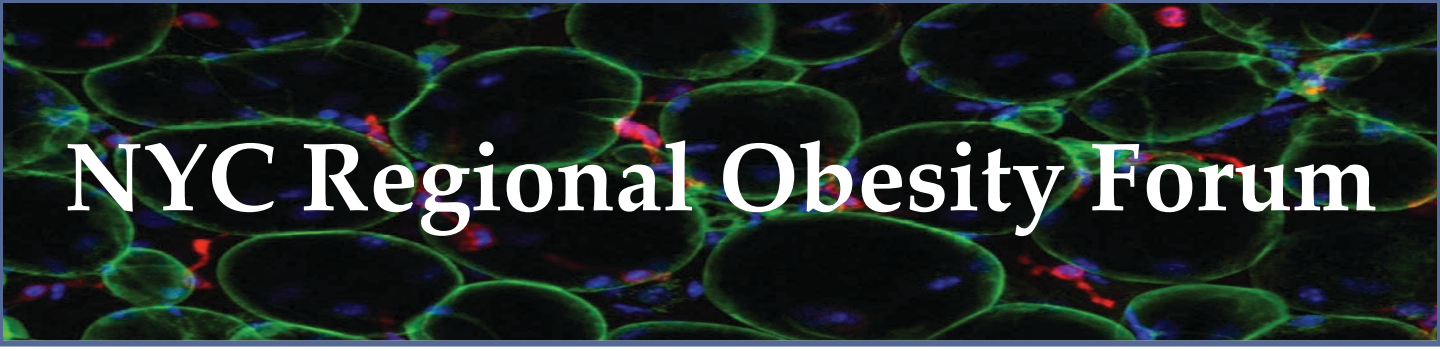



# 2nd Annual NYC Regional Obesity Forum

## Icahn School of Medicine at Mount Sinai

1468 Madison Ave (btw E 98th-101st)
Annenberg Building, Stern Auditorium
New York, NY 10029

### Keynote Speakers

Kelly D. Brownell, Ph.D.
Dean of the Sanford School of Public Policy
Robert L. Flowers Professor of Public Policy
Professor of Psychology and Neuroscience
Professor of Psychiatry and Behavioral Sciences
Member, National Academy of Medicine
Duke University

Susan K. Fried, Ph.D.
Professor of Medicine
Director of Translational Adipose Biology and Obesity
The Diabetes, Obesity and Metabolism Institute
Icahn School of Medicine at Mount Sinai

**Tuesday, September 19th, 2017**
8:30 am – 4:30 pm

**Registration (free) and Abstract Submission open May, 1**
icahn.mssm.edu/nycrobesityforum

@nycrof

# Visiting Mount Sinai Hospital

The Mount Sinai Hospital stretches from East 98th to 102nd Streets between Madison and Fifth Avenues on Manhattan's Upper East Side.

<u>The location of the NYCROF Meeting is:</u>

The Mount Sinai Hospital
1468 Madison Avenue
NY, NY 10029

entrances at 1190 Fifth Avenue (at E. 101st Street) and 1468 Madison Avenue.


# Directions to The Mount Sinai Hospital

Mount Sinai can be reached via the following public transit routes:

- Subway line number 6
- Bus lines M1, M2, M3, M4, M96, M98, M101, M102, M103, M106
- Complete public transit information and maps can be found at: http://www.mta.info/

If you are traveling to Manhattan, via train, please refer to Amtrak services New York's Penn Station and then visit the MTA website.

If you are flying to Manhattan, shuttle, bus, taxi, and limo services are available from the three major area airports—LaGuardia, JFK International, and Newark International in New Jersey.

- The Port Authority of New York and New Jersey offers comprehensive airport information, including information on transportation into Manhattan upon arrival.
- NY.com lists transportation options from each airport and estimated rates and trip times.
- Taxi and car service information is available through the New York Taxi and Limousine Service.

If you are traveling to Manhattan by car, please visit the page below for driving directions:
http://www.mountsinai.org/locations/mount-sinai/your-visit/locations

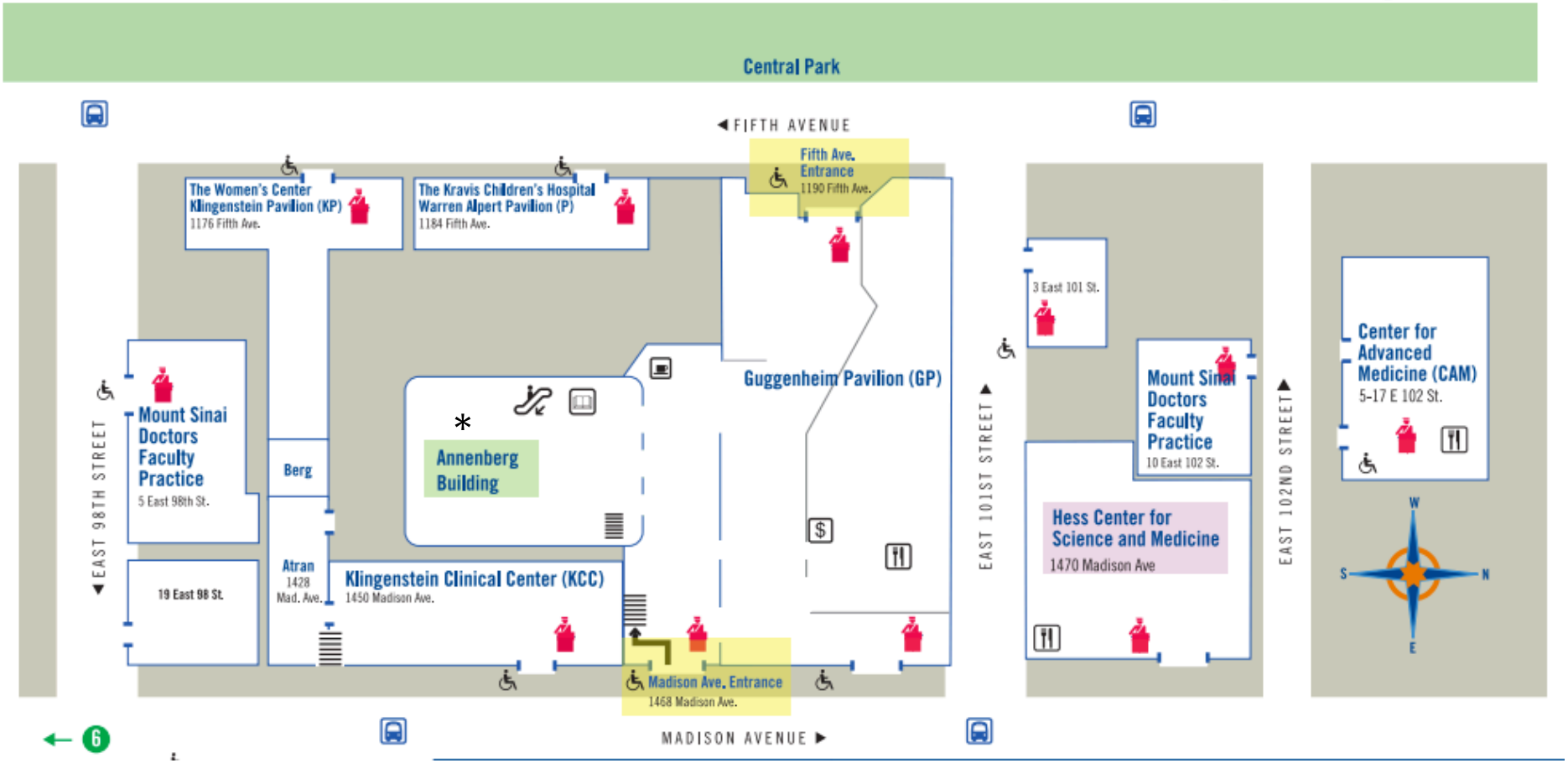
# Parking Options

The parking garage is located at 1292 Park Ave, New York, NY 10029 (on 99th Street between Park and Madison Avenues). The garage is open 24 hours a day. Cash, credit and debit cards are accepted. Call 212-241-5125 to inquire about hourly and daily rates. Metered parking on streets bordering the Mount Sinai campus is also available.

Additional parking options in the surrounding area include:

- MPG Parking: 6 East 102nd Street, New York, NY 10029, Between Madison and 5th Avenues, 212-289-2959
- Imperial Parking Systems: 1510 Lexington Avenue, New York, NY 10029, Entrance on 98th Street between Lexington and Park Avenues, 212-289-6257
- GGMC Parking: 60 East 94th Street, New York, NY 10128, Between Madison and Park Avenues, 212-369-9304

# Icahn School of Medicine at Mount Sinai Campus Map




Registration and badge pickup will be in the Annenberg Building, main level.

Keynote and Oral Presentations and will take place in the Stern Auditorium (up stairs adjacent to elevators). *Elevator access to Stern Auditorium is from the west elevator bank

Poster Presentations, refreshments and lunch will be in the Guggenheim Pavilion

# New York City Regional Obesity Forum

Tuesday, September 19, 2017

Icahn School of Medicine at Mount Sinai - Mount Sinai Hospital

1468 Madison Avenue - Stern Auditorium

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|---|---|---|
| 8:30-9:00 | 30min | Arrive, coffee, poster set-up |
| 9:00-9:10 | 10min | Welcome remarks - Rudy Leibel & Ruth Loos |
| | | **Chair:** Claudia Doege (Columbia University Medical Center) |
| 9:10-9:30 | 20min | Short talk 1 - Brian Elbel (NYU School of Medicine) - "Childhood Obesity and the Food Environment: A Population-Based Sample of Public School Children in New York City" |
| 9:30-9:50 | 20min | Short talk 2 - Nan Shen (Icahn School of Medicine at Mount Sinai) - "Microbial Metabolomics After Bariatric Surgery in Humans" |
| **9:50-10:40** | **5 intro+45min** | **Keynote 1: Kelly Brownell (Duke University) - "Harnessing Research to Create Nutrition-Related Policy Changes"**<br>**Introduction: Rudy Leibel** |
| 10:40-11:10 | 30min | Coffee/Networking/Poster viewing |
| | | **Chair:** Alpana Shukla (Weill Cornell Medicine) |
| 11:10-11:30 | 20min | Short talk 3 - Suraj Teegala (Rutgers Graduate School of Biomedical Sciences) - The Glucose Sensitivity of Lateral Hypothalamic Area (LHA) Orexin Glucose-inhibited (GI) Neurons may influence reward-based feeding via Modulation of Ventral Tegmental Ara (VTA)Dopamine (DA) Neurons |
| 11:30-11:50 | 20min | Short talk 4 - Vidhu Thaker (Columbia University Medical Center) - "Novel model of the Child Eating Behavior Questionnaire in Children with Severe Early onset Obesity" |
| 11:50-1:30 | 100min | Lunch/Networking/Poster viewing and presentations (between 12:30 and 1:30) |
| | | **Chair:** Paul Cohen (Rockefeller University) |
| 1:30-1:50 | 20min | Short talk 5 - Abhishek Vishnu (Icahn School of Medicine at Mount Sinai) - "The role of genetic and self-identified ancestry in determining obesity among African and Hispanic Americans" |
| 1:50-2:10 | 20min | Short talk 6 - Evelyn Litwinoff (New York University Langone Medical Center) - "Myeloid RAGE Downregulates Lipid Metabolism in Obese Adipose Tissue and Increases Whole-Body Obesity and Insulin Resistance" |
| **2:10-3:00** | **5 intro+45min** | **Keynote 2: Susan K. Fried (Icahn School of Medicine at Mount Sinai) - "An adipocyte is not an adipocyte is not an adipocyte: depot and sex differences in adipose growth, remodeling and function in obesity"**<br>**Introduction: Gary Schwartz** |
| 3:00-3:30 | 30min | Coffee/Networking/Poster viewing |
| | | **Chair:** Kalypso Karastergiou (Icahn School of Medicine at Mount Sinai) |
| 3:30-3:50 | 20min | Short talk 7 - Daniel Kim (Stony Brook University) - "Spatial associations between county-level income inequality and poverty with obesity rates among adults in New York State" |
| 3:50-4:10 | 20min | Short talk 8 - Loren Gianini (Columbia University Medical Center) - "Neural Mechanisms of Food Choice in Long-Term Weight Reduced Individuals" |
| 4:10-4:20 | 10min | Closing remarks, poster awards |

# Short Talk 1

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|---|---|
| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Childhood Obesity and the Food Environment: A Population-Based Sample of Public School Children in New York City |
| **Authors** * | Brian Elbel, PhD, MPH;* Kosuke Tamura, PhD; Zachary McDermott, MA; Amy Ellen Schwartz, PhD |
| **Institutional Affiliations For Each Author.** * | NYU School of Medicine, NYU School of Medicine, Institute for Education and Social Policy, The Maxwell School of Syracuse University |
| **Corresponding Author Email** * | brian.elbel@nyumc.org |

**Structured Abstract** *

Background: Some correlational studies have indicated that the food environment may play a role in shaping childhood obesity in the United States. There has been little research done to investigate this relationship using a large dataset with detailed address information that takes into account neighborhood selection and confounding. We examine the relationships between the distance from a child's home to the nearest of four different types of food outlets and childhood obesity.

Methods: We used yearly BMI measurements on 3,637,757 child-year observations (2009-2013) from New York City public school K-12 graders. We combined these data with the exact location of each food outlet type—fast-food restaurants, wait-service restaurants, corner stores, and supermarkets—to calculate the distance to the nearest food outlet. Our primary estimation strategy used census-tract level fixed effects.

Results: Having the nearest fast-food outlet more than 0.025 miles (about 1/2 of a city block) from home was associated with lower obesity, overweight and zBMI. Results ranged from 2.0 to 4.3 percent decreased obesity. Similar results were seen for corner stores further than 0.05 miles, ranging from 1.3 to 3.0 percent decreased obesity. Results for wait service and supermarkets were not consistently associated with obesity.

Conclusions: Distances to nearest fast-food restaurant and corner store were consistently, inversely related to childhood obesity. These findings can help better inform policies focused on food access in urban areas, which could, in turn, reduce childhood obesity.

# Short Talk 2

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Microbial Metabolomics After Bariatric Surgery in Humans |
| **Authors** * | Nan Shen1, Michael Ahlers2, Assumpta Caixàs3, Zhan Gao4, Ankit Shah3, Martin Blaser5, Jose C. Clemente1, Blandine Laferrère2,3 |

**Institutional Affiliations For Each Author.** *

1Icahn Institute for Genomics & Multiscale Biology Department of Genetics and Genomic Sciences Department of Medicine, Division of Clinical Immunology Icahn School of Medicine at Mount Sinai, New York, USA; 2Obesity Research Center, Department of Medicine, Columbia University College of Physicians and Surgeons; 3Division of Endocrinology, Department of Medicine, Columbia University College of Physicians and Surgeons, New York, USA; 4Endocrinology and Nutrition Department. Parc Taulí Hospital Universitari, Institut d'Investigació i Innovació Parc Taulí (I3PT)-UAB, Sabadell, Spain; 5New York University Langone Medical Center, New York, USA

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| **Corresponding Author Email** * | bbl14@columbia.edu |

**Structured Abstract** *

Background: The gut microbiome, which plays a role in obesity and metabolism, could be one of the mediators of the beneficial metabolic effects of bariatric surgery. We wished to identify changes in biomarkers of microbial metabolism and microbial composition associated with improved metabolism after bariatric surgery. Hypotheses: 1) Bariatric surgery will increase microbiome diversity; 2) The change in microbiota will be associated with decreased inflammation, increased pool of bile acids (BAs), and altered short chain fatty acids (SCFA) and products of choline metabolism.

Methods: Candidates for bariatric surgery (n=18, BMI= 46.1 ±6.3 kg/m2, age 42±12y) were recruited at 2 sites: New York, USA, and Sabadell, Spain. Fecal and blood samples were collected before and at 3, 6, and 12 months after surgery to determine microbiome composition by 16S analysis, and circulating concentrations of hsCRP, substance amyloid A (SAA), SCFA, BAs, trimethylamine N-oxide (TMAO), choline and betaine, glucose and lipids.

Results: Microbiota composition did not differ between the 2 sites prior to surgery, suggesting that the severe obesity phenotype plays a more significant role than environmental factors in shaping gut bacterial composition. Both diversity and composition of the gut microbiota changed significantly after surgery, with diversity increasing until 6 months then decreasing at 12 months. TMAO and BAs increased significantly after surgery, while inflammation markers, glucose and insulin decreased. The abundance of Akkermansia, a bacteria associated with healthier metabolism, increased after surgery but returned to baseline levels at 12 months. Furthermore, groups of co-occurring bacteria (as opposed to isolated bacteria) correlated with various biomarkers after surgery. At 3 months, Blautia and Eggerthella correlated with each other and with butyrate, a bacterial SCFA with anti-inflammatory properties. The correlation with butyrate was also observed at 6 months but with Blautia and Coproccocus instead. Additionally, HDL levels increased 6 months after surgery and was strongly correlated with Atopobium, Rothia and Peptostreptococcaceae. These data suggest that the synergistic effect of bacterial consortia may play a role to decrease inflammation and improve metabolism. However, these microbiota-metabolites correlation profiles changed in the 12 months after surgery. This suggests different mechanisms for early and late changes after surgery and highlights the importance of longitudinal studies to interpret the role of the microbiome on metabolism after surgical weight loss.

Conclusions: These findings indicate that changes in gut microbial composition and its function may be potential mediators of decreased inflammation and of improved metabolism after surgical weight loss.

