Speaker III: Introduced by: Dr. Jannette Dufour, Professor, Cell Biology & Biochemistry, TTUHSC
Dr. David Cistola, Professor & Director, Center of Emphasis in Diabetes & Metabolism
Paul L. Foster School of Medicine, TTUHSC EL Paso
“Teenagers – An Untapped Target Population for Preventing Type 2 Diabetes?”

4:00 - 4:10 Speaker IV: Introduced by: Dr. Naima Moustaid-Moussa
Dr. Kate Larson, Acting Research Leader & Nutrition Scientist
USDA ARS GFHNRC
“Epigenetics of Obesity”

4:10 - 4:30 COFFEE BREAK

4:30 - 4:55 Faculty 3 min Presentations – Introduced by Dr. Conrad Lyford, Professor, CASNR
Dr. Oak-Hee Park
Dr. Sukhbir Singh
Dr. Breanna Harris
Dr. Heather Vellers
Dr. Leslie Shen
Dr. Eric Rivas
Dr. Varuna Nargunan

4:55 - 5:05 Postdoc 3 min Presentations – Introduced by Dr. Andy Shin, Research Assistant Prof, COHS
Dr. Kembra Albracht-Schulte
Dr. Vipulkumar Patel
Dr. Jongkyoo Kim
5:05 - 5:15  **Student 3 min Presentations – Introduced by Dr. Stacy Carter, College of Education**
Makenzie Miller
Md Akheruzzaman
Mohammed Jodeiri

5:15 - 5:20  **Speaker V Dr. Kay Tindle: Managing Director, ORDC, ORIC, TTU**
“Convergence and the Art of Collaboration”

5:20 - 6:00  **Networking**
Facilitator: Cristal P. Sanchez, ORDC

6:00 - 6:05  **Closing Remarks: Dr. Naima Moustaid-Moussa**

6:05 - 8:00  **Poster Competition & Reception**

7:15  **Awards Announcement**

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**Faculty Presentations**

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**Naima Moustaid-Moussa**
Professor, Nutritional Sciences & Founding Director, ORI
Texas Tech University

Dr. Moustaid-Moussa is professor in Nutritional Sciences and Founding Director of the TTU Obesity Research Institute. She has been leading a funded basic and integrated nutrition and obesity research, with emphasis on the endocrine function of adipose tissue and nutrient-gene interactions in metabolic diseases. Her current research includes mechanisms by which (1) the adipocyte renin angiotensin system increases inflammation and insulin resistance in metabolic diseases and breast cancer and (2) bioactive compounds (such as omega 3 fatty acids, tocotrienols, tart cherry anthocyanins and other polyphenols) reduce obesity-associated white fat inflammation and activate brown fat, using cell and animal models of obesity and Alzheimer’s disease. Her secondary areas of interest include beta cell function in diabetes and childhood obesity prevention.

Dr. Moustaid-Moussa’s research is currently funded by federal agencies (NIH and USDA), international Foundations (Qatar Foundation) as well as industry. She has extensively published in top tier journals (over 120 papers) and served in leadership positions within the American Society for Nutrition (ASN), The Obesity Society (TOS), and the American Heart Association (AHA). She currently serves as a member of the NIH Clinical Integrative Diabetes and Obesity study section. Dr. Moustaid-Moussa is Fellow of AHA (FAHA) and Fellow of TOS (FTOS). Her scholarship and mentoring was recognized through several awards including the 2012 Outstanding Investigator award and 2015 Pfizer Consumer Healthcare Nutritional Sciences award, both from ASN; the 2018 Nancy J Bell Outstanding Mentor Award from the TTU Graduate School, and the 2019 Outstanding Faculty Mentor for Undergraduate Research from the TTU Center for Transformative Undergraduate Experiences.
Angela Burkham
Interim State Program Leader & Regional Program Leader
Family Community Health at Texas A&M AgriLife Extension Service

Dr. Burkham serves as the FCH Regional Program Leader for the North Region. She provides leadership for Family and Community Health programs with youth and adult audiences in the 41 North Region Counties which surround Amarillo and Lubbock. In her current role, Dr. Burkham leads program development, professional development, collaborations, partnerships and connectivity to academic departments and units in relation to county, district and regional based programs. She also coordinates the administrative leads for the Family and Community Health program in Texas through the Texas A&M AgriLife Extension Service.

Dr. Burkham holds a B.S. degree in Home Economics Education from Texas Tech University, a M.S. degree in Agriculture Education from Texas Tech University and a Doctorate of Education in Agriculture Education and Communications from Texas A&M University and Texas Tech University. She has been part of the Texas A&M AgriLife Extension Service team since 1991 in various roles including as a FCH agent, Extension Program Specialist 4-H, Regional Program Director 4-H and Youth, and an Assistant Professor and Extension Specialist.

Debra Flores
Dr. Debra Flores, Managing Director-West Texas AHEC
TTUHSC, Center F. Marie Hall Institute for Rural & Community Health

Dr. Flores has over 35 years of experience in the medical field specifically in nursing and public health. Her experiences range from working as a Licensed Vocational Nurse in an acute care setting to manager of state and federal programs at the community level and federally qualified health centers. She is the current managing director for West Texas AHEC and oversees the six centers which are located throughout West Texas in Canyon, Plainview, Midland, El Paso, Denton and the one here in Abilene. In addition to the AHEC federal funding, Dr. Flores is also the PI for the Behavioral Health Workforce grant received from HRSA. She is a certified CHW instructor, adjunct faculty for the MPH program at TTUHSC where she teaches “Issues in Rural Health”, is a recurring faculty for TTUHSC SON and teaches a section in the Rural and Global Health Certificate.

Dr. Flores is also a faculty affiliate for the Human Development and Family Studies Department at TTU. In addition to being a licensed vocational nurse, Dr. Flores has a B.S in Organizational Management from Lubbock Christian University, a M.A. in Management from Wayland Baptist University and Ph.D. in Education (curriculum and instruction) from TTU.
Faculty Presentations

David Cistola
Professor & Director, Center of Emphasis in Diabetes & Metabolism
Paul L. Foster School of Medicine, TTUHSC EL Paso

Dr. David Cistola is the Founding Director for the Center of Emphasis in Diabetes & Metabolism, a new diabetes research center at TTUHSC El Paso. He leads a medical research laboratory in clinical/translational science.

Current research areas include the development of new diagnostic and therapeutic approaches for diabetes, prediabetes, fatty liver disease, cardiovascular disease and Alzheimer’s disease. He previously served as Vice President for Research & Innovation at the University of North Texas Health Science Center, Fort Worth; Associate Dean for Research and Professor in the College of Allied Health Sciences and Professor in the Brody School of Medicine at East Carolina University, and as faculty at Washington University School of Medicine in St. Louis.

Dr. Cistola graduated from the M.D.-Ph.D. program at Boston University, and trained as an NIH Postdoctoral Fellow in the Cardiovascular Institute and held the Andrew Costello Fellowship of the Juvenile Diabetes Foundation International.

Kate Larson
Acting Research Leader & Nutrition Scientist
USDA ARS GFHNRC

Dr. Larson’s research career has focused on obesity-associated diabetes risk, inflammation and immune dysfunction, with emphasis on epigenetics of adipose tissue and placenta. Her highly innovative research constitutes a new approach to understand the metabolic basis for obesity-driven disease that integrates dietary interventions to reduce diabetes and obesity risk caused by parental diet and obesity-induced placental dysfunction. Dr. Larson’s contributions to this field have been recognized nationally and internationally through her extensive peer-reviewed publications in top journals in nutrition, obesity and immunology, and her service on various committees and leadership roles in the American Society of Nutrition (ASN); the Obesity Society and as organizer of national/international conferences, and as peer reviewer for USDA and NIH study sections. She has been a member of the Institute of Medicine Basic Science Agenda Subcommittee since 2012. She has received several USDA ARS certificates of Merit awards, ASN Normal Kretchmer Memorial Award in Nutrition and Development, and Korean Nutrition Society Award.
Faculty Presentations

**Oak-Hee Park**  
Research Assistant Professor  
College of Human Sciences, Texas Tech University

Oak-Hee Park has been conducting various research projects related to nutrition, food, health, and consumer behavior. Her current research areas are focusing on Nutrition Education, Food Environment, Public Health, Obesity Prevention, and Sustainable Food System. Since 2013, she has worked on the East Lubbock Promise Neighborhood (ELPN) Grant funded by the U.S. Department of Education. As Co-PI, she established the first community-based family cooking program for underserved populations. She also conducts a food environment study in the Lubbock area using the NEMS-S and NEMS-R surveys, and has actively led an obesity prevention project for early adolescents at Talkington School for Young Women Leaders to promote individual’s self-efficacy about healthy cooking that may encourage adolescents to reduce risky eating behavior, leveraging body acceptance and mindful eating practices for the prevention of obesity.

**Sukhbir Singh**  
Assistant Professor of Vegetable Production Systems  
Department of Plant and Soil Science, Texas Tech University

The long-term goal of my research program is to determine the parameters (varieties, agronomic practices, etc.) leading to sustainable and profitable vegetable production. The target market for this production is high-quality vegetables for mid to high-income consumers (including up-scale restaurants and hotels). To achieve this goal, my research will focus on several aspects of organic and conventional vegetable production in open field, high tunnels and hydroponics. Specifically, I will work on sensor-based irrigation, nutrient management, biotic and abiotic stresses physiology, disease and pest control, cropping systems, and soil fertility management.
Faculty Presentations

Breanna Harris
Research Assistant Professor
Department of Biological Sciences, Texas Tech University

Dr. Breanna Harris graduated with her B.S. in Marine Biology from Ohio University in 2005, and earned her Ph.D. in Evolution, Ecology, and Organismal Biology from the University of California, Riverside in 2012. After earning her Ph.D., she served as a visiting professor at Claremont McKenna College and an educator at University of California, Riverside. She joined TTU as a Teaching Postdoctoral Fellow working with Dr. Jim Carr in 2013. In 2014, Dr. Harris moved to the role of Research Assistant Professor and in 2015 she established her own lab in the Department of Biological Sciences. Dr. Harris is a behavioral endocrinologist and stress physiologist who studies how stressors influence organismal function, health, and life-history tradeoffs. Her current research program focuses on two central questions: 1) how does variation in response to and recovery from stressors translate into functional consequences in organismal behavior, cognition, risk-taking, feeding, health, life history trade-offs, and fitness, and 2) how do sensory perception, genotype, diet, sex, life history stage, and interactions with the environment alter physiological and behavioral responses to stressors. To date, she has authored over 25 peer-reviewed publications and has received funding from NIH and NSF.

Heather Vellers
Assistant Professor of Exercise Physiology
Department of Kinesiology and Sport Management, Texas Tech University

Heather Vellers is a Tenure-Track Assistant Professor of Exercise Physiology in the Department of Kinesiology and Sport Management. Prior to her new role as a faculty member at Texas Tech University (TTU), she was a Postdoctoral Research Fellow at the National Institute of Environmental Health Sciences (NIEHS) and completed her Ph.D. in Exercise Physiology at Texas A&M University. Dr. Vellers' research involves investigating the role of the mitochondrial genome with interindividual variations in adaptation to aerobic training. The broader application of her research agenda is to provide future exercise physiologists and healthcare providers a means to prescribe exercise programs that are individualized for each person (i.e. precision medicine); particularly for clinical populations.
Leslie Shen
Associate Professor of Pathology and Physiology
Texas Tech University Health Sciences Center

Dr. Chwan-Li (Leslie) Shen is an Associate Professor of Pathology and Physiology, School of Medicine and a Clinical Associate Professor of Laboratory Sciences and Primary Care, School of Allied Health, Texas Tech University Health Sciences Center, Lubbock, TX. She is also a researcher at the Laura W. Bush Institute for Women’s Health, Texas Tech University Health Sciences Center. She obtained her B.S. degree from Providence University Taiwan, her MS degree from Texas Tech University Texas, and her PhD degree from Purdue University Indiana. Within her faculty career, she has developed a broad range of expertise in molecular mechanisms and animal models of osteoporosis and osteoarthritis. Her research papers include a wide spectrum of interventions such as dietary nutrients, bioactive components, functional food, phytochemicals, herbal supplements, and exercise, and various mechanisms including anti-inflammation, anti-oxidative DNA damage, bone metabolism and structure, and bone biomechanics – often shown in different bone disease models.

Eric Rivas
Assistant Professor of Exercise Physiology
Department of Kinesiology and Sport Management, Texas Tech University

Eric Rivas, PhD., is an assistant professor of exercise physiology in Department of Kinesiology and Sport Management. His research expertise is in integrative cardiovascular human physiology with a focus on how exercise and thermal stress affect cerebral, cardiovascular, and metabolic health. He has experience with randomized control trials using exercise rehabilitation for improving cardiovascular and metabolism in clinical populations.

Current projects involve 1) cerebral blood flow hemodynamics during exercise under exertional heat stress, 2) heat therapy and omega 3s on vascular, metabolic, and microbiome health in Mexican Americans, and 3) exercise and heat stress on improving arterial stiffness, and 4) heat acclimation on cognition, cerebral autoregulation, and brain function. He is looking for collaborations that want to use exercise training or heat “therapy” in clinical populations such as aging, depression, obesity, hypertension, and other populations with arthritis and pain.
Faculty Presentations

Varuna Nargunan
Endocrinologist
Procare Endocrinology, Medical Center Hospital System, Odessa, TX, USA

Dr. Nargunan graduated from medical school from India. She did her residency and fellowship training in Buffalo, NY. She is board certified in Internal Medicine and Diabetes, Endocrinology and Metabolism. Dr. Nargunan believes in “prevention is better than cure”. Her areas of interest are Diabetes, Obesity, dyslipidemia.