# Short Talk 3

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | The glucose sensitivity of lateral hypothalamic area (LHA) orexin glucose-inhibited (GI) neurons may influence reward-based feeding via modulation of ventral tegmental area (VTA) dopamine (DA) neurons |
| **Authors** * | Suraj B. Teegala*, Zhenyu Sheng, Usman Khan, Miloni Dalal, Mark. P. Thomas, Vanessa. H. Routh |
| **Institutional Affiliations For Each Author.** * | Rutgers Graduate School of Biomedical Sciences Newark New Jersey*, Department of Pharmacology, Physiology and Neuroscience Rutgers New Jersey Medical School Newark New Jersey, Rutgers Graduate School of Biomedical Sciences Newark New Jersey, Rutgers Graduate School of Biomedical Sciences Newark New Jersey, School of Biological Sciences University of Northern Colorado Greeley CO. |
| **Corresponding Author Email** * | routhvh@njms.rutgers.edu |

**Structured Abstract** *

Obesity is a multifactorial disease which represents a major risk factor for cardiovascular diseases, some forms of cancer and Alzheimer's disease. One of the leading predictors for obesity is non-homeostatic feeding which occurs when signals of energy homeostasis are overridden. The neural circuits regulating energy homeostasis are well studied. However, their role in weight loss maintenance is relatively unknown. Signals of peripheral energy status influence non-homeostatic/reward-based feeding by modulating the glucose sensitivity of lateral hypothalamic area (LHA) orexin glucose-inhibited (GI) neurons. Low glucose directly activates ~60% of LHA orexin neurons. Leptin indirectly enhanced and ghrelin directly reduced this inhibitory effect of glucose. LHA orexin neurons project to ventral tegmental area (VTA) dopamine (DA) neurons where orexin induces glutamate plasticity. VTA DA neurons are involved in reward-based feeding behavior. We hypothesized that activation of LHA orexin-GI neurons in low glucose persistently enhances glutamate neurotransmission onto VTA DA neurons and reinforces reward-based feeding behavior. We first measured spontaneous N-methyl-D-aspartate (NMDA) and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor mediated glutamate postsynaptic currents on VTA DA neurons using voltage clamp recording in horizontal mouse brain slices containing the LHA. Lowering glucose from 2.5 to 0.25 or 0.7 mM increased NMDA current amplitude (n=8; $P<0.05$). This was blocked by an orexin antagonist suggesting that activation of orexin-GI neurons mediated the effect of low glucose. Fasting increased the AMPA/NMDA current amplitude ratio, a measure of in vivo glutamate plasticity (n=7, P=0.03). Next, we dialyzed the LHA with a range of glucose concentrations seen in the brain from fasting to hyperglycemia (0.7 – 4 mM) and measured conditioned place preference (CPP). Rats were food restricted to 85% of their body weight, bilaterally implanted with microdialysis guide cannula aimed 1mm above the LHA and maintained at 85% body weight. They were then conditioned with a positive reinforcer (chocolate) using a two compartment CPP apparatus. On day 7, animals were analyzed for CPP. On day 8 glucose was dialyzed and time spent in each compartment in the absence of chocolate was measured. The difference in time for the conditioned side on day 8 vs day 7 was calculated as an index of CPP. There was a significant negative correlation between glucose concentration and CPP ($P<0.05$, $R2=0.963$, n= 15). Our data suggest that the glucose sensitivity of LHA orexin-GI neuron may link metabolic status to reward based-feeding by altering glutamate plasticity on VTA DA neurons.

# Short Talk 4

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Novel model of the Child Eating Behavior Questionnaire in Children with Severe Early onset Obesity. |
| **Authors** * | Vidhu V Thaker*, Arielle Hoffman, Ashley Shoemaker, Molly McDonald, Sinead Christensen, Michaela Banks, Benjamin Weaver, Joel Hirschhorn and Stavroula Osganian |
| **Institutional Affiliations For Each Author.** * | Columbia University Medical Center, Mailman School of Public Health, Vanderbilt University Hospital, Massachusetts General Hospital, Boston Children's Hospital, University of Virginia, Boston University, Boston Children's Hospital, Boston Children's Hospital |
| **Corresponding Author Email** * | vvt2114@cumc.columbia.edu |

**Structured Abstract** *

Background: Child Eating Behavior Questionnaire (CEBQ) is a validated instrument for measuring eating patterns in young children. However, little is known about the validity of this instrument in children with severe obesity. We sought to study the subscales of CEBQ and use the established 7-factor model in children with severe early onset obesity.

Methods: Children with severe obesity were recruited from the Genetics of early childhood obesity (GECO) study and Obesity registries at 2 tertiary care hospitals (n = 289). Parents of children < 13 years of age completed the CEBQ. We compared this data to a simulated cohort (n = 1000 of normal weight children. We performed a confirmatory factor analysis (CFA) of the validated 7-factor construct of CEBQ. Due to inadequate model fit, we performed an exploratory factor analysis (EFA) to identify the model for our cohort.

Results: There was internal consistency in the responses, Cronbach's-$\alpha$ = 0.9. The parallel analysis and the scree plot identified 7 principal components with eigenvalue > 1.0. In the children with obesity, the constructs of Food Responsiveness, Enjoyment of Food, Emotional overeating and Desire to drink were significantly higher, and lower for Satiety Responsiveness, Slowness in Eating, Emotional under eating and the Fussiness scale. The scales were similar across gender, race and ethnicity. In the CFA, the established 7-factor model showed a sub-optimal fit for the data (RMSEA = 0.089, CFI/TLI = 0.887/0.875). The EFA identified a novel 7-factor model with constructs of Food Fascination (avidity) and Food Capacity (volume). Eating with happiness changed from Emotional undereating to Emotional overeating. These constructs were significantly related to the obesity class and older age ($p < 0.01$).

Conclusions: CEBQ is valid for assessment of appetitive traits in children with severe obesity. The factor analysis of CEBQ revealed novel latent constructs of eating behavior for personalized care and assessment of hyperphagia.

# Short Talk 5

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | The role of genetic and self-identified ancestry in determining obesity among African and Hispanic Americans |
| **Authors** * | Abhishek Vishnu, Gillian Belbin, Eimear Kenny, Erwin Bottinger, Ruth J F Loos |
| **Institutional Affiliations For Each Author.** * | AV, RJFL – The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York. 2. The Genetics of Obesity and Metabolic Traits Program, Icahn School of Medicine at Mount Sinai, New York<br><br>GB, EK, EB – The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York |
| **Corresponding Author Email** * | ruth.loos@mssm.edu |

**Structured Abstract** *

Background: Individuals of African and Hispanic ancestry are at higher risk of obesity than individuals of European ancestry in the United States (US). This may be due to differences in environmental factors, such as cultural and social practices, but may also in part be genetically driven. Here, we examined to what extent African ancestry is associated with body-mass index (BMI) among 6,368 African-American (AA) and 7,569 Hispanic American (HA) participants in the New York City based BioMe TM biobank. Methods: We quantified global genetic ancestry using ADMIXTURE, specifying two putative ancestral populations (k=2) to distinguish African from non-African ancestry. We excluded participants with significant Native American ancestry i.e. ≥30% (determined with k=3). We used linear regression to examine association between percentage African ancestry and BMI among AAs and HAs. Covariates examined were age, sex, self-reported ancestry (HA vs. AA), US vs. non-US born status, and their interactions. Stepwise regression procedures were used to select variables which significantly (P<0.05) contributed. Results: Participants exhibited varying levels of admixture, resulting in a wide spectrum of African ancestry present in both self-reported AAs (25th, 50th, 75th, 90th pcts: 79%, 86%, 92%, 96%, respectively) and HAs (15%, 26%, 41%, 60%, respectively). Among AAs and HAs, 18% and 60% of participants are non-US born, respectively. As analysis showed a strong interaction between ancestry and sex, we report results for men and women separately. Among women, genetic and self-reported ancestry, age and country of birth were significantly associated with BMI. However, the association differed between US-born and non-US-born women (P-interaction=0.004). For every 10% higher genetic African ancestry, BMI increases by 0.40 kg/m2 (P=1.7E-07; ~1.09kg for a 1.65m tall woman) and 0.19 kg/m2 (P=3.1E-04; ~0.51kg) among US-born and non-US born women, respectively. Independent of genetic ancestry, BMI in women who self-report as AA was 0.79 kg/m2 (P=1.2E-02) lower than self-reported HAs. In contrast, among men self-reported HA status (+0.96 kg/m2, ~2.94 kg for a 1.75m tall man) and being US-born (+1.37 kg/m2, ~4.21kg) influenced BMI, while no significant association was detected for genetic ancestry (P=0.2). Conclusions: Besides the contribution of environmental factors, genetic ancestry contributes to variation in BMI among HA and AA women. However, among men, only country of origin and self-reported ancestry play a role.

# Short Talk 6

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Myeloid RAGE Downregulates Lipid Metabolism in Obese Adipose Tissue and Increases Whole-Body Obesity and Insulin Resistance |
| **Authors** * | Evelyn MS Litwinoff*, Carmen Hurtado del Pozo, Ravichandran Ramasamy, Ann Marie Schmidt |
| **Institutional Affiliations For Each Author.** * | New York University Langone Medical Center |
| **Corresponding Author Email** * | Evelyn.Litwinoff@nyumc.org |

**Structured Abstract** *

In obesity, the development of insulin resistance (IR) is connected to the presence of inflammation in visceral adipose tissue (VAT), which is characterized by changes in VAT macrophage content and inflammation. These adipose tissue macrophages (ATM) are filled with lipids in processes linked to the development of IR, as macrophage lipid depletion prevents IR. However, the underlying mechanisms are not known. The receptor for advanced glycation end products (RAGE) induces inflammatory signaling in multiple cell types, including macrophages. RAGE-null (RKO) mice fed a high fat diet (HFD; 60% kcal/fat) are protected from weight gain and development of IR, and display reduced VAT macrophage content. We hypothesized that RAGE deletion in macrophages alters lipid metabolism to facilitate ATM lipid-loading during obesity. We performed a transcriptomics Affymetrix microarray analysis on wild-type (WT) versus RKO sorted ATMs from HFD fed mice and report that "lipid metabolism signaling" pathways are significantly downregulated in the RKO ATMs by CAMERA gene set enrichment analysis. Full scale lipidomics analyses also show a decrease in 40 different lipid species in RKO versus WT bone marrow derived macrophages (BMDM). RKO BMDMs challenged with fatty acids in the Seahorse XFe24 display lower oxygen consumption rates as compared to WT BMDM, which is rescued by conditioned media from RKO adipocytes that restores oxygen consumption to WT levels. Metabolic nutrient tracing studies are underway to discern if or how fatty acid □-oxidation may be altered in RKO versus WT BMDMs. To probe the myeloid-specific roles for RAGE in HFD feeding, we generated mice with a myeloid-specific deletion of RAGE (MDR). Strikingly, after one month of HFD, MDR mice are significantly more obese and IR as compared to control mice. Metabolic cage and hyperinsulinemic-euglycemic clamp studies are underway to identify the tissue specific-sites mediating IR and to assess any changes in energy expenditure. Taken together, these studies unveil a novel connection between myeloid RAGE, altered lipid metabolism, and IR. Further, this work adds to the body of evidence that RAGE has distinct roles in different cell types, and suggests an important role of RAGE in the crosstalk between ATMs and adipocytes. Studies are in progress to discern the mechanism connecting RAGE to decreased lipid metabolism and increased whole body IR.

# Short Talk 7

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Spatial associations between county-level income inequality and poverty with obesity rates among adults in New York State |
| **Authors** * | Daniel Kim*, Chrisa Arcan, Fusheng Wang |
| **Institutional Affiliations For Each Author.** * | Department of Biomedical Informatics and Department of Computer Science,<br>Department of Family Population and Preventive Medicine, Department of Biomedical Informatics and Department of Computer Science, Stony Brook University |
| **Corresponding Author Email** * | hrisanti.arcan@stonybrookmedicine.edu |

**Structured Abstract** *

Purpose: In addition to individual economic factors and area poverty, income inequality has been found to influence individual health outcomes and obesity. Studies have demonstrated correlation between socioeconomic factors and obesity, but a spatial-based approach can account for interactions between neighboring areas that standard methods of regression are not equipped to achieve. An approach based on spatial statistics may generate new insights about the relationships between county-level income inequality, poverty, and obesity across New York State (NYS).
Methods: The economic and sociodemographic data were drawn from the ACS (American Community Survey) 2015 and obesity data were drawn from CDC 2013 to examine correlations between multiple economic factors and obesity, including county-level income inequality, county-level poverty percentages, and median personal income across NYS. County-level income inequality was measured using the GINI index, a measure of statistical dispersion (higher GINI coefficient indicates a higher level of area income inequality.) The ACS data were collected through individual interviews and area estimates were derived. In this study ACS 5-year estimates (2011-2015) were used. Spatial mapping was conducted with ArcGIS software; ArcGIS's spatial-based OLS (Ordinary Least Squares) modeling was used to examine associations between county-level obesity percentages and county-level GINI index, county-level poverty rates, and individual income, adjusted for race, age, education, marital status, and US birth. Level of significance was set at $p<0.05$ two-tailed test.
Results/Findings: Between 2011 and 2015 a total of 790051 adults were interviewed in New York State. Higher income inequality and higher county-level percentages of Hispanic populations was associated with lower county-level obesity rates in males and females combined; however, among females income inequality and obesity were not significant. An increase in poverty percentage was associated with increased county-level obesity rates in both males and females. Among males, married status was linked to increased obesity rates, but for females this association was not significant. In females only, being African-American was associated with high obesity rates.
Conclusions: Increased county-level income inequality and lower county-level poverty rates were linked to lower obesity rates across New York State. These findings may indicate that at the county level, economically heterogeneous areas may include positive environmental factors that can benefit low-income residents.

# Short Talk 8

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Neural Mechanisms of Food Choice in Long-Term Weight Reduced Individuals |
| **Authors** * | Loren Gianini, PhD, Karin Foerde, PhD, Joanna Steinglass, MD, B. Timothy Walsh, MD, Laurel Mayer, MD |
| **Institutional Affiliations For Each Author.** * | Columbia University Medical Center, University of Amsterdam, Columbia University Medical Center, Columbia University Medical Center, Columbia University Medical Center |
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**Structured Abstract** *

While many individuals with obesity are able to lose weight, a striking minority are able to sustain substantial weight loss for a significant length of time. It is critical to understand the cognitive and neural systems underlying food-choice decision making associated with successful, long-term weight loss maintenance. Studies suggest the eating behavior of Long-term Weight Reduced individuals (LOWER) is characterized by repeated choice of low calorie foods from a limited diet variety. The purpose of this pilot study was to explore the brain regions and neural circuits supporting food choice-decision making in LOWER individuals (those who have sustained weight loss of 30 lbs or more for 12 months or longer) and BMI-matched healthy controls (HC). To date, 17 LOWER individuals and 7 HC have participated in a Food Choice task with functional magnetic resonance imaging (fMRI) to examine brain activity while individuals engaged in a Food-Choice Task during which they were asked to assess the value of food and make a series of choices between two foods of which food they would prefer to eat. Upon completion of the task, one of their choices was selected at random, and participants were given a snack-sized portion to eat. Whole brain analyses during food choice revealed significant activation during choice in the VMPFC in both HC and LOWER groups, suggesting engagement of reward-sensitive circuits. In the LOWER group, food choices were associated with dorsal striatum activity. However, dorsal striatum activation was not found in the HC group, even at a more lenient threshold ($p < 0.005$ uncorrected for whole-brain comparison). This engagement of the dorsal striatum supports the possibility that LOWER activate reward-insensitive, habit systems during food-choice decision-making. These results are an important step in identifying a neurobiological locus that relates to the core behavior of successful weight loss maintenance.

# Board 1, Poster 1

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|---|---|
| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Deep RNA-sequencing uncovers a broad species- and cell-specific lincRNA repertoire that modulates human macrophage activation and associates with cardiometabolic diseases |
| **Authors** * | Hanrui Zhang 1*, Chenyi Xue 1, Ying Wang 1, Xuan Zhang 1, Wenjun Li 2, Sara Nunez 3, Andrea S. Foulkes 3, Jennie Lin 4, Christine C. Hinkle 2, Wenli Yang 5, Edward E. Morrisey 2, 5, 6, Daniel J. Rader 7, Mingyao Li 8, and Muredach P. Reilly 1, 9 |

**Institutional Affiliations For Each Author.** *

1 Division of Cardiology, Department of Medicine, Columbia University Medical Center, New York, New York, USA. 2 Cardiovascular Institute, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA. 3 Department of Mathematics and Statistics, Mount Holyoke College, South Hadley, Massachusetts, USA. 4 Department of Medicine, Perelman School of Medicine at the University of Pennsylvania, USA. 5 Institute for Regenerative Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA. 6 Department of Cell and Developmental Biology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA. 7 Departments of Genetics and Medicine, Division of Translational Medicine and Human Genetics, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA. 8 Department of Biostatistics and Epidemiology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA. 9 Irving Institute for Clinical and Translational Research, Columbia University, New York, New York, USA

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**Structured Abstract** *

Background: Sustained and dysfunctional macrophage activation promotes inflammatory cardiometabolic disorders (CMDs), but the role of long non-coding RNAs in human macrophage activation and CMDs is poorly defined. We aim to elucidate the long intergenic non-coding RNA (lincRNA) landscape of human macrophages through RNA-sequencing (RNA-seq), genomics and bioinformatics, as well as selective translational and functional studies.
Methods: We used deep RNA-seq and de novo assembly to examine the lincRNA transcriptome of human monocyte-derived macrophages (HMDM) at resting and stimulated with lipopolysaccharide and interferon-gamma (IFN-γ) for M1-activation, and interleukin-4 for M2-activation.
Results: We identified 2,766 macrophage lincRNAs including 861 previously unannotated ones. The majority (~85%) was non-syntenic, or syntenic but not annotated, in the mouse genome. Many macrophage lincRNAs also demonstrated tissue-enriched transcription patterns (21.5%) and enhancer-like chromatin signatures (60.9%). Macrophage activation, particularly to the M1 phenotype, markedly altered the lincRNA expression profiles revealing 96 lincRNAs differentially expressed (fold-change >2 and FDR <0.01), suggesting potential role of many lincRNAs in regulating macrophage inflammatory functions. A subset of macrophage lincRNAs overlapped genome-wide association study loci for CMDs. For example, MacORIS, a syntenic lincRNA not annotated in the mouse genome, harbors variants associated with central obesity. MacORIS is macrophage-enriched and predominantly located in cytoplasm. Knockdown of MacORIS enhanced IFN-γ induced JAK2/STAT1 phosphorylation, suggesting its potential role as a repressor of IFN-γ signaling. Induced pluripotent stem cell-derived macrophages (IPSDM) recapitulated lincRNA transcriptome of primary macrophage and provide a high-fidelity model to study, in particular non-conserved, lincRNAs in human macrophage biology.
Conclusions: In summary, high-resolution transcriptomics identified 100s of lincRNAs that form part of the coordinated response during macrophage activation including specific macrophage lincRNAs associated with human CMDs that modulate macrophage inflammatory functions.

# Board 1, Poster 2

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Influence of Choline Derivatives on Placental Macronutrient Uptake and Transport |
| **Authors** * | Khatia Nanobashvili*, Rachel Bretter, Chauntelle Jack-Roberts, Kali Olivia Celine Blain, Bhoomi Dave, Anjana Saxena, Kathleen Axen, Xinyin Jiang |
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**Structured Abstract** *

Gestational diabetes mellitus (GDM) is characterized with excessive fat and glucose transport through the placenta to the fetus, resulting in fetal overgrowth. Our previously study suggests that supplementation of choline, an essential nutrient, normalizes fetal growth in GDM mice at mid-gestation. In this study, we further assessed the influence of choline and its oxidized derivative betaine on placental nutrient transport in GDM mice and human trophoblasts. Female C57BL6/J mice were a fed 60% kcal high-fat (HF) diet 4 weeks prior to timed-mating and during gestation to induce GDM or fed a control normal fat diet. The HF mice received 25mM choline chloride, 1% betaine, or control drinking water daily. Placentas were collected at embryonic day (E)12.5 and (E) 17.5 for histologic analysis. We found that the area of blood vessel lumen was larger ($P < 0.05$) in the HFcontrol versus the LFcontrol group, indicating increased blood flow, yet both choline and betaine supplementation prevented this change. HFcholine group also had smaller ($P < 0.05$) areas of hollow space in the junctional zone of the placenta than the other groups at E17.5, suggesting that the HFcholine group had a more developed junctional zone. To further examine the effect of choline derivatives on trophoblast functions under hyperglycemia, the human trophoblast cell lines, BeWo and JEG-3, were cultured under high (4.5 g/L) or low (1 g/L) glucose and treated with saline placebo, choline (1 mM) or betaine (1 mM) for 48 hours. While high-glucose increased ($P < 0.05$) the gene expression of glucose metabolizing genes such as glucose transporter 3, glucose transporter 1, and glucose synthase 1 in BeWo but not in JEG-3, betaine supplementation alleviated the increases. Choline and Betaine significantly decreased glucose uptake into the trophoblast cells under both low and high glucose conditions in both cell lines. In conclusion, choline and betaine modified placental responses to high-glucose in in vitro and mouse models.