Kay Tindle
Managing Director, ORDC, ORIC
Texas Tech University

Kay Tindle currently serves as the Managing Director for the Office of Research Development & Communications (ORDC) in the Office of Research & Innovation. The ORDC manages limited submissions competitions, provides editorial services for grant proposals, develops strategies for resubmissions, and offers early-stage proposal development assistance for large-scale, center-like proposals and for new faculty or first time submissions.

Prior to joining Texas Tech, Kay served as the Grant Facilitator for the College of Liberal Arts at the University of Central Oklahoma, interned at the Oklahoma State Regents of Higher Education, and taught English around the world (Japan, China, Saudi Arabia, South Korea).

Kay received her Bachelor of Arts in Teaching English as a Foreign Language from Oklahoma Christian University, her M.Ed. in Adult and Higher Education from the University of Central Oklahoma, and her Ph.D. in Higher Education Research from TTU.
Oral Presentations

Faculty Oral Presentations

Varuna Nargunan
Diet Modification and Changes in Parameters of Metabolic Syndrome: A Case Series

Postdoc Oral Presentations

Kembra Albracht-Schulte
Eicosapentaenoic Acid Improves Hepatic Metabolism and Reduces Inflammation Independent of Obesity in High-Fat-Fed Mice and in HepG2 Cells.

Vipulkumar Patel
Fingertip Assessment of Cardiometabolic Health Using Compact NMR

Jongkyoo Kim
All-trans Retinoic Acid Increases the Expression of Oxidative Myosin Heavy Chain through the PPARδ Pathway in Bovine Muscle Cells Derived from Satellite Cells

Student Oral Presentations

Makenzie Miller
Nutrition Education Needs Assessment of Farmers Market Participants in Lubbock, Texas

Md Akheruzzaman
E4orf1 Protein Reduces Adiposity-associated Hyperinsulinemia Independent of Insulin Sensitivity

Mohammed Jodeiri
Neurobehavioral Dysfunction Induced by Acute Stress Is Ameliorated by the Myokine Irisin in Male Mice
The incidence of obesity has increased dramatically over the past couple of decades. It is considered to be an epidemic in the United States. According to the US Centers for Disease Control and Prevention, obesity now affects more than one third (39.8%) of adults in the United States. Obesity is a risk factor for some of the leading causes of preventable death, including heart disease, stroke, type 2 diabetes mellitus and certain cancers.

We present the results of a simple patient initiated life style change that modified the risk in persons suffering from the metabolic syndrome. The patients were motivated to change their dietary habits to improve their health outcomes and achieve weight loss. The significant parameters monitored were body mass index (BMI), fasting glucose, blood pressure, weight, triglycerides, and waist circumference. Each patient started a low carbohydrate keto diet, which was limited to not more than 1200 calories.

Some of them continued only the diet and some incorporated exercise regimen along with their diet. The intervention resulted in weight loss that was durable at one year. In presented cases calorie restricted diet resulted in significant improvement of metabolic parameters.
Objective: Prevalence of nonalcoholic fatty liver disease (NAFLD) is increasing worldwide, concurrent with increased obesity. Thus, there is urgent need for research that can lead to effective NAFLD prevention/treatment strategies. We previously reported that eicosapentaenoic acid (EPA), an omega-3 polyunsaturated fatty acid (n-3 PUFA), reversed obesity-induced hepatic steatosis in high fat (HF)-fed B6 mice. Although n-3 PUFAs are well documented as anti-inflammatory and triglyceride lowering, mechanisms mediating the benefits of n-3 PUFAs in NAFLD treatment are less understood.

Methods: Utilizing liver tissues from HF and HF-EPA-fed mice and a series of in vitro studies in tumor necrosis factor-alpha (TNF-α)-stimulated HepG2 cells, we dissected the gene, protein, and microRNA-targeted mechanistic effects of EPA in reducing hepatic steatosis.

Results: With EPA, hepatic lipid metabolism was improved, as indicated by decreased protein and messenger RNA (mRNA) levels of fatty acid synthase (FASN) and acetyl-CoA carboxylase (Acaca) gene, and increased mRNA levels for the peroxisome proliferator activated receptor-alpha (Ppara), and carnitine palmitoyltransferase (Cpt) 1a and 2 genes in the HF-EPA mice. Additionally, inflammation was reduced, as shown by decreased Tnf-α gene expression. Accordingly, EPA also significantly reduced FASN and ACACA mRNAs in human HepG2 cells. Furthermore, we identified several miRNAs that are regulated by EPA in mouse liver, including miR-19b-3p, miR-21a-5p, and others, which target lipid metabolism and inflammatory pathways.

Significance & conclusions: Our findings provide novel mechanistic evidence for beneficial effects of EPA in NAFLD, through the identification of specific genes and miRNAs, which may be further exploited as future NAFLD therapies.
An optimal screening test for metabolic health in point-of-care settings would be rapid, inexpensive and non-invasive. With those features in mind, we engineered a compact NMR device to measure T2 in the living tissues of the human fingertip. This toaster-sized device includes a custom-built 0.5T magnet assembly. High-quality data can be collected in less than 2 minutes. Analysis of fingertip decay curves yielded three T2 peaks. Based on anatomical considerations and control samples, the peaks were assigned to distinct mobility domains in adipose tissue triglycerides.

Preliminary fingertip NMR measurements on 3 non-diabetic subjects revealed subject-to-subject variation in T2 values. We hypothesize that the variation arises from adipose tissue fluidity, which is affected by lipid composition and the fibrous connective tissue matrix. Based on the analysis of oil-phase lipids, increased saturated fatty acid content in adipose tissue is expected to lower fingertip T2 values. Saturated fatty acids are derived from the diet and from de novo lipogenesis, which is increased with insulin resistance. In addition, tissue hypoxia, inflammation and fibrosis may stiffen the connective tissue matrix surrounding adipocytes, further lowering finger T2. Adipose tissue hypoxia has been associated with obesity and obstructive sleep apnea. We hypothesize that high finger T2 values are associated with good metabolic health, and low T2 values, poor metabolic health. A study is under development to test this hypothesis.

In conclusion, the measurement of fingertip T2, reflective of adipose tissue stiffness, is feasible and practical. It shows early promise as a non-invasive marker of cardiometabolic health.
All-trans Retinoic Acid Increases the Expression of Oxidative Myosin Heavy Chain through the PPARδ Pathway in Bovine Muscle Cells Derived from Satellite Cells

J. Kim¹, K.B. Wellmann¹, Z.K. Smith², and B. J. Johnson¹

¹Department of Animal and Food Sciences, Texas Tech University, Lubbock, TX 79409
²Department of Animal Science, South Dakota State University, Brookings, SD 57007

Objective: This study was designed to investigate how different doses of ATRA impact gene expression and protein level related to muscle growth, energy metabolism, and muscle fiber types.

Methods: Bovine primary satellite cell (BSC) were isolated from the semimembranosus of crossbred steers (n = 2 steers), aged approximately 24 mo. Myogenic differentiation was induced upon cultured BSC with increasing doses (0, 1, 10, 100, and 1,000 nM) of ATRA. After 96 h of incubation, cells were harvested and used to measure the gene expression and the protein level.

Results: Gene expression of PPARδ was increased (P < 0.05) with 1,000 nM of ATRA. Protein level of PPARδ was also affected (P < 0.05) by 1,000 nM of ATRA. Gene expression of MHC I (P < 0.05) was increased with 10nM of ATRA but MHC IIX was downregulated (P < 0.05) with 100 and 1,000 nM of ATRA.

Conclusion: In muscle cells, ATRA may cause muscle fibers to transition towards the MHC isoform that prefers oxidative metabolism, as evidenced by increased expression of genes associated with the MHC I and IIX isoforms. These changes in MHC isoforms appeared to be brought about by changing PPARδ gene expression and protein levels.
High chronic disease incidence in Lubbock reflects a need for nutrition education (NE) among Lubbock residents. The objective of this study was to determine the needs for planning and implementing future NE programs for Lubbock farmers market (FM) participants. A cross-sectional study, using a self-administered questionnaire, was conducted to collect data from a convenience sample of 394 adults at two purposefully selected FMs between June and October 2018. Data were analyzed using R version 3.4.3. Fisher’s exact test of homogeneity was used to compare responses between markets. Results indicated a significantly higher proportion ($P = 0.0016$) of Lubbock Downtown Farmers Market (LDFM) participants (64%) desired NE than Texas Country Farmers Market participants (46%). The majority of participants (>50%) from both markets preferred NE programs with nutrition information, cooking demonstrations, and food tastings and to receive handouts, product information, and recipe cards. A $\leq 15$ min NE lesson held at the FM, with seating available, was most often preferred at both markets. The majority (74%) of LDFM participants favored NE lessons held in the morning, while Texas Country Farmers Market participants indicated no preference. At both markets, learning about eating more fruits and vegetables, eating less sugar, and eating right on a budget were among the top five most-desired NE lesson topics. These results will be used for future NE program development at Lubbock FMs. The results further exemplify that NE needs vary among populations and, thus, programs must be tailored to each populations’ unique needs.
E4orf1 Protein Reduces Adiposity-associated Hyperinsulinemia Independent of Insulin Sensitivity

Md Akheruzzaman¹, Patkar PP¹, Shin AC¹, Hegde V¹, & Dhurandhar NV¹

¹Obesity and Metabolic Health Laboratory, Department of Nutritional Sciences, Texas Tech University, Lubbock, TX-79409

Objective: Obesity is linked with impaired insulin sensitivity, leading to hyperinsulinemia. E4orf1 protein derived from adenovirus-36 improves glycemic control independent of insulin signaling. We hypothesized if E4orf1 reduce the requirement of endogenous insulin and reduce hyperinsulinemia without increasing insulin sensitivity.

Methods: About 29wk old wild-type (WT; n=6) or transgenic mice that express E4orf1 in adipose tissue upon doxycycline ingestion (E4Tg; n=5) started rodent chow-doxycycline for 6wk, followed by 10wk of a 60% high-fat-doxycycline diet (HFD). Serum glucose, insulin responses to oral glucose load were determined at baseline, 6wk, and 10wk after switching to HFD.

Results: WT mice gained while E4Tg mice lost body fat between wk 1 and 6 (+4.8% vs -5.6%, respectively, p<.02). When switched to HFD, WT gained more body fat vs E4Tg mice (+7.6% vs +1.7%, p<.02). In response to the glucose load, the area under curve (AUC) for glucose was lower at 6wk for E4Tg mice vs WT (p<.01), which worsened for both groups upon HFD. Reduction in AUC for insulin was significantly greater for E4Tg between baseline and wk 6 or wk 16. On HFD, the AUC for insulin increased by 1511% for WT but only by 362% for the E4Tg group (p<0.04). The response to ITT did not differ between WT and E4Tg.

Conclusion: Our findings suggest that E4orf1 reduced insulin level while increased glucose clearance independent of insulin sensitivity, which in turn indicates significant potential of E4orf1 in improving obesity-linked hyperinsulinemia.
Neurobehavioral Dysfunction Induced by Acute Stress Is Ameliorated by the Myokine Irisin in Male Mice

Mohammad Jodeiri-Farshbaf¹, Daniel Cherkowsky¹, Nabeela Manal², Dominica Moussoki¹, & Karina Alviña¹

¹Department of Biological Sciences, Texas Tech University, Lubbock, TX 79409
²Honors College, Texas Tech University, Lubbock, TX 79409

The myokine irisin, obtained after cleavage of its precursor protein fibronectin type III domain-containing 5 (FNDC5), has a variety of physiological roles such as regulation of body weight, temperature and glucose homeostasis. Irisin is also secreted during physical exercise and upon entering the peripheral circulation it can impact several organs including the brain. In fact, it has been shown that irisin levels are decreased in patients with disturbed mood as well as post-stroke depression. However, the overall effects of irisin on brain function, in normal and pathological conditions, are largely unknown.

Exercise can ameliorate certain aspects of stress-induced mental illness. Therefore, we hypothesized that irisin may indeed be involved in mediating the observed positive effects of exercise on brain function. We focused on the hippocampus as a vulnerable region to stress that is also associated with emotional regulation, and learning and memory, all functions heavily modulated by stress. Using both female and male adult C57Bl/6 mice, we first implemented an acute 3-hours restraint stress test that increased anxiety-like behaviors in the open field test and impaired novel object recognition, effects that were significantly more robust in male mice. Then using stereotaxic injections, we bilaterally injected 1 ng of irisin in the hippocampi of male mice and subjected them to 3 h of acute stress followed by open field and novel object recognition tests.