# Board 1, Poster 3

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | A multi-trait genetic association approach to identify genetic loci not identified before in single-trait GWAS of lipid traits |
| **Authors** * | Michael H. Preuss*(1,2)<br>Girish N. Nadkarni (1)<br>Ruth J.F. Loos (1,2,3) |
| **Institutional Affiliations For Each Author.** * | 1 The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai<br>2 The Genetics of Obesity and Related Metabolic Traits Program, Icahn School of Medicine at Mount Sinai<br>3 The Mindich Child Health and Development Institute, Icahn School of Medicine at Mount Sinai |
| **Corresponding Author Email** * | ruth.loos@mssm.edu |

**Structured Abstract** *

Obesity, combined with a poor lipid profile, increases the risk of cardiovascular disease substantially. Large-scale genome-wide association studies (GWAS) have identified more than 360 genetic loci associated with body mass index (BMI), and circulating triglyceride, HDL- and LDL-cholesterol (HDL-C, LDL-C) levels. These GWASs have mainly focused on one single trait at a time, and the loci identified may not fully represent the biology that underlies these correlated traits. Here, we performed a multi-trait genome-wide association analysis that considers those four traits simultaneously in one analysis. We speculate that such multi-trait approach will identify genetic loci that highlight a more comprehensive biology to further elucidate the link to cardiovascular disease.

We used mtSet (Casale FP et al. 2015), a multi-trait LMM set test that enables joint analysis across multiple correlated traits and multiple sets of variants while accounting for population structures and relatedness. MtSet uses as a sliding window (of 30kb) approach, resulting in ~174,000 windows and a Bonferroni corrected genome-wide significance (GWS) of P = 2.9x10-7. Each trait was adjusted for age, and residuals were log- and rank-transformed to normality in men and women separately. Phenotype data was extracted from electronic health records (EHRs) for 1,560 African ancestry (AA) and 1,982 Hispanic ancestry (HA) participants of the Mount Sinai BioMe BioBank for whom 1000 Genomes (Phase 3) imputed data was available.

No loci reached genome-wide significance, but six reached suggestive significance (P<10-5), covering NAALADL2 (P=1.5x10-6), KIAA1324L (P=8.5x10-6) and a CNV at 5q14 (P=1.2x10-6) in individuals of African ancestry AA, and LMCD1 (P=8.7x10-7), a lincRNA at 4q28 (P=1.8x10-6), and ZNF431 (P=2.9x10-6) in individuals of Hispanic Ancestry.

Even though our study has still insufficient power, results are promising despite the small sample size. We are now increasing the sample size to confirm our observations and to subsequently narrow-down the loci and interpret their biological relevance. None of the suggestive loci have been previously implicated in obesity and lipid-related traits. We believe that a multi-trait genome-wide analysis provides and alternative approach to further and more comprehensively elucidate the broader biology that underlies correlated traits and diseases.

# Board 1, Poster 4

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Differential effect of FTO on laboratory meal intake in youth above and below the 50th BMI percentile |
| **Authors** * | 1. Lisa M. Ranzenhofer*, 2. Haley A. Davis, 3. Hanna K. Mielke-Maday, 4. Hailey J. McInerney, 5. Rachel Korn, 6. Nikita Gupta, 7. Tobey Linhart, 8. Ilan Dubler-Furman, 9. Wendy K. Chung, 10. Janet Schebendach, 11. Marian Tanofsky-Kraff, 12. Rudolph L. Leibel, 13. B. Timothy Walsh, 14. Michael Rosenbaum, 15. Laurel Mayer |
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**Structured Abstract** *

Background: The FTO gene (rs9939609) is associated with higher body weight, possibly via increased caloric intake. We therefore evaluated whether genotypic differences in intake are evident prior to obesity onset by examining associations among genotype and intake in 5-10y non-overweight children using preliminary data from an ongoing study.
Method: At baseline, youth were genotyped and a subset completed a multi-item ad libitum laboratory lunch meal. Height and weight were measured. ANOVAs were used to test genotypic differences in intake.
Results: Youth (n=156, age 8.3±1.5y, BMI-Z 0.29±0.99, 49% female) were genotyped and a subset (n=98) completed test meals. Genotypic distribution was 19% AA, 47% AT, and 35% TT. At baseline, there was no difference in genotype distribution between youth with BMI above and below the 50th percentile. There was a non-significant genotype effect on multi-item meal intake (AA=821±296 kcal, AT=765±275 kcal, TT=678±205 kcal, p=.11). AX (785±281 kcal) ate significantly more than TT (678±205 kcal, p=.05). Controlling for fat free mass, there were no genotype effects on intake (p's≥.30). In exploratory analysis, there was a significant genotype effect on intake in youth with baseline BMI above the 50th percentile (AA=897±58 kcal, AT=712±40 kcal, TT=702±42 kcal, p=.02), controlling for fat free mass.
Conclusions: Among children with BMI above the 50th percentile, those with AA genotype consume more calories at a multi-item laboratory meal. These data suggest that AA youth with BMI above the 50th percentile may be at greatest risk for excess weight gain.

# Board 2, Poster 5

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Linc-GPATCH2-9, a long intergenic noncoding RNA mapping to 1q41 central obesity-associated locus, modulates human adipocyte differentiation |
| **Authors** * | Authors: Xuan Zhang*, Chenyi Xue, Jane F. Ferguson, Raymond Soccio, Mingyao Li, and Muredach P. Reilly |
| **Institutional Affiliations For Each Author.** * | Columbia University Medical Center, Columbia University Medical Center, Vanderbilt University School of Medicine, University of Pennsylvania Perelman School of Medicine, University of Pennsylvania Department of Biostatistics and Epidemiology, Columbia University Medical Center. |
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**Structured Abstract** *

Background: Long intergenic noncoding RNAs (lincRNAs) are emerging as important modulators of cellular functions and human pathophysiology. Recently disease-associated single-nucleotide polymorphisms (SNPs) within or proximal to lincRNAs are increasingly revealing novel lincRNA functions in human diseases.
Methods and Results: Here we report that linc-GPATCH2-9, a non-conserved human lincRNA transcribed from the intergenic region between LYPLAL1 and SLC30A10 at 1q41, harbors SNPs associated with central obesity (waist-hip ratio adjusted for BMI) in women. 77 central obesity-associated SNPs identified in large genome-wide association studies are mapped to linc-GPATCH2-9 and its 5' regions (lead SNP rs4846567, P=2.37x10-12; ~100 kb 5' of lincRNA transcription start site,) including 21 intronic SNPs (top intronic SNP rs12031603, P=2.15x10-9). Notably, published expression quantitative trait locus (eQTL) analyses in human tissues demonstrate that these central obesity-associated SNPs show in cis-regulation for linc-GPATCH2-9, but not for nearby protein coding genes. Linc-GPATCH2-9 expression is enriched in adipose tissues, markedly induced during in vitro human adipocyte differentiation and elevated in obese humans. Interestingly, linc-GPATCH2-9 abundance is lower in visceral adipose than subcutaneous adipose of obese patients. Chromatin immunoprecipitation sequencing (ChIPseq) in human adipocytes demonstrates high occupancy of PPARγ in the 5' region of linc-GPATCH2-9, suggesting PPARγ may mediate linc-GPATCH2-9 expression in human adipose. In addition, many central obesity-associated SNPs overlap with PPARγ-binding sites near linc-GPATCH2-9. Linc-GPATCH2-9 knockdown markedly impairs adipogenic differentiation of human adipose stromal cells (ASC), resulting in over 65% decrease in triglyceride content as well as 40-70% reduction of adipogenic gene expression (e.g. PPARγ, CEBP, SREBF1 and FASN).
Conclusions: In summary, our results demonstrate a regulatory role of linc-GPATCH2-9 in human adipocyte differentiation and provide supportive evidences for linc-GPATCH2-9 association with central obesity susceptibility in this 1q41 locus.

# Board 2, Poster 6

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Choline and Placental Macronutrient Metabolism in Gestational Diabetes-Complicated Pregnancies |
| **Authors** * | Chauntelle Jack-Roberts, Madar Dalloul MD, Michelle Haughton-Miller MD, John Kral MD-PhD, Xinyin Jiang PhD |
| **Institutional Affiliations For Each Author.** * | Chauntelle Jack-Roberts, Xinyin Jiang PhD - Brooklyn College, Madar Dalloul MD, Michelle Haughton-Miller MD, John Kral MD-PhD SUNY Downstate Medical Center |
| **Corresponding Author Email** * | Chauntelle.r@gmail.com |

**Structured Abstract** *

Background: Gestational diabetes mellitus (GDM), characterized by hyperglycemia and glucose intolerance onset, complicates 4.6 - 9.2% of pregnancies in the United States. Maternal obesity is a major risk factor of GDM. Macrosomia, defined as a birth weight greater than 4kg, is one of the leading neonatal complications of GDM which has lasting influence on affected infants, increasing their risk of cardio-metabolic diseases in adulthood. Increased placental transport has been proposed as a mechanism by which fetuses amass excess macronutrients and develop macrosomia. Choline, a semi-essential nutrient that participates in lipid metabolism and transport, normalizes fetal growth and placental macronutrient transport at mid-gestation in GDM mice in our prior study.
Objective: In this study, we seek to determine whether choline intake/status is associated with fetal growth outcomes in human pregnancies affected by GDM.
Method: We are recruiting GDM and non-GDM pregnant women 20 - 33 weeks gestation from SUNY Downstate Medical Center. We collect their 3-day 24-hr dietary recalls to assess choline intake using the NDSR software and fasting blood samples to assess plasma choline metabolite status via LC/MS. We also collect the placentas to assess nutrient transporter expression using real-time qPCR and birth outcome data at delivery. Final data was analyzed using IBM SPSS Statistics. Data are presented as mean ± standard deviation.
Results: Our preliminary results include 10 non-GDM and 10 GDM women. The GDM group had higher pre-pregnancy BMI than the non-GDM group (GDM: 33.8±8.5 vs Control: 25.5±4.1). The two groups demonstrated numerical differences in choline intake (GDM: 448.2±113.2 vs Control: 485.5±265.6 mg) and blood choline metabolite concentrations (GDM: 67.6±137.6 vs Control: 7.5±1.2 μmol/L) but did not reach statistical significance. Birth weight and macrosomia incidence did not differ between GDM and non-GDM participants and were not associated with choline intake or status in this pilot dataset. Placental fatty acid transport protein 1 (FATP1) expression was positively associated with the expression of choline pathway enzymes [(phosphatidylethanolamine methyltransferase (PEMT) $p=0.039$ and choline-phosphate cytidylyltransferase A (PCYT1A) $p\leq0.003$ and plasma choline concentrations ($p\leq=0.051$).
Conclusion: The preliminary data provides initial evidence that choline status may be altered in GDM pregnancies and that choline metabolism in the placenta may be correlated with placental macronutrient transport. Further study with a larger sample size will is needed to provide more insights into the correlation of maternal choline status with GDM birth outcomes and placental function.

# Board 2, Poster 7

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Understanding and predicting childhood obesity at individual and population levels using the NYU Langone Comprehensive Program on Obesity's DataBridge. |
| **Authors** * | Narges Razavian*, Melanie Jay, Yindalon Aphinyanaphongs, Po Lai Yau, Mary Jo Messito, Michelle Katzow, Pasquale Rummo, Benjamin Spoer, Courtney Abrams, Christopher Bates, Brian Elbel |
| **Institutional Affiliations For Each Author.** * | NYU School of Medicine for all authors, except Brian Elbel (NYU School of Medicine & NYU Wagner School of Public Service) |
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**Structured Abstract** *

BACKGROUND: Childhood obesity is a pressing public health and policy concern in the United States. Nationally, nearly 17% of children are obese, and even more are overweight and at high risk of obesity later in life. Literature suggests a range of prenatal and early life factors—such as high maternal pre-pregnancy BMI and high infant birth weight—as contributory to obesity outcomes, as well as a number of population factors, including ethnicity, socioeconomic status and features of both the food and built environments. However, despite this wealth of knowledge, current standard methods allow us to predict only 7-10% of the variance in childhood obesity. We hypothesized that the use of machine learning techniques would allow us to better predict the obesity outcomes for 2-year-old children at the age of 5.

METHODS: To test this, we gathered the electronic health records (EHRs) of 52,945 children and their mothers who had visited Lutheran Family Health Centers in Brooklyn, New York, at least twice between 2008 and 2016. Of this sample, 1,896 girls and 1,954 boys had their BMI measured at the ages of both 2 and 5. We created up to 85,645 variables from the health records of these children, which include one indicator for every possible diagnosis, and an indicator for child and mother's ethnicity, mother's delivery age, and the latest vital sign measured before age of 2.

RESULTS: Our initial analysis—including Lasso Regression, Random Forest Regression, and Multi-layered Perceptron—has yielded an area under the curve of .82 for predicting the BMI of 2-year-old children at the age of 5. This analysis revealed standard predictors such as high BMI, but also novel predictors related to infection and cardiovascular issues before the age of 2. It was also found that ethnicity and mother's country of origin had higher predictive value of obesity for girls than boys.

CONCLUSIONS: From initial analyses, machine learning techniques applied to pediatric EHR data appear to be an effective mechanism for identifying previously unknown factors that contribute to childhood obesity. Ongoing and future work will include incorporation of additional clinical data such as lab measurements and medication utilization, as well as integration of population data related to the food and built environments. In addition, we plan to include temporal trends of the vital signs as predictors using Gaussian processes and recurrent neural networks.

# Board 2, Poster 8

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | A Phenome-Wide Association Study (PheWAS) of the common variant PNPLA3.pI138M reveals novel disease associations that broaden its clinical implications. |
| **Authors** * | *Ryan W Walker1, *Gillian M Belbin1, Elena P Sorokin2, Genevieve L Wojcik2, Christopher R Gignoux2, Judy H Cho1, #Ruth JF Loos1, #Eimear E Kenny1 |
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**Structured Abstract** *

Background: Nonalcoholic fatty liver disease (NAFLD) impacts 80-100 million people in the United States and is a leading cause for liver transplantation. NAFLD can progress to steatohepatitis, cirrhosis and hepatocellular carcinoma, but little is known about the etiology of these progressions. The Ile138Met molecular alteration in PNPLA3 has been repeatedly associated with NAFLD and its severity, conferring a 2-fold higher liver fat content in homozygous vs. non-carriers of the risk allele. The risk allele (rs738409:G) is more common in Hispanic (HA) populations than in African (AA) and European (EA) ancestry populations. Insight in the extent of the effect of the risk-allele is limited and the mechanisms that underlie the associations are poorly understood. We performed a Phenome-Wide Association (PheWAS) to examine the relationship of the PNPLA3.pI138M with a broad spectrum of Electronic Health Record (EHR)-derived health outcomes.

Methods: We utilized data from the diverse New York City biobank BioMe to perform a PheWAS. We used a linear mixed model to perform association in 21,624 participants (44% HA, 30%AA, 8% EA and 17% other) who were directly genotyped for PNPLA3.pI138M using EHR-derived ICD9 billing codes (n=10,095) as the outcome variable and adjusting for age, sex, body mass index and the first 10 principal components. The threshold for significance was $p<4.95x10-6$.

Results: The frequency of the PNPLA3.pI138M risk allele was 0.35, 0.14 and 0.23 for HA, AA and EA, respectively. The risk-allele of the PNPLA3.pI138M was most significantly associated with ICD9-derived NAFLD ($\beta = 0.01$/per allele [SE±0.002]; $p = 6.62 \times 10-12$) in all individuals. Other ICD9-derived phenotypes related to liver disease also reached significance including, portal hypertension ($\beta = 0.01$[SE±0.001]; $p = 2.81 \times 10-10$), nonalcoholic cirrhosis ($\beta = 0.01$[SE±0.002]; $p = 5.57 \times 10-8$) and alcoholic liver damage ($\beta = 0.005$[SE±0.001]; $p = 3.59 \times 10-6$). Interestingly, esophageal varices diagnosis was also significantly associated with the risk allele ($\beta = 0.007$[±0.001]; $p = 6.98x 10-8$).

Conclusions: Using a PheWAS approach, we identified a novel association between PNPLA3.pI138M and esophageal varices, a phenotype with pathology that falls within the context of liver disease. Additionally, we confirmed prior GWAS associations of PNPLA3.pI138M with NAFLD, cirrhosis and portal hypertension. We show that PheWAS can harness the power of a large, genotyped biobank linked to EHRs to better describe the clinical phenotypes associated with genetic variants, and improve our understanding of the underlying biology.

# Board 3, Poster 9

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Dance for Health: An intergenerational program to increase access to physical activity |
| **Authors** * | Krista Schroeder, PhD, RN*, Sarah J. Ratcliffe, PhD, Adriana Perez, PhD, RN, CRNP, ANP-BC, FAAN, David Earley, Cory Bowman, BA, Terri H. Lipman, PhD, CRNP, FAAN |
| **Institutional Affiliations For Each Author.** * | University of Pennsylvania School of Nursing, University of Pennsylvania Perelman School of Medicine, University of Pennsylvania School of Nursing, In the Dance, University of Pennsylvania Netter Center for Community Partnerships, University of Pennsylvania School of Nursing |
| **Corresponding Author Email** * | krsch@upenn.edu |

**Structured Abstract** *

Purpose: The purpose of this study was to evaluate Dance for Health, an intergenerational program to increase access to physical activity in an underserved, high risk urban community.
Design and Methods: Dance for Health was developed using community-based participatory research methods and evaluated using an observational study design. The program entailed two hour line dancing sessions delivered by trained dance instructors in the neighborhood recreation center. The weekly sessions were delivered for one month in the spring and one month in the fall from 2012-2016. Nurse practitioner students mentored local high school students to assess outcomes: achievement of target heart rate, Borg Rating of Perceived Exertion, number of pedometer steps during dance session, Physical Activity Enjoyment Scale, and adiposity. Analytic methods included descriptive statistics and mixed effects models.
Results: From 2012-2016, 521 participants ranging from 2-79 years attended Dance for Health. Approximately 50% of children and 80% of adults achieved target heart rate. Achievement of target heart rate was not related to perceived exertion, though it was related to pedometer steps in adults. All participants rated the program highly for enjoyment. There was no change in adiposity.
Discussion: Dance for Health demonstrated high levels of community engagement and enjoyment. It led to adequate levels of exertion, particularly for adults. Changes in adiposity were not noted in this pilot evaluation; this is an expected finding given low frequency, moderate intensity, and short duration. Our evaluation can inform program scale up and future intergenerational physical activity programs.
Conclusion: Dance is an enjoyable, culturally appropriate, low cost method for increasing access to physical activity for children and families. It may be a promising modality for promoting exercise in underserved, high risk urban communities.