Irisin-injected stressed mice showed reduced anxiety-like behavior and improved working memory compared to non-injected or vehicle-injected stressed mice. In addition, we measured mice’s skin temperature and irisin injection in the hippocampus markedly reversed the decrease in temperature triggered by acute stress, which is consistent with the well-known thermogenic function of irisin. Mechanisms underlying irisin effects are currently being investigated, emphasizing the role of brain-derived neurotrophic factor and mitochondrial function. Overall our findings suggest a possible role of the exercise-induced protein irisin in counteracting the anxiogenic effects of acute stress. Furthermore, our results may provide novel insights into the neurobiological mechanisms of stress-induced brain dysfunction and suggest putative therapeutic avenues.
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<td><strong>Maternal Obesity in Early Pregnancy Is Associated with the Decreases Active IGF-I Availability</strong></td>
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<td><strong>Parental Elevated Salt Consumption in Mice and the Development of Autism Spectrum Disorder (ASD)-like Behavior in the Offspring</strong></td>
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Introduction: Maternal obesity (MO) is the global health problem, associated with high fetal and maternal mortality. Cardiovascular remodelling in obese pregnant patients has been linked to development of preeclampsia. IGF-I is important for maternal adaptation to pregnancy, e.g. maternal tissue growth and metabolism. Pregnancy-associated plasma protein-A2 (PAPP-A2) is a metalloproteinase that specifically cleaves IGFBP-3 and IGFBP-5 releasing active IGF-I. Leptin, produced by placenta and adipose tissue, is a cytokine with cardio metabolic properties, the concentration of which is increased in obese subjects and in pregnant patients. The aim of this study was to evaluate serum levels of PAPP-A2 (as an indicator of IGF-I pathway activation) and Leptin in the obese and non-obese patients in first trimester of pregnancy. Glycosylated fibronectin (GlyFib) and Inhibin A (Inh A) were evaluated as additional markers of remodelling and placental function (respectively).

Material and methods: This was a prospective observational study, patients were enrolled during first trimester of pregnancy according to the IRB-approved protocol (L#17-136): obese (OB, BMI≥ 30 kg/m², n=13) and non-obese (nOB, BMI<30 kg/m², n=14). Patients with pre-pregnancy cardiovascular pathology were excluded. Concentrations of PAAP-A2, GlyFib, Inh A and Leptin were measured, using commercially available ELISAs: AL- 109, AL-123 and AL-160 from AnshLab (Webster, TX, USA) and Invitrogen (Waltham, MA). The data are presented as Mean ± SEM.

Results: In 3/13 OB and in 1/14 nOB patients PAAP-A2 concentrations were below 0.3 MoM (CI [4.8 – 34 ng/ml] and [CI 5.8-21 ng/ml] respectively). GlyFib (OB, 303.4 ± 35.6 µg/ml; nOB, 208.7 ± 19.26 µg/ml) and Leptin concentrations (OB, 72775 ± 3802 pg/ml; nOB, 48882 ± 4052 pg/ml) were increased in OB patients compared to nOB (*p<0.05; **p<0.0005, Mann-Whitney test, respectively). Inh A concentrations (OB, 479.6 ±105.9 pg/ml; nOB, 381 ± 81.65 pg/ml) did not differ between two groups. In the nOB population, incidence of preeclampsia was 1/15 (0.13%) which was not associated with low PAPP-A2 levels. In the OB population, incidence of preeclampsia was 3/9 (33%) where 2/3 of cases were associated with low PAPP-A2 levels.

Discussion: IGF-I and Leptin regulate cardiovascular remodelling and placentation. The mismatch of these two factors might represent the mechanism of pathological cardiovascular adaptation in OB pregnant patients. Limitations of this study are small sample size, non-inclusion of other predisposed populations (PCOS, GDM, autoimmune disorders) and loss for follow-up.
Alzheimer’s Disease Is Associated with Impaired Branched-chain Amino Acid Metabolism

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Objectives: Alzheimer’s disease (AD) is a neurodegenerative disorder characterized by accumulated plaques, tangles, and diminished neurotransmitters in the brain. AD is strongly associated with type 2 diabetes (T2D) and branched-chain amino acids (BCAAs) are involved in the pathogenesis of T2D. In support of this concept, BCAA degradation is impaired in diabetic individuals, and BCAA supplementation leads to insulin resistance. It is currently unknown if similar defective BCAA metabolism exists in AD. Since BCAAs are critical for maintenance of brain neurotransmitters, here we tested if BCAA metabolism in liver – an organ with high BCAA degradation activity – is impaired in AD.

Methods: Serum profile of BCAAs and their metabolism were examined in both AD patients and a transgenic mouse model of AD. Proteins and genes related to BCAA metabolism in mouse liver were determined by western blot and RT-qPCR, respectively.

Results: The activity of branched-chain α-ketoacid dehydrogenase (BCKDH), the rate-limiting enzyme in BCAA degradation, in liver was significantly suppressed in AD mice. This is most likely due to an increased hepatic BCKDH Kinase – a potent inhibitor of BCKDH – at both protein and gene levels. Serum BCAAs and/or their metabolites were higher in both AD mice and humans compared to healthy controls, indicating impaired BCAA metabolism.

Significance & Conclusions: Our findings suggest that hepatic BCAA catabolism is impaired in AD mice. This may lead to high plasma BCAAs and their metabolites that can potentially contribute to an imbalance of brain neurotransmitters and development of AD.
Whole Blood T2s and T2p as Biomarkers for Early Insulin Resistance: Potential for Point-of-care Screening

Vipulkumar Patel, PhD, Ina Mishra, PhD, Sneha Deodhar, MS, & David P. Cistola, MD, PhD

Prediabetes and metabolic syndrome identify individuals with increased cardiometabolic risk. However, by the time those conditions develop, a large decline in pancreatic insulin secretion has occurred. To preserve beta cell function and arterial wall integrity, an optimal screening and intervention strategy would identify at-risk individuals earlier. Here we describe a new approach for detecting early metabolic dysregulation, a condition that includes compensatory hyperinsulinemia (early insulin resistance) and subclinical inflammation.

The method uses a fingerstick drop of whole blood and a tabletop magnetic resonance device about the size of a toaster. It measures the 1H transverse relaxation times (T2) for the plasma supernatant (T2s) and the cell pellet (T2p) of settled anti-coagulated whole blood. No chemical reactions or reagents are required, and the measurement takes 3 minutes.

In a discovery cohort of 43 asymptomatic non-diabetic subjects, remarkably strong bivariate correlations were observed between T2p and markers of poor metabolic health, namely hyperinsulinemia, inflammation and dyslipidemia. By contrast, T2s correlated with red blood cell count and hematocrit, even though the supernatant was devoid of blood cells. Upon varying the ratio of supernatant to pellet, we observed that T2s was inversely proportional to the sixth power of hematocrit. Thus, a magnetic susceptibility gradient from the paramagnetic pellet was enhancing T2 relaxation in the supernatant.

After mathematically correcting for this gradient, T2s can be used to estimate plasma water T2, another early marker of poor metabolic health. This approach is powerful and practical, and shows promise for translation into clinical point-of-care settings.
Cardiorespiratory Fitness is Lower in Obese Compared to Lean Boys

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Purpose: The assessment of cardiorespiratory fitness using ratio scaling to total body mass (TBM) is confounded by fat mass in obese populations. Because fat tissue does not contribute to oxygen utilization during exercise, our objective was to isolate the independent effects of obesity on fitness by normalizing oxygen consumption to total lean body mass (TLM) and leg lean mass (LLM). We tested the hypothesis that boys with obesity would have attenuated cardiorespiratory fitness compared to age-matched non-obese boys.