# Board 3, Poster 10

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The CHANGE Challenge: A Pediatric Obesity Group Intervention |
| **Authors** * | Migdalia Morel, BS, Yomiuri Ortiz, BA*, Donna Martinez, BSW, Margaret Walsh, FNP, Miriam Serrano, MSED, RDN, CDN, CDE, Alexandra Aarons, MD, Monalisa Ghose, MD, Syeda Shah, Christina McGeough, MPN, CDN, CDE, Sandra Arevalo, MPH, RD, CDN, CDE, Laura Kaplan-Weisman, MD |
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**Structured Abstract** *

Background: Pediatric obesity is a significant public health problem in the United States. At the Institute for Family Health, we have a high prevalence of pediatric obesity despite providers' best efforts at routine visits. The gold standard treatment for pediatric obesity is centered around comprehensive, intensive behavioral interventions. We sought to determine whether an intensive group intervention in a primary care setting would help obese children in our clinic improve their BMI percentile at the end of the group, 6 and 12 months after the conclusion of the group.
Methods: The CHANGE (Community, Health, Activity, and Nutrition Group Education) Challenge, an IRB approved pediatric obesity intervention modified from Montefiore's Starting Right program, met weekly for two hours over a ten week period at Walton Family Practice in the fall 2016. Using a culturally sensitive, interactive, and flexible curriculum, each session incorporated hands-on activities conducive to promoting healthy dietary and lifestyle modifications, including nutrition education, physical fitness, and the children, parents, and session leaders working together to prepare a healthy meal or snack.
Results: Fourteen unique children aged 5-11 with a BMI greater than the 85th percentile joined the group. Six participants attended at least four sessions. At the conclusion of the group, we did not find a significant improvement in BMI, BMI percentile, BMI Z-score, or blood pressure reduction. These measures will be reassessed 6 and 12 months after the conclusion of the intervention. Although attendance was low, families were actively engaged. Discussions in shared medical visits showed that they had increased knowledge about pediatric obesity and had begun dietary and lifestyle changes. Prominent challenges were recruitment, attendance, and interpersonal problems between participants.
Conclusion: Though attendance was low and BMI percentiles did not decrease at the conclusion of the group, our first CHANGE Challenge was a successful first step in addressing pediatric obesity at the IFH. Going forward, we plan to schedule the group later in the day, during warmer weather, led by a consistent team, offer better prizes, and advertise it as a necessary medical intervention. Future steps include replication and expansion to other IFH sites and publication of our findings.

# Board 3, Poster 11

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Associations between medical students' beliefs about obesity and their clinical counseling proficiency |
| **Authors** * | Victoria Fang*, Colleen Gillespie, Dennis Popeo, Ruth Crowe, Melanie Jay |
| **Institutional Affiliations For Each Author.** * | New York University School of Medicine, New York, NY, USA (all) |
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**Structured Abstract** *

BACKGROUND: There is strong evidence that genetics and heredity are a main determining factor in obesity. Yet much of society, including health care professionals, still believes that obesity is a choice, attributing obesity more to controllable behavioral issues (such as a lack of willpower) than to factors outside a person's control (such as genetic factors). Attributing obesity to controllable factors correlates with negative biases towards patients with obesity. Few prior studies have evaluated whether beliefs and attitudes about obesity in medical school students correlates with their ability to effectively communicate with and counsel patients with obesity. We evaluated 1) the correlation between medical students' beliefs about the causes of obesity and their attitudes toward people with obesity 2) whether certain beliefs about the causes of obesity correlate with negative biases; and 3) whether these beliefs and biases affect students' ability to communicate effectively with patients with obesity as measured by a standardized patient encounter (OSCE).

METHODS: Clerkship-year medical students at NYU School of Medicine completed a standardized patient OSCE that tests ability to effectively communicate with and counsel patients with obesity. Additionally, using questions primarily generated from previously published studies about obesity attitudes, we surveyed these students to evaluate their beliefs about the causes of obesity and their biases towards obese people. We analyzed the correlation between student beliefs and biases against obese people and determined the correlation between beliefs and biases to student OSCE performance.

RESULTS: The overall response rate was 60.7% (n=71). Students rated controllable factors such as unhealthy diet, physical inactivity, and overeating as more important than genetics or biological factors ($p<0.01$). Believing obesity is caused by primarily uncontrollable factors was modestly negatively correlated with negative obesity bias ($r=-0.447$; $p<0.0001$). Believing that obesity is caused by factors within a person's control was slightly negatively correlated with counseling skills $r=-0.235$; $p<0.05$).

CONCLUSIONS: This study demonstrated a correlation between attribution of obesity to external factors and greater ability to counsel a patient with obesity during an OSCE, and suggests that educating healthcare providers on the biological causes of obesity could help reduce bias and improve provider care. Limitations included the small sample size, and social desirability bias on negative bias items within the survey. Ongoing work includes exploring hierarchical linear regression analyses and future studies should evaluate whether provider attitudes towards obesity affects health outcomes for people with obesity.

# Board 3, Poster 12

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Using daily self-weighing to prevent age-related weight gain. |
| **Authors** * | Levitsky, D.A.*, Barre, L.K., Sewall, A,, Zhong, Y. |
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**Structured Abstract** *

We have published our work demonstrating that daily self-weighing can prevent age-related weight gain in (a) freshmen during their first semester at Cornell, (b) freshmen during their first year at Cornell, and (c) older adults for a two-year test period. These populations, however, do not represent the "normal" population. We have recently begun a two-year study using non-student, non-faculty, workers at Cornell to further test the effectiveness of daily self-weighing to prevent age-related weight gain. We attempted to recruit younger employees, between the ages of 20 and 40 because weight gain during this period seems to be the most powerful predictor of diabetes, hypertension, heart disease, stroke, and certain kinds of cancer. Our methods are based on the Caloric Titration Method (CTM) of controlling weight where the experimental group is given an internet based scale. The scale provides a graph showing the last seven weights. The weight data is time stamped and is immediately transmitted to our server where we can monitor the progress of each participant. We hope to expand our testing using minority populations and pre-adolescent obese children.

# Board 4, Poster 13

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Weight loss outcomes in medically managed patients on concomitant psychotropic medication |
| **Authors** * | Alpana Shukla*, Lindsay Mandel*, Ethan Litman**Louis Aronne* |
| **Institutional Affiliations For Each Author.** * | *Weill Cornell Medical College<br>**Albany Medical College |
| **Corresponding Author Email** * | aps2004@med.cornell.edu |

**Structured Abstract** *

Background: Obesity is commonly associated with psychiatric co-morbidity, and weight gain is a frequently observed side effect of several psychotropic medications. There is currently a paucity of data related to the use and effectiveness of more recently approved weight loss drugs in this population and whether weight loss outcomes are similar to or different than those of patients not on such medications.
Methods: In a retrospective study, 999 consecutive new patients with overweight/obesity seen at the Weill Cornell Comprehensive Weight Control Center between 4/1/14-4/1/15 were identified through an EPIC search. 359 patients met eligibility requirements and were included in the analysis. Demographics, medications, co-morbidities, and weight changes after initiation of pharmacotherapy were recorded during a 1 year follow-up by reviewing electronic medical records. Patients were included in either the control (no psychotropic medication, n=248) or the psychotropic medication group (n=111) based on use of antidepressants, mood stabilizers, or anti-psychotics at any point during the study period.
Results: In control and psychotropic medication groups, the mean age (49.2±14.3 vs 48.9±14.8, p=0.8), gender distribution (65.7% vs 74.8% female, p=0.99), proportion with type 2 diabetes (16.5% vs 18.0%, p=0.7), initial BMI (36.4±7 kg/m2 vs 35.6±7.1 kg/m2, p=0.3), and average number of medications taken (1.5±0.7 vs 1.4±0.7, p=0.2) were similar. At 12 months, the control group lost 2% more body weight than the psychotropic medication group (9±7.5% vs 7±8.1%, SD=8.1%, p=0.02). 69.5% of the control group achieved ≥5% weight loss and 42.4% achieved ≥10%, compared to 60.4% and 31.9% in the psychotropic medication group (p=0.22 for 5%, p=0.07 for 10%).
Conclusion: In this retrospective analysis, the concomitant use of psychotropic medication in patients treated for overweight/obesity was associated with clinically significant, albeit lower weight loss outcomes.

# Board 4, Poster 14

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Use of Antiobesity Medications Post Bariatric Surgery for Inadequate Weight Loss or Weight Regain |
| **Authors** * | Diana He*, Alpana P. Shukla, Katherine H. Saunders, Rekha Kumar, Leon Igel, Jonathan A. Waitman, Louis J. Aronne |
| **Institutional Affiliations For Each Author.** * | Weill Cornell Medicine |
| **Corresponding Author Email** * | dih2013@med.cornell.edu |

**Structured Abstract** *

Background: Although bariatric surgery is the most effective treatment for class 2 and class 3 obesity, many patients experience weight regain following bariatric surgery, resulting in the re-emergence of co-morbidities and persistence of obesity. There are limited data assessing the utility of antiobesity medications in this clinical setting.
Methods: In a retrospective study, 98 subjects seen at the Weill Cornell Comprehensive Weight Control Center from October 2012 through December 2016, and prescribed at least one antiobesity medication after bariatric surgery were identified through an EPIC query. Demographic information, medication history, and weight changes were recorded from patient electronic medical records.
Results: Mean age of subjects was 52 years (SD±11.9 years, range 26-73 years), mean BMI was 39 kg/m2 (SD±7.7 kg/m2, range 27-63 kg/m2), and 80.6% were female. In addition to obesity, baseline comorbidities included psychiatric illness (42% of subjects), dyslipidemia (40%), hypertension (35%), obstructive sleep apnea (22%), hypothyroidism (20%), type 2 diabetes (16%), CAD (12%), and PCOS (8%). 61% of subjects had Roux-en-Y gastric bypass, 25% had sleeve gastrectomy, 12% had gastric banding, 1% had Vertical Banded Gastroplasty, and 1% had duodenal switch. 85.7% were treated after regaining an average of 15.3% (SD±9.1%) of the weight lost from bariatric surgery whereas 14.3% were treated at weight plateau (defined as ≤3% total body weight regain). Median time between surgery and start of medications was 5.38 years (range 0.04 to 30 years). Mean total body weight loss was 3.8% (n=80, SD ±4.2%), 7.4% (n=57, SD±6.3%), and 7.3% (n=31, SD±6.8%) at 3 months, 6 months, and 12 months after medication initiation respectively. At 6 months, 67% lost ≥ 5% of total body weight and 30% lost ≥ 10% of total body weight (n=57). At 12 months, 61% lost ≥ 5% of total body weight and 35% lost ≥ 10% of total body weight (n=31). Subjects were prescribed a combination of FDA-approved and off-label medications, the most common of which was metformin (63.2% of subjects at 6 months, 83.9% at 12 months). The average number of medications prescribed was 1.7 medications per subject at 6 months and 2.2 medications at 12 months.
Conclusion: Antiobesity medications can be an effective tool to help patients with obesity counter weight regain and maximize weight loss post bariatric surgery. Prospective studies are needed to further evaluate the long-term effectiveness of antiobesity medications for the treatment of weight regain and inadequate weight loss after bariatric surgery.

# Board 4, Poster 15

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Pilot RCT of Technology Assisted Weight Management Intervention within Primary Care at the VA New York Harbor Healthcare System |
| **Authors** * | Irene Chen*, BS, Katja Lazar, BA, Alia Dixon, BA, John Rezkalla, BA, Clare Viglione, MPH, Melanie Jay, MD, MS, Yixin Fang, PHD |
| **Institutional Affiliations For Each Author.** * | NYU Langone Medical Center VA Harbor Healthcare System, NYU Langone Medical Center VA Harbor Healthcare System, NYU Langone Medical Center VA Harbor Healthcare System, NYU Langone Medical Center VA Harbor Healthcare System, NYU Langone Medical Center VA Harbor Healthcare System,<br>NYU Langone Medical Center VA Harbor Healthcare System,<br>New Jersey Institute of Technology. |
| **Corresponding Author Email** * | k.m.lazar@gmail.com |

**Structured Abstract** *

Background: Obesity is under-treated and primary care teams have difficulty providing effective weight management counseling. We conducted a pilot RCT of a technology-assisted intervention called Goals for Eating and Moving (GEM) to improve counseling within primary care (PC). We report 3-month outcomes of a pilot study with 2 phases.
Methods: Veterans with a Body Mass Index ≥ 30 or 25-29.99 with existing comorbidities were recruited by phone in two phases approximately six months apart, and randomized into GEM or a control group. Participants in GEM met with a health coach to set lifestyle and weight-loss goals, used a tablet-delivered goal-setting tool to facilitate in-person and four phone coaching sessions. In Phase 1, patients received baseline counseling immediately prior to their PC visit in order to activate discussion with their doctor. In Phase 2, we changed the protocol so that patient met with the health coach independent of the PC visit in order to facilitate scheduling and recruitment. In both phases, patients in the control received patient education materials. At baseline and 3 months, participants were weighed and completed surveys. Per-protocol analyses assessed the relationship between GEM and variables and also differences between phase 1 and 2, while ITT analysis assessed the 2 phases together using multiple imputations to account for missing data.
Results: 31 Veterans enrolled in phase 1 (mean age 53.48, 63% male, mean BMI 31.71, 54.84% African American, 22.58% White) and 14 enrolled in phase 2 (mean age 56.57, 79% male, mean BMI 30.27, 50% African American, 23.08% Hispanic). In phase 1, 25 Veterans returned at 3-months and in phase 2, 12 Veterans returned at 3-months. From baseline to 3-months, GEM patients in phase 1 had greater weight loss than GEM patients in phase 2 (-1.59±1.76 vs. -0.00±1.13). GEM patients overall (phase 1 and 2) had a statistically significant decrease in weight as compared to control (-1.06±1.72 vs. 00.07±2.40; p=0.03). GEM patients reported a trend toward higher dietary self-efficacy than control (6.90±9.06 vs. 3.64±11.37, p=0.16).
Conclusion: The GEM intervention was feasible within the PC setting at the Veterans Affairs and led to small amounts of weight loss at 3 months. Phase 1 patients lost significantly more weight than Phase 2, highlighting the potential importance of engaging the primary care providers in counseling. However, this finding may also be due to patient-level differences. This pilot study informed the development of a multi-site RCT of GEM (NIH # 1R01DK111928-01).

# Board 4, Poster 16

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Carbohydrate-last meal pattern lowers postprandial glucose and insulin excursions in type 2 diabetes |
| **Authors** * | Alpana P. Shukla*, Jeselin Andono*†, Samir H Touhamy*†, Anthony Casper*, Radu G. Iliescu*, Elizabeth Mauerł, Yuan Shan Zhu‡, David S. Ludwig,** Louis J. Aronne* |
| **Institutional Affiliations For Each Author.** * | *Comprehensive Weight Control Center, Division of Endocrinology, Diabetes & Metabolism, Weill Cornell Medical College, New York<br>łDept of Healthcare Policy & Research, Weill Cornell Medical College, New York<br>‡Clinical and Translational Science Center & Weill Department of Medicine, Weill Cornell Medical College, New York<br>†Institute of Human Nutrition, Columbia University, New York<br>**The New Balance Foundation Obesity Prevention Center, Boston Children's Hospital |
| **Corresponding Author Email** * | aps2004@med.cornell.edu |

**Structured Abstract** *

Background: There are limited data regarding the timing of carbohydrate ingestion during a meal and postprandial glucose regulation.
Methods: Sixteen subjects with type 2 diabetes(T2DM) consumed the same meal on 3 days in random order: carbohydrate first, followed 10 minutes later by protein and vegetables; protein and vegetables first, followed 10 minutes later by carbohydrate; or all components together. Blood was sampled for glucose, insulin, glucagon-like peptide-1(GLP-1), and glucagon measurements at baseline (just before meal ingestion) and subsequently at 30 min intervals upto 180 min.
Results: The incremental areas under the curve for glucose (iAUC0-180) and incremental glucose peaks(IGP) were 53% and 54% lower respectively, when carbohydrate was consumed last compared to carbohydrate consumed first(3124.7±501.2 vs 6703.5±904.6 mg/dl x180min, p<0.001 ; 34.7 ± 4.1 vs 75.0 ± 6.5 mg/dl, p< 0.001) and 44% and 40% lower,respectively, compared to the all components together condition(3124.7± 501.2 vs 5587.1± 828.7 mg/dlx180min, p=0.003; 34.7 ± 4.1vs 58.2 ± 5.9 mg/dl, p<0.001). Postprandial insulin excursions were lower(iAUC0-180:7354.1± 897.3 vs 9769.7±1002.1 microU/ml x min; p=0.003) and GLP-1 excursions higher (iAUC0-180 : 3487.56±327.7 vs 2519.11±494.8 pg/mlxmin; p=0.019) following the carbohydrate-last meal order compared to carbohydrate-first. Glucagon excursions were not significantly different between meal conditions.
Conclusion:In this study, we demonstrated that the temporal sequence of carbohydrate ingestion during a meal has significant impact on postprandial glucose, insulin and incretin hormone excursions. The carbohydrate-last meal pattern may be an effective behavioral strategy to improve postprandial glucose control in patients with type 2 diabetes.

# Board 5, Poster 17

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Disease Prevalence as a Function of BMI in Men and Women with Class 2 and 3 Obesity |
| **Authors** * | Jessica Weiss*, Simon Klebanov, Betty Kovacs, Richard Weil |
| **Institutional Affiliations For Each Author.** * | Mount Sinai St. Lukes Hospital |
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**Structured Abstract** *

Background: More than 2 in 3 American adults are overweight or obese; 1 in 3 are considered obese. Obesity is associated with a wide range of chronic illness and medical conditions, and it is generally assumed that as BMI increases, so does disease incidence. We were interested in the incidence of disease in our population across BMI, the odds ratios of developing one of the diseases, and a comparison of our population disease incidence to the population at large.

Methods: This is a retrospective chart review of 440 male and female adult patients enrolled in the Mount Sinai St. Luke's Weight Loss Program, a 52-week, out-patient, lifestyle-change weight loss program, where the average baseline BMI is 43.1 (SD=6.7 Class 2 and 3 obesity), and the average percent weight change after 52 weeks is 9.1% of initial body weight. We report here on data collected prior to the start of the program related to osteoarthritis, asthma, type 2 diabetes, hypertension, irritable bowel syndrome, kidney stones, migraine headaches, osteoporosis, polycystic ovarian syndrome, and thyroid disease. We present prevalence data for each disease across BMI quartiles, and performed logistic regression analyses to determine whether prevalence changes with BMI. Chi-square test was used to test for goodness of fit.

Results: For many diseases in our population, prevalence was comparable to adults in the general population, adjusted for age. For all the diseases we studied logistic regression analysis showed no statistically significant increase in prevalence (odds ratio) with increase in BMI.

Conclusions: In a population of men and women with Class 2 and 3 obesity, disease prevalence was comparable to the general population. The odds ratios of developing any of the diseases we studied was not predicted by BMI from 30.0 to 88.5 Although BMI is generally associated with an increase in disease as BMI rises, we did not find evidence to support this contention in this higher range of BMI we studied.

# Board 5, Poster 18

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Satisfaction with Weight and Quality of Life in Patients with Class 2 and 3 Obesity Following Participation in a 52-Week Lifestyle-Change Outpatient Weight Loss Program |
| **Authors** * | Richard Weil*, Betty Kovacs, Simon Klebanov |
| **Institutional Affiliations For Each Author.** * | Mount Sinai St Luke's Hospital, Mount Sinai St Luke's Hospital, Mount Sinai St Luke's Hospital |
| **Corresponding Author Email** * | rich.weil@mountsinai.org |

**Structured Abstract** *

Background: Weight loss of 5% to 10% of body weight is recommended to reduce the risk of obesity-related comorbidities. This amount of weight loss can be expected from lifestyle-change, behavioral weight loss programs. However, a 5% to 10% loss is perceived as unsatisfactory by most individuals with obesity.
Individuals with obesity also suffer significant decreases in psychological and physical quality of life (QOL) related to their weight. It is known that weight loss can improve QOL; in our weight loss program the average increase in QOL is 21.7% after 52 weeks of treatment (measured with the Impact of Weight on Quality of Life Questionnaire).
We hypothesized that percent weight loss and absolute body weight after weight loss would be positively associated with weight satisfaction and QOL.
Methods: This was a retrospective chart review of male and female adult patients with an average BMI of 43.1 (SD=6.7) enrolled in the 52-week, out-patient, lifestyle-change Mount Sinai St. Luke's Weight Loss Program. We performed linear regression analyses to determine the associations of interest. QOL and Satisfaction with Weight questionnaires were administered to all patients.
Results: At week 52, there were significant positive associations between percent weight change and satisfaction with current weight ($R2 = 0.3277$ $p<.00001$), body weight and satisfaction with current weight ($R2 = 0.1184$ $p=.0005$), and percent weight change and QOL ($R2 = 0.124$ $p<.00001$) There was a negative association between body weight and QOL ($R2 = 0.1311$ $p<.00001$). Satisfaction with weight was positively associated with QOL ($R2 = 0.1325$, $p=.0004$ $n=90$).
Conclusions: Despite weight losses in the range recommended for health, 85% of the patients did not find their weight satisfactory in any way (41%) or could accept their weight but were not happy with it (44%). The associations show that weight loss and absolute body weight may be predictors of both satisfaction with weight and improvements in QOL. Although QOL was positively associated with weight loss, the average QOL score was still relatively low (112.8 on a scale from 31 to 155), indicating that patients still have room for improvement. Patients should be stroked for losing enough weight to improve health, but clinicians should understand that the majority of patients with Class 2 and 3 obesity may not be satisfied with their weight after losing clinically significant weight and should be supported in their desire and efforts to lose more.

# Board 5, Poster 19

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Feasibility of a brief e-health Intervention to Increase Fruit and Vegetable Intake and Physical Activity Levels Among African-American Mothers and Children |
| **Authors** * | Alicia Chung, Ed.D., MPH, Azizi Seixas, PhD., and Jean-Louis Girardin, PhD. |
| **Institutional Affiliations For Each Author.** * | New York University,<br>NYU School of Medicine<br>Department of Population Health<br>Center for Healthful Behavior Change |
| **Corresponding Author Email** * | alicia.chung@nyumc.org |

**Structured Abstract** *

Introduction: Compared to Whites, African-American youth are disproportionately affected by childhood obesity. Culturally tailored electronic media solutions educate and promote positive behavior change through vicarious learning, utilizing constructs of Social Cognitive Theory during storyboarding and animation design. This study aimed to identify the feasibility of a culturally-tailored e-health tool - a website hosting avatar cartoons, to influence fruit and vegetable intake and physical activity among African-American mothers with children between 8 to 14 years old. Methods: In a sample of 93 African-American mother-child dyads, we conducted a cross-sectional study to assess the feasibility of the e-health tool to influence healthful behavior change. First, the mother and child explored the website and watched avatar cartoons focusing on the three target behaviors of interest: (1) fruit and vegetable selection on their plate, (2) food choices when outside of the home, and (3) engaging in physical activity. Second, we debriefed participants after using the e-health tool to ascertain how mothers and children individually rated the quality of the website and videos based on aesthetics, literacy level, content, and recommendations for improvement. Mothers recorded how their child rated the videos depicting characters exhibiting healthy diet and physical activity choices. Lastly, observations of re-occurring responses during content analysis informed coding and development of key themes. Results: Of the total mother-child dyads, 65% were predominantly single mothers and had an annual income between $20,000 to $60,000. Mothers were on average 38 years old. Children were nearly 50% female and were10 years of age on average. Key themes stated by children for recommending the videos included: 1. Dietary knowledge gains from watching the videos, 2. Positive representation and identification with brown-skinned avatar cartoons, 3.Vicarious learning of an urban landscape that reflected the child's neighborhood, and 4. Improvements in animation quality and graphic design. Key themes stated by mothers included: 1. Watching the cartoons broached the topic of diet with her child, 2. Dietary knowledge gains from watching the videos; 3. Wanting to share the website with other mothers in peer group and 4. Improvements in animation design. Conclusion: Participant dyads found the e-health tool to be educational, but felt animation design improvements were needed. Findings of this study indicate that cultural competent electronic media intervention hold the potential to promote healthful eating behaviors and physical activity patterns. Future research should consider interactive e-health interventions in African American families.

# Board 5, Poster 20

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Does artificial sweetener consumption impair glucose tolerance in mice? |
| **Authors** * | John I. Glendinning*, Hyunseo Lee, Abdias Sanchez, Sarah Shelling, Stephanie Hart, Young San Ryu |
| **Institutional Affiliations For Each Author.** * | Barnard College, Columbia University |
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**Structured Abstract** *

Background: There are reports that artificial sweetener consumption causes metabolic derangement in rodents (e.g., Swithers 2013; Suez et al. 2014). Interpretation of these reports is complicated by the facts that (a) the artificial sweeteners were not presented alone, but in binary mixture with sugars or yogurt; and (b) the glucose was administered intragastrically during the glucose tolerance tests (GTT), despite the fact that oral administration is more physiologically relevant and produces better glucose tolerance in rodents (Glendinning et al. 2017). The present study asked whether glucose tolerance is impaired by prolonged consumption of a solution containing an artificial sweetener, glucose or a binary mixture of an artificial sweetener plus glucose.

Methods: We used 8 week-old C57BL/6J mice. They were maintained for 4 weeks on an ad libitum diet of chow, water and a sweetened solution. We tested three artificial sweeteners, each at isopreferred concentrations: sucralose (30mM), saccharin (30mM) and acesulfame K (100mM). For comparison, we also tested glucose (0.3M), a binary mixture of glucose (0.3M) plus saccharin (30mM) [G+S], and water alone. We conducted four oral GTTs on each mouse—at baseline and 1, 2 and 4 weeks after dietary exposure commenced. Immediately prior to each GTT, the glucose was administered orally by allowing the mice to take 200 licks.

Results: The mice consumed all of the sweetener solutions avidly across the 4-week exposure period. This prolonged consumption, however, failed to impair glucose tolerance in the mice. In fact, glucose tolerance was significantly improved following exposure to the acesulfame K, sucralose, glucose and G+S solutions. It was unaffected by exposure to the saccharin solution.

Conclusions: Our results contradict the claim that artificial sweeteners cause metabolic derangement in rodents. They indicate that consumption of some artificial sweeteners actually improves the metabolic health of mice, at least with respect to glucose tolerance. The strengths of our study include the use of a within-subject design, the inclusion of three different artificial sweeteners, and the fact that glucose was administered orally rather than intragastrically during the GTT. The major limitation of our study is that it spanned only 4-weeks. We are currently exploring the contribution of diet-induced changes in insulin secretion to the improved glucose tolerance.

References

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# Board 6, Poster 21

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | IS METFORMIN POISED FOR A SECOND CAREER AS AN ANTI-MICROBIAL AGENT? |
| **Authors** * | Syed Faizan Mehdi, Faiza S.Malik, Amrat Kumar, Haroon Ali, Ashok, Anam Basharat, Wunnie Brima, Michelle Bravo, Daniel Eden, Mohammad M.Wiese, Prashant Malhotra, Jesse Roth. |
| **Institutional Affiliations For Each Author.** * | Feinstein Institute for Medical Research/Hofstra Northwell School of Medicine, |
| **Corresponding Author Email** * | jesserothmd@hotmail.com |

**Structured Abstract** *

Metformin, a widely used first-line anti-hyperglycemic, has a good safety profile, reasonably manageable side-effects, is inexpensive, and causes a desirable amount of weight loss. These favorable attributes combined with the recently discovered antimicrobial properties has led to interest in its role as a novel antimicrobial agent for the treatment of difficult-to-treat infections. In this poster, we review published studies from multiple sources (including our own lab) that have examined metformin as an antimicrobial agent. Three particular features of metformin as an anti-microbial are (i) the remarkable range of phyla that are sensitive to it; (ii) the absence of overgrowth of other organisms on prolonged use (as with hyperglycemic patients who used metformin for decades). (iii) The effective dose range for hyperglycemia is quite similar to the dose range for antimicrobial therapy.
In studies thus far (some in vivo, some in vitro) metformin has been found to have anti-microbial effects against Mycobacterium tuberculosis, Staphylococcus aureus, Pseudomonas aeuroginosa Trypanosoma cruzi, Trichinella spiralis, Hepatitis B and sepsis.

# Board 6, Poster 22

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Allograft Inflammatory Factor-1 Deficiency Counters Obesity by Enhancing Thermogenesis and Improving Glucose Handling |
| **Authors** * | Prameladevi Chinnasamy1, Aparna Srinivasan1, Isabel Casimiro1, Dario F.Riascos-Bernal1, Lander Egaña-Gorroño1, Haihong Zong2, Dippal Parikh1, Vanessa Almonte1, Jeffrey E. Pessin2, and Nicholas E. S. Sibinga1 |
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**Structured Abstract** *

Energy expenditure depends in part upon uncoupled respiration within brown and beige adipocytes, and methods that increase these activities have therapeutic potential to limit obesity and the metabolic syndrome. Studies in human populations link sequence variants at the allograft inflammatory factor-1 (AIF1) locus to obesity, adipose inflammation, and diabetes. We find that loss of AIF1 expression in mice blocks diet-induced obesity, limits white adipose depot expansion, decreases lipid accumulation in brown adipose tissues, reduces hepatosteatosis, and improves glucose handling. This protection reflects elevated basal metabolic activity, with increased thermogenic activity in brown adipocytes and increased beiging of white adipocytes. Mechanistically, Aif1-deficient mice display increased beta-adrenergic and canonical protein kinase A signaling in brown and white adipocytes, respectively. These results describe a novel function for AIF1, and point to its inhibition as a potential new strategy to prevent obesity and improve metabolic health.

# Board 6, Poster 23

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | T3 AND GLUCOSE COORDINATELY STIMULATE ChREBP-MEDIATED UCP1 EXPRESSION IN BROWN ADIPOCYTES |
| **Authors** * | Liora S. Katz*, Shiliyang Xu, Kai Ge, Donald K. Scott, Marvin C. Gershengorn |
| **Institutional Affiliations For Each Author.** * | Diabetes, Obesity and Metabolism Institute, Icahn School of Medicine at Mount Sinai, New York, NY, USA<br>Laboratory of Endocrinology and Receptor Biology, NIDDK, NIH, Bethesda, MD, USA |
| **Corresponding Author Email** * | liora.katz@mssm.edu |

**Structured Abstract** *

Introduction: T3 is known to positively regulate mitochondrial uncoupling protein1 (UCP1) and deiodinase 2 gene transcription in BAT leading to increased thermogenesis and decreased body weight. Carbohydrate-responsive element-binding protein (ChREBP) is a glucose-responsive transcription factor (TF) that plays an important role in regulating lipogenic and glycolytic genes in white adipocytes.
Methods: Adipogenesis was induced in immortalized mouse brown pre-adipocytes using minimal adipogenic media (MAM) containing DMEM, 10% resin-stripped FBS, dexamethasone, indomethacin and insulin for 2 days. T3 was added at a concentration of 10nM. Cells were analyzed at day 8 after the initial treatment. mRNA levels were measured by Illumina RNA-seq and qRT-PCR. Mitochondrial content was measured by FACS using Mitotracker®. Glucose and lactate were measured in the media after 6-8 days of adipogenesis. ChREBP was silenced and overexpressed using a lentiviral shRNA and adenovirus, respectively. TF binding sites were predicted using the Genomatix database and validated using ChIP and EMSA. In order to look at the coordianate regulation of glucose and T3 in-vivo, we used the diet induced obesity and hyperglycemic calmp mouse models.
Results: By adding MAM to BATs, adipogenic markers such as peroxisome proliferator-activated receptor gamma (PPARg), adiponectin, leptin, and UCP1 were induced. Adding T3 to MAM between days 0-2 of adipogenesis resulted in a strong upregulation of UCP1, which correlated with mitochondrial biogenesis and with the cells becoming hypermetabolic (decreased glucose:lactate ratio), but with minimal to no change in PPARg, leptin or adiponectin transcription. In addition, we found the glucose transporter 4 (Glut4) and ChREBP mRNAs to be upregulated by T3. We found that ChREBP was upregulated by T3 in BAT in both hyperglycemic mouse models. In brown preadipocytes, T3 and glucose synergistically and dose dependently upregulated Ucp1 mRNA by 1000-fold compared to low glucose concentrations. Additionally, we observed increased ChREBP and Ucp1 protein by 11.7- and 19.9-fold, respectively, along with concomitant induction of a hypermetabolic state. We confirmed binding of T3 receptor and ChREBP on the promoters of Ucp1 and ChREBP. Moreover, down-regulation of ChREBP inhibited T3 and glucose upregulation of Ucp1 by 100-fold, whereas over-expression of ChREBP upregulated Ucp1 by 5.2-fold.
Conclusions: We conclude that T3 and glucose signaling pathways coordinately regulate the metabolic state of BAT and suggest that ChREBP is a target for therapeutic regulation of BAT activity.

# Board 6, Poster 24

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Cortisol interacts with meal-induced signals to acutely regulate human adipose tissue gene expression |
| **Authors** * | Kalypso Karastergiou, MD, PhD,1,2 Pornpoj Pramyothin, MD,2,3 Ava Port, MD,2,4 Mi-Jeong Lee, PhD,1,2 Caroline M. Apovian, MD,2 Susan K. Fried, PhD1,2 |
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**Structured Abstract** *

Background: Glucocorticoids (GC) are powerful regulators of adipose metabolic and endocrine functions. GC increase insulin sensitivity in human adipose tissue in vitro and circulating leptin in the fed state in vivo, suggesting that the synergism of insulin and GC is important for adipose metabolic health.
Methods: Five healthy volunteers (3M- 2F, age 26.8±5.9y, BMI 27.8±3.1 kg/m2) completed a cross-over study with four study visits (in random order): (A) no meal/placebo, (B) no meal/cortisol, (C) meal/placebo and (D) meal/cortisol. Cortisol (30mg)/placebo was administered at 11:30 and meal (30% of daily energy reguirements)/H2O at 12:00 followed by an abdominal subcutaneous adipose aspiration at 15:00. Circulating hormones were measured with ELISAs and adipose gene expression was studied with microarray and qPCR analysis. This design tested the hypothesis that meal-induced signals interact acutely with GC to regulate gene expression.
Results: A physiological dose of cortisol and ingestion of a meal upregulated 216 and 235 genes and downregulated 45 and 24 genes respectively (FDRq< 0.05 and change >30%). Both known and novel GC/insulin targets were identified. Gene set enrichment analysis showed cortisol effects on the mTOR, IGF1 and EGF pathways (FDRq<0.1) and meal effects on TCA cycle and cholesterol/triglyceride biosynthesis (FDRq≤0.001). Two-way ANOVA identified 549 genes with significant cortisol /meal interaction (p<0.01).
Conclusion: GC and meal-induced signals coordinately regulate short-term gene expression in human adipose tissue.

# Board 7, Poster 25

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Blast Brain Injury results in Metabolic Dysfunction |
| **Authors** * | DJ Oberlin*, Georgina Perez-Garcia, Miguel A. Gama Sosa, Greg Elder, Christoph Buettner |

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**Structured Abstract** *

Background: Soldiers and law enforcement officers risk exposure to explosive blasts in the line of duty, where even non-contact injuries may affect their health. There is evidence that blast-related traumatic brain injury (TBI) leads to physical and mental health complications. Blast-related TBI has been associated with pituitary dysfunction, and post-traumatic stress disorder has been associated with a 40% increase in risk of developing diabetes. TBI caused by blast exposure in armed conflict has been studied in relation to several stress and psychiatric disorders; however systemic metabolic dysfunctions have not been researched.
Methods: Long-evens rats were anesthetized and placed in a compression-driven shock tube to simulate the effects of non-contact blast injuries. The animals had their body mass tracked during the course of the study. After they had recovered from the blast, their glucose metabolism was studied using glucose tolerance (GTT) and insulin tolerance tests (ITT). At the end of the study the animals were sacrificed, and had their fat pads removed and weighed.
Results: The animals had no differences in body mass ($p=0.41$) and no differences in any of the fat pad masses. Despite no differences in body mass or fat mass, blast animals had impaired glucose tolerance during a GTT compared to the control animals ($p=0.01$) using a repeated measures ANOVA. The blast animals also showed reduced insulin sensitivity compared to control animals during the ITT ($p=0.03$) using repeated measures ANOVA.
Conclusion: The blast exposure led to impaired glucose tolerance and insulin resistance despite similar body mass and fat masses. Blast-induced traumatic brain injury may lead to disruption in the brain's ability to regulate glucose metabolism, as the changes seem to be independent of changes in adiposity or body mass. The exact malfunctions are difficult to determine as blast injuries show varied distribution of injuries within the brain. This discovery requires further study to mitigate the risks of metabolic diseases to individuals exposed to blast injuries.