Methods: Values are expressed as means ±SD with significance at P<0.05. Ten non-obese boys (10.6±0.9y, 141.8±6.5cm, 35.5±7.0kg, 24±5%bodyfat) and nine age-matched obese boys (10.6±1.4y, 146.3±10.6cm, 60.25±13.0kg, 44±2% body fat) completed a cardiorespiratory fitness test (VO2peak) and body composition scan (DXA).

Results: Utilizing an independent T-test, both groups had comparable VO2peak test times (9.1±1.4 min; P=0.80), and peak heart rates (187±12 bpm; P=0.50). Boys with obesity had a reduction in VO2peak when normalized to TBM (54% of age-matched boys without obesity); however, this effect was less when compared to LBM (76%) and LLM (68%). Simple linear regression found that total body fat accounted for 69% variance for mL/kgTBM/min, 49% variability for mL/kgLBM/min, and 40% variance for mL/kgLLM/min.

Conclusions: These data indicate obesity in young boys impairs cardiovascular fitness which supports the concept of later life health consequences in regard to cardiorespiratory fitness and all-cause mortality. Lastly, we show that the normalization of VO2 to LBM and LLM can provide an independent measure of fitness.
Objective: Curcumin, a traditionally used spice in Asia has several health-protecting effects. However, its role on gut microbiota and obesity-associated inflammation is still poorly understood. The objective of this study was to determine whether the protective effects of curcumin in high fat diet (HFD)-induced obesity are mediated by reduced white adipose tissue (WAT) inflammation and changes in gut bacteria.

Methods: Male B6 mice were fed a HFD (45% kcal fat) or HFD supplemented with 0.4% (w/w) curcumin (HFC) for thirteen weeks. Body weight, adiposity, glucose and, insulin tolerances, as well as serum triglycerides, insulin, leptin and resistin levels were measured. Gut microbiome composition was determined by 16S RNA metagenomics sequencing. Expression of inflammation-related genes in WAT was measured by qRT-PCR. Macrophage contents in WAT were evaluated by galectin-3 immunohistochemical staining.

Results: Pro-inflammatory transcription factor nuclear factor NF-kappa-B p65 subunit (p65) and toll-like receptor-4 (Tlr-4) gene expression were downregulated in HFC group compared to HFD mice. Furthermore, curcumin reduced total macrophage infiltration in WAT in HFC mice compared to HFD group. Expression of both M1 (CD80, CD38) and M2 (Arginase-1, CD11c) associated genes was decreased. The relative abundance of bacteria representing the Lactococcus, Sutterella, Turicibacter and Oscillospira genus, which contains numerous short-chain fatty acid (SCFA) producing species, was increased by the curcumin supplement.

Conclusion: Curcumin exerts protective effects in dietary obesity, in part through downregulation of adipose tissue inflammation which may be due to the production of SCFA and, possibly other curcumin metabolites by gut microflora.

Funding: Startup funds and Come N Go award from the College of Human Sciences at Texas Tech University
Objective: Obesity is an important risk factor for breast cancer progression, especially in post-menopausal women. Various pro-inflammatory cytokines, and growth factors secreted by obese adipocytes significantly promotes the formation of tumor microenvironment. Dietary interventions such as n-3 polyunsaturated fatty acids (n-3 PUFAs) may lessen these negative effects respectively by reducing pro- and increasing anti-inflammatory adipocytokines secretion. Hence, we hypothesize that eicosapentaenoic acid (EPA) will reduce breast cancer cell inflammation, and proliferation via altered adipocytoine secretion.

Methods: MCF-7 and MDA-MB-231 human breast cancer cells were treated with 100 uM EPA for 48h. Conditioned medium (CM) was collected from murine 3T3-L1 adipocytes pretreated with 100 uM EPA and transferred to breast cancer cells for 48h. Changes in gene and protein levels were determined, along with cell migration via wound healing assays. Two-tailed student’s t test was used to detect statistical significance at p<0.05.

Results: Direct EPA treatment reduced inflammation, and proliferation in MDA-MB-231 breast cancer cells but not in MCF-7 after 48h, as determined by reduced mRNA levels of IL-6, NF- KB, STAT3, and FASN respectively. In addition, CM obtained from EPA pretreated adipocytes significantly reduced inflammation and fat synthesis in both breast cancer cells after 48h as shown by significant lower phosphorylation of NF-KB and STAT3, and reduced FASN protein levels. Furthermore, CM derived EPA reduced cell migration in both breast cancer cells as determined by decreased percent wound healing after 48h.

Conclusions: Our research provides strong evidence to develop EPA as a potential anti- inflammatory dietary intervention in obesity-mediated breast cancer treatment.
Early Cardiometabolic Risk: The Prevalence of Compensatory Hyperinsulinemia in U.S. Populations

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Metabolic syndrome (MetS) and prediabetes (PreD) identify individuals at increased risk for type 2 diabetes. However, by the time MetS or PreD develops, a large decline in pancreatic insulin secretion has already occurred. The earliest stage in diabetes pathogenesis is compensatory hyperinsulinemia (CH), the heightened response of functional beta-cells to early insulin resistance. This stage is not detected by conventional glucose or lipid screening. The study objective was to estimate the prevalence of CH in U.S. populations and quantify its possible association with inflammation and dyslipidemia.

Methods: Population-weighted data from the 2013-2014 cycle of the U.S. National Health and Nutrition Examination Survey were analyzed. After applying exclusion criteria, subjects age 12 and above were divided into four groups with respect to metabolic health: reference group (RG), compensatory hyperinsulinemia (CH), PreD and/or MetS without diabetes (PM) or diabetes mellitus (DM). The CH group was defined as hyperinsulinemia without PreD, MetS or DM.

Results: The U.S. prevalence of CH was 10.3% (95% CI: 9.4, 11.4). After adjusting for gender, race/ethnicity and body-mass index, CH prevalence was highest in the 12-19 age group, 24.2% (95% CI: 20.4, 28.5), and declined with age. With respect to inflammation, CH was associated with subclinical elevations in white blood cell count and % serum globulins. Also, CH was associated with subclinical elevations in triglycerides, but not total cholesterol.