# Board 7, Poster 26

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Identification of novel gene targets of the heat shock factor HSF1 in adipose tissues |
| **Authors** * | Narendra Verma1, Yanjie Sun1 and Elisabetta Mueller1** |
| **Institutional Affiliations For Each Author.** * | 1Department of Medicine and Division of Endocrinology, Diabetes and Metabolism<br>NYU School of Medicine, New York, NY 10016, USA |
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**Structured Abstract** *

Background. Adipose tissues act as key determinants of whole body metabolism and energy homeostasis. The adipocytes present in white fat depots (WAT) mainly store lipids, while those present in brown adipose tissue play a role predominantly in expending energy through thermogenesis. Our laboratory is interested in unraveling the transcriptional nodes involved in the regulation of energy balance in thermogenic fat tissue. We have recently demonstrated that the heat shock factor 1 (HSF1), a conserved transcription factor involved in stress responses, regulates energy expenditure through activation of a PGC1α–dependent metabolic program in adipose tissues. Genetic modulation of HSF1 levels altered white fat remodeling and thermogenesis, and pharmacological activation of HSF1 via its activator celastrol was associated with increased levels of UCP1, enhanced energy expenditure, increased mitochondrial function in fat and muscle and protection against obesity, insulin resistance and hepatic steatosis during high-fat diet regimens (Ma et al., 2015). Although our published data point to a novel function of HSF1 in energy metabolism through the control of the levels of the metabolic coactivator PGC1α, the global gene expression programs set in motion by HSF1's activation in fat tissues have not been yet elucidated.
Methods. To identify novel targets of HSF1 in fat depots we have now taken several approaches, including candidate gene analysis involving in silico identification of putative heat shock responsive elements in key thermogenic genes and a systematic evaluation of genome-wide transcriptional targets of HSF1.
Results. Our preliminary data indicate the presence of a HSE element in the regulatory region upstream of the uncoupling protein UCP1. Chromatin immuno-precipitation studies performed in brown adipocytes have revealed the presence of HSF1 at the UCP1 promoter.
Conclusions. Our data demonstrate that UCP1 is a new target of HSF1 and reveal a novel mechanism of HSF1-mediated regulation of thermogenesis involving direct activation of uncoupling protein 1.

# Board 7, Poster 27

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Lipocalin-2 protects against obesity and insulin resistance |
| **Authors** * | Peristera I. Petropoulou*, Ioanna Mosialou, Steven Shikhel and Stavroula Kousteni |
| **Institutional Affiliations For Each Author.** * | Department of Physiology and Cellular Biophysics, Columbia University Medical Center, New York, NY |
| **Corresponding Author Email** * | sk2836@cumc.columbia.edu |

**Structured Abstract** *

Background: Regulation of food intake is a recently identified endocrine function of bone mediated by Lipocalin 2 (LCN2). Osteoblast-secreted LCN2 suppresses appetite, induces postprandial satiety and decreases fat mass while improving glucose tolerance, insulin secretion and sensitivity. Circulating LCN2 levels are inversely correlated with body weight and glycated hemoglobin in patients with type 2 diabetes. However, studies in humans have indicated a positive association of LCN2 with adiposity and insulin resistance.
Methods: To dissect this apparent discrepancy, we studied the connection between LCN2 and insulin resistance in mouse models of obesity. siRNA against LCN2 was delivered to Leprdb mice by subcutaneous injections using a polymer-based reagent every 2 days, for a total of 30 days. Body weight, appetite, fat pad mass, fasting glucose, fed- and glucose-stimulated insulin secretion were measured.
Results: We found that circulating LCN2 levels are 2-fold higher in 8 week-old obese, insulin resistant leptin-deficient (Lepob) and leptin receptor-deficient (Leprdb) mice and 25% higher in mice placed on a high fat diet for 12-16 weeks. The administration of siRNA against LCN2 decreased circulating LCN2 levels by 50%, thus normalizing them to those of wild type mice. This reduction in LCN2 serum levels, exacerbated the metabolic phenotype of Leprdb mice. Hyperphagia was increased by 24% and correlated with 50% increase in body weight gain and a corresponding 44% increase in gonadal fat pad weight. In addition, fasting blood glucose levels increased by 50%, and glucose-stimulated insulin secretion was substantially impaired. In contrast, as we have shown in previous studies, treatment of Leprdb mice with recombinant LCN2 suppressed food intake by 16.5% and improved glucose tolerance and insulin sensitivity to levels similar to those of control Leprdb/+ littermates. Body weight and gonadal fat were decreased and energy expenditure was increased in LCN2-treated Leprdb mice.
Conclusions: These results support a protective role for LCN2 in obesity and insulin resistance. An overproduction of inflammatory cytokines, excess of nutrients and possibly other factors may trigger LCN2 release as a compensatory response to combat the deleterious effects of obesity and insulin resistance.

# Board 7, Poster 28

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | 12-hydroxylated bile acids regulate gastric emptying through GPR119 in the intestine |
| **Authors** * | Sei Higuchi1*, Tiara Ahmad1, Enrico Bertaggia1, Kristian K. Jensen2, Liangsu Wang2, Rebecca Haeusler1 |
| **Institutional Affiliations For Each Author.** * | 1. Naomi Berrie Diabetes Center and Department of Pathology and Cell Biology, Columbia University<br>2. Merck Research Laboratory |
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**Structured Abstract** *

Background: One important risk factor of obesity is considered to be excessive food intake. Feeding behavior is governed partly by lipid mediators in the gastrointestinal tract. Particularly, intestinal lipid plays an important role for regulation of satiation through gastric emptying. Bile acids (BAs) are synthesized from cholesterol in the liver and modulate intestinal lipid metabolism. Previously, we reported that certain subset of BAs- the 12-hydroxylated BAs (12-OH BAs) - are positively correlated with BMI in humans. Furthermore, our recent studies show that deficiency of the essential enzyme to produce 12-OH BAs, Cyp8b1, reduces body weight and impairs lipid absorption in mice. Our data show that Cyp8b1 deficiency reduces ab lib food intake. From this evidence, we hypothesized that Cyp8b1 deficiency reduces gastric emptying through lipid metabolism in the intestine.
Methods and Results: To determine the direct effects of 12-OH BAs on gastric emptying, we used Cyp8b1 null mice. We evaluated gastric emptying by two methods. In the first method, mice were fasted for 16h and allowed access to chow for 5h. We measured food intake and food content in stomach to determine the gastric emptying. In the second method, gastric emptying was assessed by acetaminophen absorption test. Acetaminophen absorption is rate-limited by gastric emptying, thus it is used to evaluate gastric emptying. Both tests showed that Cyp8b1 deficiency decreased gastric emptying. To test the effect of intestinal lipid on this phenotype, we gave mice a fat-free diet. On this diet, gastric emptying was not different between wild-type and Cyp8b1-deficient mice. However, stimulation with a single fat-rich meal decreased gastric emptying in Cyp8b1-deficient mice. Our previous report showed that Cyp8b1 knockouts excrete threefold more monoacylglycerol in mice. Luminal monoacylglycerol activates the G protein-coupled receptor GPR119 in the intestine, which induces slow gastric emptying. Antagonization or knockout of GPR119 normalized the slow gastric emptying of Cyp8b1 deficient mice. These data suggest that 12-OH BAs regulate gastric emptying by modulation of dietary lipid absorption and GPR119 activation in the intestine.
Conclusions: Suppression of Cyp8b1 may be a novel target to reduce gastric emptying and increase satiation for obesity and diabetes treatment.

# Board 8, Poster 29

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Role of Telomere in Endothelial Dysfunction in Type 2 Diabetic Mice |
| **Authors** * | Yuanfeng Gao 1 *, Junxi Wu 2, Jian Cui 1, Junjie Bai 3, Cuihua Zhang 2, Michael A Hill 2, Hanrui Zhang 1 |
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**Structured Abstract** *

Background: Telomere shortening is associated with age-related disorders. Leukocyte telomere length was shorter in patients with type 2 diabetes (T2D), but the role of telomere function in the pathogenesis of vascular complications and vascular aging in T2D remains incompletely defined.
Methods: We hypothesized that vascular telomere malfunction accelerates vascular aging and endothelial dysfunction. To test this hypothesis, we examineed telomerase activity, telomere length, vascular apoptosis and endothelium-dependent vasorelaxation in the aortas of control mice (m Leprdb) and T2D mice (Leprdb) at 3 and 10 months of age (n=6 mice).
Results: Telomere length was shorter in the aortas of 3-months and 10-months Leprdb vs. 3-months control mice, but comparable between 3-months and 10-months control mice. The apoptotic cell numbers, caspase 3/7 activity and the expression of Chk2 were elevated in the aortas of 3-months and 10-months Leprdb, but not of 10-months control mice. Aortic acetylcholine-induced endothelium-dependent vasorelaxation was impaired in 3-months and 10-months Leprdb mice, but the 10-months control mice showed comparable vasodilatory function compared with 3-months control mice. The aortic endothelial function, telomerase activity and telomere length showed no further impairment in 10-months Leprdb compared with 3-months Leprdb mice, suggesting an early vascular aging in the T2D mice. In cultured mouse coronary artery endothelial cells, high glucose treatment (30 mmol/L) for 48 hours significantly reduced the mRNA expression of telomerase reverse transcriptase (Tert), supporting the potential role of hyperglycemia in impairing telomere elongation. Thus, telomere shortening, a marker of telomere malfunction, is associated with increased vascular apoptosis and impaired endothelial function in T2D mice.
Conclusions: Telomere shortening plays an important role in diabetes-associated early vascular aging and endothelial dysfunction by promoting apoptosis of T2D vasculature.

# Board 8, Poster 30

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Adiporedoxin protects adipocytes from oxidative stress |
| **Authors** * | Alessandro Peschechera*, Collette J Laflamme, Mi-Jeong Lee, Varuna Shibad, Kalypso Karastergiou, Paul F Pilch, and Susan K Fried |
| **Institutional Affiliations For Each Author.** * | Diabetes, Obesity and Metabolism Institute, Icahn School of Medicine at Mount Sinai, New York, NY<br>Department of Biochemistry and Department of Medicine, Boston University School of Medicine, Boston, MA |
| **Corresponding Author Email** * | susan.fried@mssm.edu |

**Structured Abstract** *

Background: Adiporedoxin is an ER-resident protein highly expressed in adipocytes and regulates the secretion of disulfide-bonded proteins including adiponectin. The primary sequence of Adrx contains a -CXXC motif, indicating it belongs to the thioredoxin/peroxiredoxin family of redox active proteins, suggesting it functions as a redox sensor and thereby contributes to the altered adipokine and secretory function in response to the chronic inflammatory and metabolic stress of obesity.
Methods: Studies were conducted in cultured 3T3-L1 mouse adipocytes and primary cultures of human adipocytes.
Results: To test the hypothesis that Adrx protects adipocytes from oxidant stress, we subjected differentiated 3T3-L1 adipocytes to hypoxia (up to 8 hours) and menadione. We verified that hypoxia and menadione enhanced the production of reactive oxygen species (ROS) and inhibited adiponectin secretion. Overexpression of Adrx attenuated ROS production and prevented the inhibition of adiponectin secretion. Furthermore, Adrx overexpression reverted the hypoxia-mediated regulation of several ER stress markers. We also examined the physiological role of Adrx in newly-differentiated human adipocytes. Preliminary siRNA Knockdown of Adrx in human adipocytes induced the carbonylation of total proteins, a hallmark of the oxidation status of the proteins. Because we previously found that Adrx expression in human adipocytes was inversely related to pJNK/totalJNK ratio, a marker of inflammation and downstream target of TNF, we tested the effect of TNF on Adrx protein levels. Consistent with the hypothesis that Adrx can protect cells from oxidant stress, treatment of cultured human adipocytes with low concentrations of TNF (1-3 ng/ml) for 3 h increased Adrx protein levels by 1.5-fold ($p<0.0001$, $n=5$), but higher doses tended to decrease its expression. In contrast, incubation of human adipocytes for with all tested TNF concentrations (1-ng/ml) for 24 h tended to reduce Adrx protein levels.
Conclusions: Current results are consistent with the hypothesis that Adrx serves an important antioxidant protein in the adipocyte ER and that its downregulation in obesity may contribute to ER stress and high cellular ROS that leads to altered secretory and cellular dysfunction in obesity.

# Board 8, Poster 31

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Role for Allograft inflammatory factor-1-like (AIF1L) in diet-induced obesity |
| **Authors** * | Dippal Parikh*, Prameladevi Chinnasamy, Dario F. Riascos-Bernal, Smitha Jayakumar, Nicholas E. Sibinga |
| **Institutional Affiliations For Each Author.** * | Wilf Family Cardiovascular Research Institute, Department of Medicine (Cardiology), and Department of Developmental and Molecular Biology, Albert Einstein College of Medicine, Bronx, NY |
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**Structured Abstract** *

Background: Adipose tissue expansion is a hallmark feature of obesity which is characterized by hyperplasia and hypertrophy of adipocytes. This in turn leads to adipose tissue inflammation and ultimately to systemic insulin resistance. Allograft inflammatory factor-1 (AIF1) is a pro-inflammatory molecule with roles in stress-induced survival, phagocytosis, and migration of macrophages. We have found that inactivation of AIF1 in mice prevents high fat diet (HFD)-induced weight gain and preserves insulin sensitivity. Interestingly, a paralog of AIF1 called AIF1-like (AIF1L) was identified by the human genome project. AIF1L has amino acid sequence identity of 63% and similarity of 80% when compared to AIF1. AIF1L has not been well studied and very little is known about its physiological function. Based on our findings with AIF1 and the known homology of these proteins, we hypothesize that AIF1L may play a functional role in diet-induced obesity (DIO).

Results: 8 weeks old Aif1L-/- mice (KO) and wild-type littermates (WT) were subjected to HFD for 16-18 weeks. Strikingly, we observed that KO mice weigh significantly more than the WT mice and that these differences can be attributed to an increase in adipose tissue mass, while lean mass in mice of both sexes is preserved. In females, both subcutaneous (inguinal) and visceral (perigonadal) fat depots increased in mass. In males, on the other hand, increased adiposity was accompanied by larger subcutaneous fat depots and a fatty-appearing liver, while visceral fat depots were not different. Brown adipose tissue (BAT) depots from KO male mice appeared 'whiter' in color and twice as large as those of WT mice, suggesting increased lipid accumulation in BAT as well; this difference was not seen in females. These findings suggest a differential and sex-dependent role for AIF1L in the different adipose depots. Also, preliminary studies of mouse metabolic profiles after 18 weeks of HFD and with clear differences in adipose burden show decreased physical activity in KO mice, without differences in energy expenditure. Food intake, activity, and metabolic profile measurements prior to and early in HFD feeding will give us more insight into the source of the phenotype.

Conclusions: These findings suggest that AIF1L expression is protective, in that it limits DIO—a direct contrast to our findings with AIF1, which is required for DIO. Considering the multi-factorial nature of obesity pathogenesis, uncovering novel regulators such as AIF1 and AIF1L will provide further insights into adipocyte biology and its interaction with other cell types.

# Board 8, Poster 32

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Novel role of the immune checkpoint B7-H3 (CD276) in metabolic homeostasis and obesity |
| **Authors** * | Elodie Picarda*, Haihong Zong, Jeffrey Pessin, Xingxing Zang |
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| **Corresponding Author Email** * | xingxing.zang@einstein.yu.edu |

**Structured Abstract** *

Background: Member of the immune checkpoints B7 family, B7-H3, is a transmembrane protein with limited expression at steady state but dramatic overexpression in human cancers. It exerts multiples functions, from the regulation of innate and adaptive immunity to the control of bone formation and cancer cell metabolism. Interestingly, we observed that mice knock-out for B7-H3 gained significantly more weight than their WT counterparts, being 165% heavier after 4 months fed a chow diet with 20% calories from fat. We thus studied the metabolic and adipose immune phenotypes of these KO obese mice compared to lean WT.
Methods: All experiments were done around 16 weeks on female mice. To assess the fat versus lean mass proportion, we performed MRI. For the study of mouse energy metabolism, mice were housed in a calorimeter and tested for 02/CO2 consumption, energy expenditure (EE), physical activity and food intake. Insulin and glucose sensitivity were assessed by ITT and GTT. Histologic analysis and qPCR were conducted on white, brown and beige fats to evaluate morphologic and functional differences. Lastly, after dissociation of gonadal fat pads, the stromal vascular fraction was stained for immune and progenitor cells and analyzed by flow cytometry.
Results: B7-H3-/- mice displayed an increased fat and total mass compared to WT. Their respiratory exchange ratio was higher suggesting an increase in glucose utilization. However, EE, activity and food intake were similar, i.e no hyperphagic behavior. Although glucose sensitivity was normal, fasting glucose was significantly elevated in the KO group, together with a lower insulin sensitivity. Moreover, white fat pads from the KO mice were significantly bigger than WT and contained hypertrophic adipocytes. Brown fat showed signs of 'whitening'. Leptin gene expression was increased in the KO group but no difference was seen for multiple classical beige (PAT2) and brown (Ucp1) markers, suggesting that obesity was independent of metabolic function of beige and brown fats. Furthermore, we found evidence of chronic inflammation in the KO gonadal fat, in particular through the recruitment of mast cells, proinflammatory M1 macrophages and T cells. Very interestingly, we also detected a high expression (>95%) of B7-H3 on the WT adipocyte progenitors (CD45-CD31-CD29+Sca1+PDGFR1a+), raising the question of a hypothetical role of B7-H3 in adipogenesis.
Conclusion: Our study will help to unravel the exciting novel role and mechanism of immune checkpoints in the control of metabolism and inflammation, and could lead to potential new immunotherapies for obesity and related diseases.

# Board 9, Poster 33

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Addictive eating is linked with depressive symptoms and left asymmetry in adults with overweight and obesity |
| **Authors** * | Aviram-Friedman, Roni*<br>Zibman, Sam<br>Kinreich, Itamar<br>Zangen, Abraham |
| **Institutional Affiliations For Each Author.** * | Department of Life Sciences and the Zlotowski Center for Neuroscience, Ben-Gurion University of the Negev, Israel |
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**Structured Abstract** *

Introduction: Obesity has co-morbidities, among which is depression and addictive eating. However, the association between these two co-morbidities have not yet been studied. Further, left prefrontal brain asymmetry [(greater activation in the left- relative to the right prefrontal cortex (PFC)]) in response to food has been linked with approach motivation and cue reactivity in overeaters, while right asymmetry with inhibitory control, but the link between prefrontal asymmetry and addictive eating has not yet been explored. Purpose: To study the association between addictive eating and depression, as well as between addictive eating and prefrontal asymmetry, in a group of overweight and obese adults. Methods: We recruited 15 obese and overweight adults (BMI 27-42; M:F 3:12) , who were weighed following an overnight fast and prior to the consumption of a standardized breakfast. We administered the Yale Food Addiction Scale (YFAS), which provides two scores: a symptom count (1-7; YFAS-S), and a clinically significant impairment score (1 or 0 for its presence or absence, respectively; YFAS-D). We also administered the Beck Depression Inventory (BDI) to measure depressive symptoms, and we recorded participants' brain activity with electroencephalography (EEG) during a 5-min resting period. We calculated the alpha power, which is negatively correlated with localized brain activity. Results: Among the 15 participants, depression score average was 8.8, with a range of 0 to 24 (i.e., no- to moderate depression). YFAS-S score average was 4.4, with a range of 1 to 7, and YFAS-D was present in 8 participants. Scores on the BDI correlated with both YFAS-S and YFAS-D ($r2 = 0.57$, $p = 0.009$, and $r2 = 0.51$, $p = 0.02$, respectively). This model remained significant after controlling for BMI ($r2 = 0.52$, $p = .03$ for YFAS-S, and $r2 = .59$, $p = .02$ for YFAS-D). Partial correlation analysis between prefrontal alpha power and YFAS-D, controlling for the contralateral prefrontal electrode, yielded a positive correlation on the right PFC (F6; $r = 0.67$, $p = 0.03$) and a negative correlation on the left PFC (F5; $r = -0.59$, $P = 0.01$). Discussion: Addictive eating is positively linked with depressive symptoms and left asymmetry in overweight and obese adults. Further studies with larger sample size, to examine the nature of this link, are needed. This information may help guide our understanding of obesity phenotypes and help tailor future therapeutic interventions.

# Board 9, Poster 34

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Sex and menstrual cycle modulate the prefrontal cortex response during eating as measured by fNIRS |
| **Authors** * | JA NASSER*, E ALBAJRI, L LANZA, A DEL PARIGI, H AYAZ |
| **Institutional Affiliations For Each Author.** * | Drexel University, Philadelphia, PA, United States |
| **Corresponding Author Email** * | jan57@drexel.edu |

**Structured Abstract** *

Background: PET imaging post-ingestion suggests that women show increased lateral prefrontal cortex (PFC) activity while men show increased medial PFC activity. Unlike PET or fMRI, functional infrared spectroscopy (fNIRS) allows for assessing PFC response during ingestion. Using functional infrared spectroscopy (fNIRS), we examined the interaction of sex and menstrual cycle on prefrontal cortex (PFC) response during eating of participant-designated preferred (PF) and non-preferred (NP) food. We hypothesized 1) that greater medial PFC vs lateral PFC activity would result in greater intake of PF (grams), and 2) that PFC response to PF, in women, would resemble that in men during the follicular phase of the menstrual cycle.
Method: 79 men and women, (30 men, 36 women follicular phase, 13 women
luteal phase) matched for age and BMI (26 ± 8 years, BMI 24 ± 4) completed two fNIRS sessions consuming PF or NP on separate days.
Results: For NP, intake was greater if mPFC activity was > lPFC activity (167 ± 110, 119± 70, mPFC vs lPFC respectively, F = 5.1, p = 0.027, n = 79,) in all participants. For PF, intake in men was greater when mPFC was > lPFC (247 ± 120, 174± 70, mPFC vs lPFC respectively, F = 4.2, p = 0.05, n = 30). During the follicular phase, the relationsip of PFC activity and intake trended towards significance (115 ± 36, 153 ± 61, mPFC vs lPFC respectively, F = 3.4, p = 0.076, n = 36) but was opposite to that observed in men. No significant difference in PF intake between mPFC > lPFC was observed in the luteal phase (137 ± 88 vs 141± 52, respectively, F = 0.012, p = .92, n = 13).
Conclusions: These data suggest 1) that the relative activity of PFC regions may affect food intake, and 2) menstrual cycle phase appears to affect PFC response during eating PF.
Supported By: Drexel University Clinical Translational Research Institute, Office of the Provost and Dept of Nutrition Sciences

# Board 9, Poster 35

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Functional Magnetic Resonance Imaging (fMRI) in Participants with and without Binge Eating Disorder (BED) Pre and Post Bariatric Surgery |
| **Authors** * | Allan Geliebter, Lauren Puma, Nerys Astbury, Shaunte Baboumian*, Spiro Pantazatos, Charles Swencionis |
| **Institutional Affiliations For Each Author.** * | Mount Sinai Icahn School of Medicine Dept of Psychiatry, Touro College and University System, Nuffield Dept of Primary Care Health Sciences University of Oxford, Mount Sinai Icahn School of Medicine, Dept of Psychiatry, Molecular Imaging and Neuropathology Division New York State Psychiatric Institute Department of Psychiatry Columbia University, Department of Psychiatry and Behavioral Sciences, Albert Einstein College of Medicine |
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**Structured Abstract** *

Background: To examine differences in brain activation by functional magnetic resonance imaging (fMRI) between obese binge eaters (BE) and non-binge eaters (NB), who underwent bariatric surgery vs. no treatment.
Method: In a prospective cohort study: 15 bariatric surgery (S: 8 BE, 7 NB) and 13 non-treatment (NT: 7 BE, 6 NB) obese females completed fMRI brain scans before and 4-months after surgery, viewing high (HED) and low energy-dense (LED) food (F) and non-food (nF) visual stimuli. BE and NB females were similar in age, body mass index (BMI), and % body fat.
Results: Postsurgery, the BEs no longer engaged in binge eating. At baseline and postsurgery in response to F > nF, BE (vs. NB) showed greater precuneus activation, and greater thalamic activation postsurgery, all p's < .01. Postsurgery, BE vs. NT had less middle occipital gyrus ($p < .001$) and inferior frontal gyrus activation ($p < .004$) to high energy dense (ED) vs. low ED. Overall S (vs. NT) had a greater reduction in dorsomedial prefrontal cortex (dmPFC) activation to HED and showed an increase in activation to LED ($p < .006$). There was no significant difference in weight loss with binge status. An ROI analysis at 4 months revealed significantly greater thalamus activation in Binge Eaters compared to Non-Binge Eaters in response to F vs nF cues at 4-months post ($p < .014$).
Conclusions: Overall postintervention (S vs NT) had reduced dmPFC activation in response to HED and increased activation to LED. From baseline to postsurgery, BE vs. NB showed reduced neural responsivity in middle occipital gyrus and inferior frontal gyrus activation to HED vs. LED. Postsurgical BEs appear to pay less attention to HED vs. LED food cues and require less inhibition to control their eating behavior. Findings provide evidence consistent with a distinct neural binge eating phenotype, which seems to persist postsurgery. RYGB BEs experienced significant weight loss similar to NBs. Neural activation suggest that F vs nF stimuli continues to be more salient and relevant for binge eaters, which supports a binge eating phenotype, whereas HED vs. LED food cues continue to be equally salient and relevant for Binge Eaters and Non-Binge Eaters. Findings suggest that the neural binge eating phenotype may persist beyond surgery, even after losing BE status per DSM-5 criteria.

# Board 9, Poster 36

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Identification of neural population in the whole brain that project to both pancreas and adrenal gland |
| **Authors** * | Xin-an Liu, Yan shi, Maria Gonzalez, Zuxin Chen, Paul Kenny |
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**Structured Abstract** *

It is now know that central nervous system (CNS) plays an important role in the regulation of glucose homeostasis. Accumulating evidences show that dysregulated CNS circuits causes abnormal glucoregulatory mechanisms. That is because the peripheral organs (pancreas, adrenal gland, liver etc) involved in glucose regulation are tightly controlled by the CNS through direct and indirect descending neural innervations. Thus, understanding of the connections between the CNS and these peripheral organs will provide us a new window to improve glucose regulation. Here, by using retrograde neuronal tracer pseudorabies virus, we identify the populations of neuron in the whole brain that project to pancreas, adrenal gland and both. The common area innervating both pancreas and adrenal gland include PVH, NTS, DMV, AP, Amydala etc. A detail of these areas will be showed in our poster. We also further characterized the common neuronal population with specific markers.

# Board 10, Poster 37

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | The relationship of birth weight and adiposity across the life course to semen quality in middle age: results from a follow-up to the Child Health and Development Studies |
| **Authors** * | Linda G. Kahn*, Elizabeth M. Widen, Nadine Straka, Xinhua Liu, Piera M. Cirillo, Barbara A. Cohn, Germaine M. Buck Louis, Pam Factor-Litvak, |

**Institutional Affiliations For Each Author.** *

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**Structured Abstract** *

Background: Adiposity has been identified as a potentially modifiable risk factor for poor semen quality, yet prior studies of body mass index (BMI) and semen quality have been inconclusive, with most finding no linear association between current BMI and any of the three primary semen quality measures: sperm concentration, percent motile sperm, and percent sperm with normal morphology. Using longitudinal data from the Study of the Environment and Reproduction follow-up to the Child Health and Development Studies birth cohort, we examined independent and cumulative relationships of birth weight for gestational age and of adiposity measures throughout early childhood and adulthood with semen quality in middle age

Methods: One hundred ninety-three non-azoospermatic participants (mean age 44 years) who provided semen samples were included in our analysis. In addition to birth weight for gestational age percentile, we created age-specific adiposity measures for three time points in childhood and calculated BMI for three periods in adulthood. In multivariate regression models, we then tested whether each individually and cumulatively predicted sperm concentration, percent progressive motility, percent normal morphology, as well as parameter-specific and combined measures of subfertility based on 2010 World Health Organization reference levels.

Results: For every percentile increase in birth weight for gestational age, square-root sperm concentration increased by 0.02x103/mL in covariate-adjusted analyses (95% confidence interval (CI) [0.002, 0.04]). We found increasingly negative associations between child overweight from 4 months to 4 years and sperm concentration. Adiposity in participants' twenties and thirties, but not childhood, was associated with low percent progressive motility (OR20s=1.11, 95% CI [1.00, 1.22]; OR30s=1.08, 95% CI [1.00, 1.17]), as were cumulative adiposity measures across 3 adult time periods. We observed a similar trend for low percent normal morphology. Adiposity in adulthood was also associated with higher odds of meeting WHO subfertility criteria for at least 2 of the 3 semen outcomes.

Conclusion: Our novel findings suggest that the relationship between adiposity and sperm concentration, motility, and morphology may vary according to timing and duration of excessive adiposity. Additional life course studies of adiposity and semen quality are warranted to confirm our results and expand them to include the important developmental periods of puberty and adolescence, as well as indicators of potential biological mechanisms.

# Board 10, Poster 38

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Using SMART Goals to Help Teens Modify Their Health Behaviors |
| **Authors** * | Sarah N. Martin, MPH*; Camille C. Jimenez MPH, Irene Orejudos, MPH; Julia Cox; Jean Lim; Judith Wylie-Rosett EdD RD |
| **Institutional Affiliations For Each Author.** * | Albert Einstein College of Medicine, Albert Einstein College of Medicine, New York University, New York University, HealthCorps, Albert Einstein College of Medicine |
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**Structured Abstract** *

Background: S-M-A-R-T (Specific, Measurable, Action-oriented, Realistic, Time-bound) goal setting is used to increase task performance in education and business. However, little is known about its use related to teaching how to modify health behaviors.

Methods: We used participatory action methods to develop and evaluate SMART Goal Setting modules in collaboration with our partner, HealthCorps, and with student input. The modules guide students in developing a lifestyle-related SMART Goal using personalized feedback from a lifestyle questionnaire related to fruits/vegetables, breakfast, physical activity, junk/fast food, sugary beverages and sedentary behavior. The students received a personalized "Healthy Me Snapshot Report" which summarized the student's health behaviors highlighting areas of strength as well as suggested areas for improvement. In the subsequent module, students completed a worksheet to develop a personal SMART Goal to work on over the next several weeks. Students were encouraged to consider their Healthy Me Snapshot Feedback Report when developing their SMART Goals. The research team scored the students' goals on the basis of the SMART criteria, assigning 1 point for meeting each of the 5 criteria (Specific, Measurable, Action-oriented, Realistic, Time-Bound) with a maximum of 5 points. Two raters scored each worksheet and kappa scores were calculated to measure inter-rater reliability. Kappa scores were deemed acceptable, ranging from .6 (Action-oriented) to .9 (Time-bound).

Results: During the Fall 2016 semester, 640 students (60% female, 39% male) from 9 HealthCorps high-schools completed the SMART Goal modules. School participation size ranged from 27 to 121 students. Students frequently chose goals related to increasing frequency of breakfast (24%) and physical activity (22%) and decreasing sugary beverages (16%) and junk/fast food (17%). A mean score of 3.6 (SD 1.2) out of 5 possible points was obtained across all students, with significant differences found between schools (mean range 2.3 to 4.6, P<.0001). Female students scored higher (mean score 3.7, SD 1.2) than male students (mean score 3.4, SD 1.2). Goals related to increasing breakfast (mean score 4.1, SD .96) scored significantly higher (P<.0001) than goals related to the other categories including Sedentary Behavior (3.0, SD 1.3). Mean scores ± SDs were Specific (.86±.35), Action-oriented (.85±.36), Measurable (.53±.5), and Time-bound (.40±.49).

Conclusion: After participating in this module, students were able to develop heath-related SMART Goals. Particular components (i.e. Measurable, Time Bound), as well as particular categories (e.g. Junk/Fast Food Intake) were more challenging for students to develop. Teachers should take extra time when discussing these topics.

# Board 10, Poster 39

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Evaluating the Influence of Racially Targeted Food and Beverage Advertisements on Black and White Adolescents' Perceptions and Preferences |
| **Authors** * | Marie A. Bragg, PhD*, Alysa N. Miller, MPH, Yrvane K. Pageot, Tenay Greene, Brian D. Elbel, PhD, MPH, Christina A. Roberto, PhD |

**Institutional Affiliations For Each Author.** *

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**Structured Abstract** *

Background: Adolescent obesity rates continue to rise, and exposure to food marketing is linked with poor dietary habits among youth. Black adolescents are disproportionately affected by diet-related health conditions and face higher levels of exposure to unhealthy food ads compared to White adolescents.
Methods: 1,503 Black (N=734) and White (N=769) 12-17 year old adolescent participants were recruited through Survey Sampling International's online panel to complete an online survey in which they were randomized to view either four food and beverage ads featuring Black actors or four food and beverage ads featuring White actors. After viewing each commercial, participants completed the survey.
Results: The primary outcomes were self-reported attitudes toward the ad and brand, purchase intentions, and willingness to engage with the brand on social media. In this sample, Black participants were more likely to report a positive affective response toward ads that targeted them compared to Whites. However, White participants were more likely to like ads that were not targeted toward them compared to Black participants. The groups did not differ in ratings of perceived tastiness, brand liking, willingness to engage with the brands on social media, and purchase and consumption intentions.
Conclusions: Both Black and White adolescents report more positive affective responses to Black-targeted ads compared to White-targeted ads. Future research should examine the influence of racially-targeted marketing in real-world contexts like social media, as well as the influence of longitudinal exposure to targeted advertising on behavior.

# Board 10, Poster 40

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Adiposity measures and type 2 diabetes in the UK Biobank |
| **Authors** * | Abhishek Vishnu,1 Naveed Sattar,2 Ruth J F Loos1,3,4 |
| **Institutional Affiliations For Each Author.** * | 1 The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA; The Genetics of Obesity and Metabolic Traits Program, Icahn School of Medicine at Mount Sinai, New York, NY, USA. 2 Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, UK. 3 The Mindich Child Health and Development Institute, Icahn School of Medicine at Mount Sinai, New York, NY, USA. 4 Department of Environmental Medicine and Public Health, Icahn School of Medicine at Mount Sinai, New York, NY, USA |
| **Corresponding Author Email** * | ruth.loos@mssm.edu |

**Structured Abstract** *

Aim: Use of more than one adiposity marker may improve risk classification for type 2 diabetes (T2D), but it is unclear how much additional risk is explained by these markers especially among different sex and ancestral groups. We aimed to compare the association between three adiposity traits i.e. body-mass index (BMI), waist-hip ratio (WHR) and body fat% (BF%), and the presence of T2D among major ancestral groups in the UK Biobank.
Methods: Normal/overweight/obese categories were determined as follows - body-mass index (BMI; <25, 25 to <30 and ≥30 kg/m2; cut-offs for South Asians: 23 and 27.5 kg/m2) and sex- and ancestry-specific tertiles of waist-hip ratio (WHR) and body-fat percentage (BF%). Trait values were standardized using inverse normal transformation to allow comparison of effect estimates. T2D was self-reported and confirmed with the use of medications and presence of hospital ICD-10 codes. We tested the association between each adiposity trait, as well as their combinations, with T2D using logistic regression analyses. Analyses were performed by sex (nwomen= 260,983, nmen = 217,481), and ancestry (n Europeans =462,998, nAfro-Caribbean = 7,744 and nS. Asians = 7,722). Hierarchical models were compared with likelihood-ratio tests for improvement in model performance.
Results: The prevalence of T2D varies across ancestries (P < 2.2E-16), with higher prevalence among S. Asians (14.6%) than among Afro-Caribbean (8.6%) and Europeans (3.8%). Overall, WHR had the strongest association with T2D among the three traits, and BF% the weakest. Receiver operating curve analyses showed that the area under curve (AUC) was highest for WHR (0.74), followed by BMI (0.73) and BF% (0.72) among all sex-ancestry groups, except among Afro-Caribbean and S. Asian men (WHR > BF% > BMI). Further, addition of BF% in the model for T2D containing BMI and WHR improved its performance only slightly - P>0.05 among S. Asian (δAUC<3E-3) and European (δAUC=62E-5) women, 0.01< P <0.05 among Afro-Caribbean men (δAUC=82E-4) and women (δAUC=75E-4), and P < 5E-4 among S. Asian (δAUC=12E-3) and European (δAUC=6E-3) men. Addition of BMI to model containing WHR and BF% improved model performance among S. Asian women (δAUC=5E-3), and European men (δAUC=12E-3) and women (δAUC=20E-3). In contrast, addition of WHR in the regression model T2D containing the BMI and BF% significantly improved performance in all groups (all P <5E-20, mean δAUC=54E-3).
Conclusion: While WHR overall has the strongest association with T2D of all traits, sex, ancestry specific groups differ in how other traits are related with DM.

# Board 11, Poster 41

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Portion size and frequency of sugary drink consumption among New York City adults, 2013–14 |
| **Authors** * | Tamar Adjoian*, Divya Prasad, Amaka Anekwe |
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**Structured Abstract** *

Background: Restaurants and retailers often employ value-based pricing; larger portions are more economical, and single-serving beverages can exceed 50 ounces. Consumption of sugary drinks is associated with weight gain and increased chronic disease risk, including type 2 diabetes and heart disease. This analysis examines frequency and portion size of sugary drink consumption among New York City adult residents in 2013–14.
Methods: The New York City Community Health Survey is a cross-sectional population-based representative annual survey of non-institutionalized NYC residents aged 18 years or older. In 2013 and 2014, respondents were asked about their sugary drink consumption frequency and typical portion size; these years of data were combined for this analysis. Participants were asked how often they drink sugar-sweetened soda and other sweetened drinks (e.g., sweetened iced tea, sports drinks, or fruit-flavored drinks). Response options were open ended and combined to derive values for total sugary drinks/day. Respondents were also asked what size soda and other sweetened drinks they usually drink. Categorical response options were: <12, 12, 16, 20, 32, and >32 ounces. To quantify the two extreme size options, <12 ounces was recoded to 6 ounces, and >32 ounces was recoded to 33 ounces. Values for soda and other sugary drinks/day were multiplied by usual drink size to determine total ounces/day. For combined values of soda + other sugary drinks, an average of typical drink size was applied. Calories/ounce were sourced from a report including comprehensive nutritional analyses of sugary drinks.
Results: Overall, respondents typically consumed sugary drinks that were 12 ounces or smaller (77%). However, those reporting that they consumed more than two sugary drinks daily were more likely to drink 16 ounces or larger as their usual size (7% vs. 14%, p<.001). Sugary drink consumers took in 14 ounces/day on average; ounces consumed increased significantly with each one unit increase in sugary drinks/day. Those drinking more than 2 sugary drinks/day took in 53 ounces on average. High-calorie sugary drinks, including fruit drinks and energy drinks, can have about 17 calories/ounce; therefore, 53 ounces of sugary drinks could amount to over 900 calories of poor nutritional value.
Conclusions: Public health practitioners aiming to reduce negative health consequences of sugary drinks should examine frequency and portion size of sugary drink consumption, together with price, and use this information in designing interventions. Clinicians should gather information on patients' frequency and portion size of sugary drinks when designing tailored dietary recommendations.