Conclusion: Compensatory hyperinsulinemia is surprisingly prevalent in the U.S, particularly among teenagers. Thus, teenagers with early insulin resistance represent an untapped target population for diabetes prevention.
Identification of miRNAs Mediating Effects of the Renin Angiotensin System in Adipose Tissue

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Objectives: The renin angiotensin system (RAS) classically known to regulate blood pressure, is also involved in obesity. Interestingly, RAS components are highly expressed in adipose tissue; however, mechanisms underlying RAS-obesity interactions are still ambiguous. We identified previously that RAS overactivation induces ER stress and inflammation, and our goal is to characterize additional mechanisms linking RAS to obesity. Hence, we hypothesized that overactivation of angiotensinogen, modulates processes linked to metabolic diseases in adipocytes.

Methods: mRNA and small RNA profiling were performed in adipose tissues of male wild type (Wt) and transgenic mice (Tg) which were fed either a low fat (LF) or a high fat (HF) diet with or without RAS inhibitor captopril.

Results: We identified 18 and 5 miRNAs, which were significantly either up or downregulated respectively in Tg compared to Wt mice. Of these, we validated expression of mir195 and 690 which were significantly higher in Tg compared to Wt mice. Furthermore, these miRNAs were significantly reduced in HF Tg mice treated with captopril compared to non-treated HF mice, indicating a possible role of angiotensin II in regulation of these miRNAs. Additionally, we identified and validated several genes involved in physiological processes such as oxidative stress and autophagy. Additional mechanistic studies are ongoing in cultured adipocytes to further dissect molecular mechanisms linking RAS to obesity.

Conclusions: Overexpression of RAS in adipose tissue alters various physiological processes which could be mediated, in part through regulatory miRNAs. These pathways and miRNAs could be potential therapeutic targets to reduce RAS-associated metabolic diseases.
We are developing a powerful software tool to analyze microscopic images of adipose and cancer tissues and cells. In this software we plan to use several image processing algorithms to analyze different types of microscopic images and acquire the data required by biomedical researchers.

The software can process different types of microscopic images including H&E and fluorescence stained images. After automatically enhancing the quality of the image, the Software can calculate the number of the cells (including and excluding the boundary cells) and their areas in an image accurately, by using the calibration factor assigned by the lab user. Furthermore, the software can generate the histograms required for the biology research (number of small, medium, and large size cells). In biological images, each color represents a specific organelle and calculating the area of these colors can be a big step for biomedical researchers. This software will not only allow the researcher to deal with the different colors to get the information from the image but will also allow the researcher to specify the range of the colors they want to analyze. Thus, using the software, the analyses and results will be more specific.

Using this software doesn't need a vast knowledge in computer sciences as it is user friendly and exclusively developed for cell biologists. It is equipped with different analysis tools such that researchers can manage the majority of the image processing requirements including cell counting, scratch assay, color separation and area of interest.

This Software is been developed in collaboration with some Obesity Research Cluster members, who study fat and cancer cells and tissues. Once optimized, this software will be available to other researchers. We expect that this image analysis tool will be save time while providing the most accurate results due to the novel image processing algorithms used in the software.
Objective: The exposome consists of factors an individual is exposed to across the lifecourse. The exposome is dynamic, meaning the factors are constantly changing, affecting each other and individuals in different ways. Our exposome dataset includes social determinants of health as well as policy, climate, environment and economic factors that could impact individual obesity development. The objective is to translate spatial and temporal exposure to these factors with development of obesity into actionable population based policies.

Methods: Hot Spot Analysis was performed followed by Graph Analysis to model the multifactorial connections within geographic obesity hot spots. Factors spatially identified as related to obesity were further evaluated using Graph Analysis to develop models. The models provide insights into, potentially, modifiable factors for obesity mitigation and prevention at the county level.

Results: Preliminary results confirm prior graph analysis modeling of risk variables originating at the individual, neighborhood and biome levels. Fifteen density models of potentially modifiable factors were generated. The models encompass constructs such as geographic area exposure (pollution, weather); neighborhood stress (crime, occupations, work commutes); social determinants of health (poverty, education, wages); lifestyle (smoking, soda consumption, physical activity). The results are preliminary and we will continue to verify models for different levels (high and/or low) and concentration (clusters) of obesity in various areas.

Conclusion: Big Data algorithms can be used to provide insight into complex multifactorial connections of obesity on both an individual and population level.
Protective Effects of Eicosapentaenoic Acid on Cognitive Impairment and Inflammation in the APPswePS1dE9 Alzheimer's Mouse Model

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Alzheimer’s disease (AD) is a neurodegenerative prevalent disorder, characterized by the presence of amyloid plaques and intracellular neurofibrillary tangles. Exact etiology, treatment and early diagnosis of AD remain unknown. Obesity is an important risk factor of neurodegenerative disorders, possibly it could cause brain and peripheral inflammation.

Eicosapentaenoic acid (EPA), a long chain omega-3 fatty acid from fish oil, exerts anti-inflammatory effects in white adipose tissue, and thermogenic effect in brown adipose tissue of diet-induced obese (DIO) mice. The objective of this study is to determine whether EPA supplementation in DIO improves overall metabolic and cognitive health in an amyloidogenic mouse model of AD (APPswePS1dE9), specifically, by reducing brain inflammation and amyloid beta deposits.

To test this hypothesis, at 2 months of age, transgenic (Tg) and non-transgenic (non-Tg) mice of both sexes were assigned to one of three dietary groups; low fat (LF), high fat (HF) and HF supplemented with EPA (HF-EPA) for 8 months. Blood samples are collected for AD and obesity related markers; body weight, body composition, and glucose tolerance test are conducted throughout the study. Morris water maze and novel object recognition tests for memory will be conducted.

At the end of the study, mice will be euthanized and brain, adipose tissues and serum will be collected. We predict that genotype (Tg vs. Non-Tg) and diet (HF vs. LF) will result in decreased cognitive performance and increased levels of inflammatory markers, EPA supplementation will exert preventative effects. This study is ongoing and preliminary findings will be presented.
Background: Exercise barriers in people with obesity, such as low quality of life, physical activity enjoyment, and self-efficacy may contribute to an increased sedentary lifestyle. However, the use of an anti-gravity treadmill that is able to support and elevate weight during exercise to reduce these barriers has not been examined.

Purpose: To examine how an anti-gravity treadmill during 12 weeks of aerobic exercise effects Physical activity enjoyment (PAE), physical functioning, self-efficacy, and quality of life (QOL) in people with obesity.

Methods: 26 participants (10 male, 16 female) participated in a self-directed 12 week walking program using an anti-gravity treadmill. Participants were randomized into two groups (N=13/group): weighted (W), exercising at 100% of their weight, or unweighted (UW), who self-selected their workout weight. PAE, self-efficacy, QOL, and timed up and go test (TUG) and a 6 minute walk test were administered pre and post. The QOL questionnaire has different subsections that were used for analysis: overall quality of life, physical health, psychological, social, and environmental. Analysis included participants that completed the pre and post visits (N=17): W (N=9) and UW (N=8).

Results: Weight and BMI for W (107.7kg, 36.0kg/m2) and UW (109.8kg, 37.7kg/m2) were not significantly different (P>.05). However age was significantly different, W (27.6 years) and UW (36.4 years) (p=.028). Total duration, and total energy expenditure were not significantly different between groups W (617.5 min, 8501.6kcal) and UW (712.2 min, 9209.7kcal), respectively, (P>.05). After adjusting for baseline values, there wasn’t a significant difference between groups in PAE, self-efficacy, TUG, and the 6 minute walk test (p>.05). As a group, UW scored 13.1 points higher than W on overall quality of life (F=6.601, P=.023). Psychological, social, and environmental subscales of QOL were not significantly different when adjusting for pre values (P>.05). However, physical health QOL was significantly different when adjusting for pre values (F=6.761, P=.020).

Conclusion: This pilot study confirms that using an anti-gravity treadmill with the unweighting feature can significantly increase overall quality of life and physical health quality of life in people with obesity.
**Poster Abstract**

**Do Weight Cycling, Age of Onset, Three Factor Eating Questionnaire, and Power of Food Scale Predict Initial Weight Loss in People with Obesity?**

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**Objectives:** Identifying predictors of early weight loss may have value in predicting longer-term success. We examined if weight-history variables (weight cycling; WCH, age of onset of obesity; AOO), and baseline Three Factor Eating Questionnaire (TFEQ) and Power of Food Scale (PFS) predict body weight loss (BWL) and fat mass loss (FML) following a brief intervention.

**Methods:** Thirty-two adults with obesity (30-39.9 kg/m²) participated in a 3-week calorie-restriction intervention (1120 kcal/d). WCH, AOO, subscale scores for TFEQ (dietary restraint, TFEQ-R; disinhibition, TFEQ-D; susceptibility to hunger, TFEQ-S), and PFS (food-available, PFS-FA; food-present, PFS-FP; food-tasted, PFS-FT) were obtained at baseline; post-intervention BWL and FML were measured. Principle Component Analyses (PCA) were performed on TFEQ and PFS subscales. Multiple linear regression analyses were performed using WCH, AOO, and TFEQ and PFS subscales to predict BWL and FML.

**Results:** PCA demonstrated good internal reliability of TFEQ and PFS subscales, with PFS presenting as more reliable than TFEQ. BWL and FML decreased (3.71 kg, P=2e-8; 1.93 kg, P=3e-5). Baseline scores of TFEQ-R, TFEQ-D, TFEQ-S, PFS-FA, PFS-FP did not predict BWL (r=-.09, r=-.08, r=.01, r=-.25, and r=-.33, all P>.08 respectively) or FML (r=.12, r=-.28, r=-.28, r=.32, and r=.26, all P>.10 respectively). Baseline PFS-FT scores predicted BWL (r=-.40, P=.03). WCH and AOO did not predict BWL (P=.27, P=.31) or FML (P=.60, P=.30).

**Conclusions:** Psychological and weight-history variables considered in this study were not robust predictors of weight and fat loss. However, results for PFS suggest there may be value in further exploring this measure using larger sample sizes.
An Insulin independent approach to stimulate insulin signaling pathway

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Background: In type 2 Diabetes, hyperinsulinemia and decreased glycemic control are caused by impaired proximal insulin signaling. A therapeutic agent that enhances cellular glucose uptake without requiring proximal insulin signaling would be desirable for improving glycemic control.

The E4orf1 protein (E4) derived from human adenovirus 36(Ad36) promotes cellular glucose uptake in vitro and in vivo, independent of insulin. E4 bypasses a proximal insulin signaling pathway to upregulate AKT phosphorylation. However, it is unknown if E4 activity affects other downstream candidates of the insulin signaling pathway by phosphorylating GSK3α/β at serine 9/21 position, p70S6Kinase at serine 371, and FOXO1 at serine 256 similar to insulin. GSK3α/β is involved in insulin resistance, and insulin inactivates it by phosphorylation. In response to insulin, p70S6Kinase phosphorylation promotes cell growth and proliferation, and FOXO1 phosphorylation upregulates the process of adipocyte differentiation.

Methods: 3T3-L1 preadipocytes inducibly expressing E4 or a null vector(NV) were exposed to doxycycline for 24 h and serum starved for 2 h. E4 and NV cells was treated with 0 nM or 100nM insulin for 30 min. Protein extracted from cells were analyzed by western blotting.

Results: Similar to insulin, E4 expression significantly increased phosphorylation of GSK3α/β, p70S6Kinase, and FOXO1 as compared to untreated cells. Insulin treatment of E4 expressing cells did not show any additive effect in phosphorylation of distal downstream candidates, implicating positive influence of E4orf1 on the insulin-signaling pathway independent of insulin. Therefore, E4orf1 offers a promising template to stimulate insulin-signaling pathway when the proximal insulin signaling is impaired.
Objective: Autism Spectrum Disorder (ASD) is a very prevalent neurodevelopmental disorder with no known etiology or cure. As an environmental factor, recent studies have identified a possible causal relationship between maternal gut dysbiosis mediated immune system imbalance and ASD in the offspring. On the other hand, several recent studies show that elevated salt consumption has a significant effect on the gut microbiota and immune system where it can cause gut dysbiosis and immune imbalance. The main objective of this research is to find whether parental high salt consumption induced gut dysbiosis as well as immune imbalance can contribute to the development of ASD in the offspring.

Methods: To test our hypothesis, we fed male and female mice with high-NaCl chow and NaCl in their drinking water for 8-weeks while Control groups (CD) were fed with low NaCl chow and regular water. Then we paired HSD- or CD-fed males and females. The offspring from CD and HSD breeding pairs were then weaned and kept for behavioral analysis at 8 weeks-old. After all the behavior test mice are euthanized and blood and brain tissue samples were collected for the molecular detection.

Results: HSD male or female didn’t show any significant metabolic or behavioral changes compared to the control. In the offspring, male mice from HSD parents showed less social interaction, exploration, along with increased repetitive behaviors in comparison with the offspring from CD-fed mice. However, we haven’t seen any significant difference in the expression of HDAC1 protein in the prefrontal cortex of the offspring mice from the HSD fed parents than the control.

Conclusion: Our results support the idea that maternal HSD might increase the chances of ASD-like behaviors in the offspring. We are currently investigating mechanisms underlying these findings. Fecal samples from the parental females and offspring generation were collected for the gut microbiota sequencing. Additionally, detection of inflammatory gene expression in the parental female intestine and the flow cytometry for measuring the pro-inflammatory immune cell differentiation are also in process. In the offspring, as HDAC1 protein expression was not very different from the control, we will try to detect the expression level of some other protein associated with ASD.
Thank you for participating in the ORC 2019 Annual Meeting & Poster Competition!

Program & Abstracts

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Watch for us next year.
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