# Board 11, Poster 42

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | The effects of parental education on breakfast consumption among 7th and 8th grade students. |
| **Authors** * | Jamil M Lane, MPH, PhD Student*, Silvia Sorensen, PhD |
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**Structured Abstract** *

Background: Eating breakfast has been identified as an important determinant in healthy nutrition, especially during developmental stages. Early adolescence is a vital period that requires a salubrious diet as it helps to optimize psychological and physical well-being. Socioeconomic status (SES) variables such as parents' education levels are significant predictors of adolescents' health behaviors such as dietary habits and health problems including obesity.
Objective: The primary aim of this study is to examine adolescents' breakfast habits, food choices for breakfast, and breakfast skipping frequency among 7th and 8th-grade students according to the effects of parental education levels.
Design/Methods: Analyses were based on data from Wave I of the National Longitudinal Study of Adolescent Health, a nationally representative sample of all adolescents in the United States who were surveyed in 1995. The level of parental education was examined to understand its impact on 7th grade (n = 979) 15.1% and 8th grade (n = 992) 15.3 % breakfast consumption activity. Binary logistic regression analysis was used to determine if parental education level is a predictor of adequate or inadequate breakfast habits in junior high school adolescents.
Preliminary Results: We hypotheses that parents with education levels less than secondary schooling will, in fact, have adolescents that will bear poorer breakfast eating habits and higher frequencies of breakfast skipping when compared to young teens with parents with education levels beyond the 8th grade. We also anticipate highly educated parents (e.g., > four-year college education) will have a positive effect on adolescents' daily breakfast eating habits with adequate consumption of healthier foods and lower frequencies of breakfast skipping.
Conclusion: One way to improve poor breakfast habits and the obesity crisis among middle school aged adolescents is to initiate health educated campaigns and prevention programs for families. The goal is for these initiatives to serves as tools to apply such interventions of health promotion to disseminate information to help parents establish healthy eating habits for families early in life and to maintain these behaviors throughout their lives.

# Board 11, Poster 43

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Association between large for gestational age infants and excessive pregnancy weight gain by pre-pregnancy body mass index |
| **Authors** * | Mary Huynh*, Ying Sun, Gretchen Van Wye |
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**Structured Abstract** *

Background: Large for gestational age (LGA) infants have been found to be at greater risk for obesity later in life. Maternal weight gain during pregnancy has been linked to LGA. However, much of the literature on LGA has focused on the increased risk for mothers who are overweight or obese prior to pregnancy and their weight gain during pregnancy as compared to normal weight and underweight mothers. This analysis aims to examine gestational weight gain and LGA by maternal pre-pregnancy BMI category.

Methods: This cross-sectional analysis utilized a cohort of singleton births in New York City, 2008-2014 (n=804,580). Pre-pregnancy BMI was categorized into underweight (BMI < 18.5), normal weight (18.5-24.9), overweight (25-29.9), and obese (30+). Gestational weight gain was calculated as maternal pre-pregnancy weight subtracted from weight at delivery. Excessive gestational weight gain was classified using the Institute of Medicine 2009 guidelines for weight gain during a singleton pregnancy. LGA was defined as an infant with a birthweight above the 90th percentile for gestational age. Covariates in the logistic regression model included race/ethnicity, maternal age, parity, maternal education, Medicaid status, and presence of pre-pregnancy hypertension and/or diabetes or gestational hypertension and/or diabetes.

Results: In this cohort, 5.6% of the mothers were underweight prior to pregnancy, 23.7% overweight, and 16.4% obese. Almost 40% of the mothers gained weight excessively with the highest proportion in obese women (57%). LGA infants accounted for 5.7% of the birth cohort with obese women having the highest proportion as compared to underweight women (9.4% vs 2.1%, p<0.0001); women who gained weight excessively were more likely to have a LGA infant as compared to women who did not gain weight excessively (8.5% vs 3.8%, p<0.0001). Obese women who had gained weight excessively were 55% more likely to have a LGA infant compared to obese women who did not gain weight excessively (odds ratio (OR) = 1.55; 95% CI 1.50-1.62). Normal weight and underweight women who gained weight excessively were almost 2.4 and three times more likely, respectively, to have a LGA infant as compared to their counterparts who did not gain weight excessively (normal weight: OR=2.43; 95% 2.36-2.40; underweight: OR=2.98; 95% CI 2.61-3.41). Similar results were found after adjusting for covariates in the BMI stratified models.

Conclusion: These results indicate the need to address excessive weight gain during pregnancy with normal weight and underweight women, as well as with overweight and obese women.

# Board 11, Poster 44

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Adiposity Indicators in Women With and At Risk for HIV infection. The Women's Interagency HIV Study |
| **Authors** * | Dellinger E*, Weber K, Keating S, Holman S, Cohen M, Minkoff H, Gustafson D. |
| **Institutional Affiliations For Each Author.** * | State University of New York – Downstate Medical Center, Cook County Health & Hospitals System/Hektoen Institute of Medicine, Blood Systems Inc., State University of New York – Downstate Medical Center, Stroger Hospital, Maimonides Medical Center, State University of New York – Downstate Medical Center |
| **Corresponding Author Email** * | elaine.dellinger@downstate.edu |

**Structured Abstract** *

Background: Antiretroviral therapies (ART) are often accompanied by overweight and obesity (OWOB). HIV+ adults (particularly women) are OWOB. Since both aging and OWOB are associated with risk of cognitive im-pairments and dementia in populations without HIV, it is vital to understand the association of OWOB with cognition in ART-adherent HIV+ or at risk (HIV-) women. OWOB are estimated via BMI and waist circumference (WC). Blood measures of leptin are positively correlated (r=0.70) with BMI and WC in HIV+ and HIV- women enrolled in the Brooklyn Women's Inter-agency HIV Study (WIHS). Leptin has numerous effects on brain development and health. In uninfected populations, higher BMI and leptin levels are protective against later life cognitive impairments and dementias. This contrasts higher mid-life BMI or WC as risk factors for late-life dementia. Within the WIHS at average age 40 years, higher BMI and lower leptin levels were associated better cognition. In Summer 2017, we will explore these associations in a WIHS metabolic subsample at average age 50 years.

Material and Methods: Type of Study. A cross-sectional analysis including BMI, WC, and cognitive test scores among HIV+ and HIV- women; a leptin substudy at 2 WIHS sites (Brooklyn/Chicago). Sample size. BMI, WC, cognitive tests, and blood leptin levels are measured in 417 women. Outcome measures. Cognitive tests include: Trails B, Stroop Interference, Symbol Digit, Stroop Color Naming and Reading, Letter-Number Span, Hopkins Verbal Learning Test, Letter Fluency, Semantic Fluency, Wide Range Achievement Test (WRAT-3).

Exposure measures. BMI is calculated as kg/m2; and WC measured in inches according to the U.S. National Health and Nutrition Examination Survey (NHANES) III protocol. Leptin levels are determined using leptin ELISAs (Millipore, Billerica, MA) by Blood Systems, Inc. (San Francisco, CA).

Analysis Plan: Following descriptive evaluation, linear regression analyses will estimate associations between BMI, WC, and leptin, and raw cognitive test scores with consideration of age, race, education, WRAT score, HIV medications, CD4 count, CD4 nadir, Diabetes Mellitus, systolic and diastolic blood pressures, use of anti-hypertensive medications, use of exogenous insulin, blood lipid levels, smoking status, and use of marijuana, 'crack,' cocaine, and/or heroin.

Results: Our results will be shared for the first time at NYCROF 2017 in New York City.

Conclusions: We anticipate a similar finding as our previous reports, however we also expect aging WIHS participants to perform worse on cognitive tests, have higher levels of BMI and WC, and exhibit more variability in blood leptin levels.

# Board 12, Poster 45

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Weight loss efficacy is enhanced with photo recognition of food in a weight loss app |
| **Authors** * | Daniela Ben Neriah*, M.Sc.; Allan Geliebter, PhD |
| **Institutional Affiliations For Each Author.** * | Institute of Human Nutrition Columbia University in the City of New York, Mount Sinai St. Luke's Hospital |
| **Corresponding Author Email** * | Allan.Geliebter@mountsinai.org |

**Structured Abstract** *

Background: Tracking of dietary intake is a useful tool in weight management and is associated with increased weight loss in individuals who are overweight. Mobile phone based apps can help with tracking and can provide instant feedback of calories consumed. The process of food tracking can become daunting, and many individuals have difficulties adhering to a program for long periods. Recently, there have been new technologies that utilize photography for image recognition to identify specific foods and record consumption.
Methods: We have begun studies with the Lose It app, a mobile based weight loss program, to test their food image recognition tool, Snap It. In a retrospective study analysis of app users (n = 116,693) comparing users who have used the additional Snap It feature to users who have not.
Results: Of the app users, those who used the Snap It feature (n = 955) logged on average for 17.7 days (p-value: < 0.001) more than those who did not use the new feature (n = 115,738). Both groups lost weight (mean = -3.21 kg, SD = ± 4.48 kg), but the group that used the Snap It feature lost more weight (mean = -3.69 kg , SD = ± 4.56 kg) than those who did not use the feature (mean = -3.20 kg, SD = ±4.47 kg) after adjusting for age and gender. The effect diminishes when accounting for length in the program.
Conclusion: Use of a photo app appears to enhance compliance and weight loss, but this effect is likely attributed to increased motivation of the experimental group. We are currently conducting an RCT to test these findings.

# Board 12, Poster 46

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Associations between types of physical activity and sedentary behaviors with overweight and obesity among adults in NHANES 2013-2014 |
| **Authors** * | Daniel Kim, Chrisa Arcan, Fusheng Wang |
| **Institutional Affiliations For Each Author.** * | Department of Biomedical Informatics and Department of Computer Science, Department of Family Population and Preventive Medicine, Department of Biomedical Informatics and Department of Computer Science, Stony Brook University |
| **Corresponding Author Email** * | Hrisanti.arcan@stonybrookmedicine.edu |

**Structured Abstract** *

Purpose: Physical and sedentary activities are major modifiable risk factors for overweight, obesity and related chronic diseases. Longitudinal studies have shown that different levels of physical activity (PA) may attenuate the long-term weight gain among adults. Engaging in sedentary activities was associated with obesity independent of PA. However, little is known about the link between types of physical and sedentary activities and overweight or obesity among a large sample of adults.

Methods: Cross-sectional data from CDC's National Health and Nutrition Examination Survey (NHANES) 2013-2014 were used to examine associations between overweight or obesity and types of PA (vigorous/moderate work, vigorous/moderate recreational, walking and bicycling for transportation) and sedentary (TV or video watching, computer or video game use) between male and female adults. Logistic regression using Python's statsmodels module was used to examine associations between overweight or obesity with related PA and sedentary variables by gender, adjusted for race, age, education, marital status, US birth, and household size.

Results/Findings: A total of 4,046 adults (age: 20-65 years, mean: 42 years; male: 49%) were included. Overweight prevalence was higher among males (38%) than females (25%) but obesity prevalence was higher among females (43%) than males (34%). Males engaged in all types of PA more frequently than females, while both genders had similar frequency of sedentary behaviors. Among males, walking or biking to work was linked to lower obesity and higher TV use, being African American or Hispanic, being born in the US, and being married was linked to higher obesity. Among females, engaging in vigorous recreational PA was linked to lower obesity but TV use, being African American or Hispanic, older, and being born in the US were linked to higher obesity. Among overweight adults, only higher age was significant among males.

Conclusions: Modifiable factors such as increased physical activity, either recreational or work related, and reduced TV watching can be protective against obesity among African American or Hispanic male and female adults.

# Board 12, Poster 47

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Hidden In Plain Sight: Unexpected Food Sources Around Urban High Schools – Implications For Obesity |
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**Structured Abstract** *

Background: Individuals increasingly obtain more of their own foods and beverages and make more of their own dietary decisions during adolescence. Adolescents may be exposed to a variety of foods and beverages around their schools, yet not all food-and/or-beverage sources may be intuitive. Understanding non-intuitive sources may provide a more complete picture of total food-and-beverage exposure for adolescents, and greater appreciation of dietary risk for obesity. We characterized all food-and/or-beverage sources around select high schools in a diverse urban county, considering dietary risk and implications for obesity.

Methods: Researchers selected 10 demographically distinct high schools across the Bronx, NY. Teams walked all streets within a ½-mile of each school to identify all sources of food and/or beverage. Sources were categorized as 'food businesses' (e.g., supermarkets, grocery stores, vending machines, farmers' markets, street vendors, restaurants) or 'other businesses' (e.g., laundromats, auto shops, hardware stores, hair salons, dollar stores). 'Healthful' items were fruits, vegetables, whole grains, nuts, milk, or water. 'Less-healthful' items were a variety of processed or prepared, calorie-dense, low-nutrition foods and drinks (candies, chips, cookies, sodas, burgers, fries, etc.).

Results: There was considerable variation in the number of businesses around each school (n=72-373) and in the proportions offering food and/or drink (29.7%-54.9%), any healthful items (28.8%-45.6%), or less-healthful items only (1.7%-8.9%); p values all < 0.03 for chi-squared tests. 'Less-healthful' items predominated around all schools. 'Food businesses' almost always offered some healthful options (in 91.3%-98.1% of cases), but offered 'less-healthful' choices as well. 'Other businesses' accounted for 54.9%-77.5% of all businesses around each school, with 9.3%-32.1% offering food and/or drink and 17.6%-59.5% of these offering only 'less-healthful' items. 'Less-healthful' items from 'other businesses' included candies, chips, cookies, frozen confections, and sodas. 'More-healthful' items from 'other businesses' included fresh produce, canned beans, salsa, granola bars, nuts, milk, water, and seltzer.

Discussion: Food-and/or-beverage sources around schools often included a wide range of businesses not generally thought of as focusing on food-and/or-beverage provision. The extent and healthfulness of food-and/or-beverage offerings differed substantially around selected schools, although there was a preponderance of 'less-healthful' options in all cases. Food-and/or-beverage sources around schools may present a risk for 'less-healthful' consumption and may increase the risk for obesity in adolescents.

# Board 12, Poster 48

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Comparisons and associations between sedentary behaviors and obesity among adults in NHANES between 2003–2004 and 2013–2014 |
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**Structured Abstract** *

Purpose: Extended time engaging in different types of sedentary activities have been shown to be major risk factors for obesity. This study examines the differences in sedentary activities with data from 2013–2014 and from 2003–2004, to examine temporal changes and associations between sedentary activities and obesity.
Methods: Analysis of cross-sectional data from the CDC's National Health and Nutrition Examination Survey (NHANES) for 2003–2004 and 2013–2014 were used to examine temporal changes in sedentary activities (TV or video watching, computer or video game use) between male and female adults, and associations between sedentary activities and obesity. Welch's t-tests were used to find differences between the two datasets. For obesity modeling, the data from both time periods were combined into one dataset, with membership in the new dataset being coded as a binary variable. After a suitable model was created, it was tested on the individual datasets for consistency. Logistic regression with Python's statsmodels module was used to examine these associations, adjusted for race, age, education, marital status, US birth, and household size.
Results/Findings: Over the span of a decade, the average number of hours spent watching TV or videos has increased, but the average hours per day using the computer or playing games has not increased. The average BMI and obesity prevalence rates have both increased. In the combined dataset, obesity prevalence and average BMI were higher among females than males. Being a part of the new dataset was coded as a binary variable which was significant; therefore, living during 2013–2014 significantly increased obesity rates compared to living during 2003–2004. More TV/video time was linked to greater obesity rates, while computer usage was not associated with obesity. Age was also correlated with greater obesity rates, as was being a female.
Conclusions: Time spent watching TV or videos have increased between the 2003–04 and 2013–14; obesity rate has also increased. Watching TV/videos was significantly associated with obesity, while using the computer or playing video games was not.

# Board 13, Poster 49

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Disordered Eating and Psychopathology: Characteristics of an Urban Community Sample of Bariatric Surgery Patients |
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**Structured Abstract** *

Background: The aim of the present analysis was to evaluate emotional health and eating disorder symptoms in a community sample of ethnic minority, obese, weight-loss surgery candidates drawn from the New York metropolitan area, in comparison to a demographically matched normal weight sample, to identify potential predictors of weight-loss outcomes after surgery in this under-researched population.
Methods: Self-report measures of emotional eating, emotion dysregulation, eating pathology, anxiety, depression, perceived stress, impulsivity, reward valuation, and childhood trauma were administered to 44 bariatric surgery candidates (African-American 27.69%, Hispanic 38.46%, women = 34, $\bar{x}$ age = 34.03 ± 1.63 years, BMI = 45 ± 0.84 kg/m2,), prior to weight-loss surgery and again approximately 3 months post-surgery (post-surgery n = 30). The same instruments were administered to a healthy-weight comparison group (N = 29; African-American 29.23%, Hispanic 26.15%, $\bar{x}$ age = 29.6 years ± 1.45, BMI = 21.6 ± .77 kg/m2). Patients at baseline were compared to controls using a non-parametric, Wilcoxon rank-sums test, and a signed-rank test was used to compare pre- and post-surgery scores in the patient group.
Results: Patients' scores were significantly ($p < .05$) higher than controls' on measures of restraint (group median = 1.8, IQR = 2 versus = 0 and .6) global eating pathology (median = 2.63, IQR = 1.27 vs. .26, .7), hunger (median = 7, IQR = 5 vs. 3, 3), disinhibition (median = 7, IQR = 6.5 vs. 3, 4), trait-anxiety (median = 34, IQR = 10 vs. 29, 14), food addiction (median = 3.5, IQR = 5.5 vs. 0, 1), binge-eating (median = 2, IQR = 10 vs. 0, 2), and perceived stress (median = 67, IQR = 23 vs. 52, 28). Patients did not differ significantly from controls at baseline on measures of emotional eating, emotion dysregulation, depression, food valuation, and childhood trauma. After surgery, patients' scores decreased significantly ($p < .005$) on measures of emotional-eating ($\bar{x}$ decrease = -0.42, SE = .13), global eating pathology (-.7, SE = .19), hunger (3.7, SE = .58), disinhibition (-3.6, SE = .55), binge-eating (-5.1, SE = 1.33) and food valuation (.61, SE = .16), and increased on restraint ($\bar{x}$ = 5.6, SE = .96).
Conclusion: High levels of stress, anxiety, and eating pathology reported by African-American and Hispanic weight-loss surgery candidates may predict post-surgical outcomes. In addition, the decrease in patients' eating pathology scores after surgery suggests that surgery may partially ameliorate disordered eating.