# 3rd Annual NYC Regional Obesity Forum

## Columbia University

535 West 116th Street
Lerner Hall, Lerner Auditorium
New York, New York 10027

## Keynote Speakers

### Rebecca A. Haeusler, Ph.D.

Assistant Professor of Pathology and Cell Biology
Naomi Berrie Diabetes Center
Columbia University College of Physicians and Surgeons

### Michael D. Jensen, M.D.

Professor of Medicine
Division of Endocrinology, Diabetes, Metabolism, & Nutrition
Mayo Clinic

## Tuesday, September 26th, 2018
9:00 am – 5:00 pm
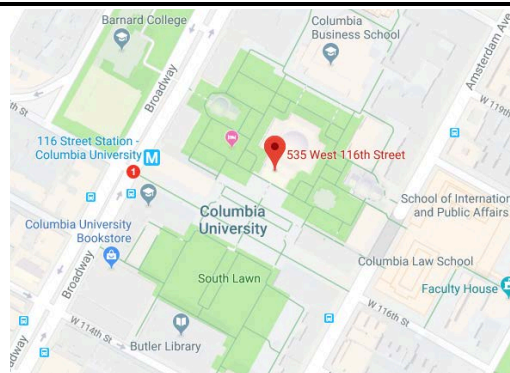
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# Support

# Visiting Columbia University

Columbia University stretches from West 114th St. to West 120th St between Broadway and Amsterdam on Manhattan's Upper West Side.

The 2018 NYCROF meeting will be held in Lerner Hall Auditorium. The main entrance to the campus is at:

Columbia University
535 West 116th Street
New York, New York 10027

# Directions to Columbia University

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Trains to New York arrive at Grand Central Station or Pennsylvania Station; buses stop at the Port Authority Bus Terminal. Visitors arriving at these stations can take either public transportation or a taxi north to the campus.

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Five bus lines (M4, M5, M11, M60, M104) and one subway line (the No. 1 local) serve the Columbia neighborhood. The Columbia stop for the buses and the subway is 116th Street. The M60 bus is a direct link between campus and LaGuardia Airport.

Maps of bus and subway routes are available on the Metropolitan Transportation Authority website:
http://web.mta.info/maps/

**By Car**
If you are travelling top Manhattan by car, please visit the website below for driving directions or use the following GPS address:
535 West 116th Street New York, New York 10027
https://visit.columbia.edu/content/directions-morningside-heights-campus-1

*MTA bridges and tunnels do not accept cash for tolls. For more information on how to pay visit:
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|---|---|---|
| 1090 West 114th Street | 420 Riverside Dr (120th) | 234 West 108th Street |
| (between Broadway & Amsterdam Avenue) | (between Claremont Avenue & Riverside Drive) | (between Broadway & Amsterdam Avenue) |
| 212-523-1051 | 212-870-6736 | 212-222-8800 |
| Open from 6 a.m. to midnight | Open from 6 a.m. to midnight | Open from 7 a.m. to 2 a.m. |
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# Lerner Hall Auditorium
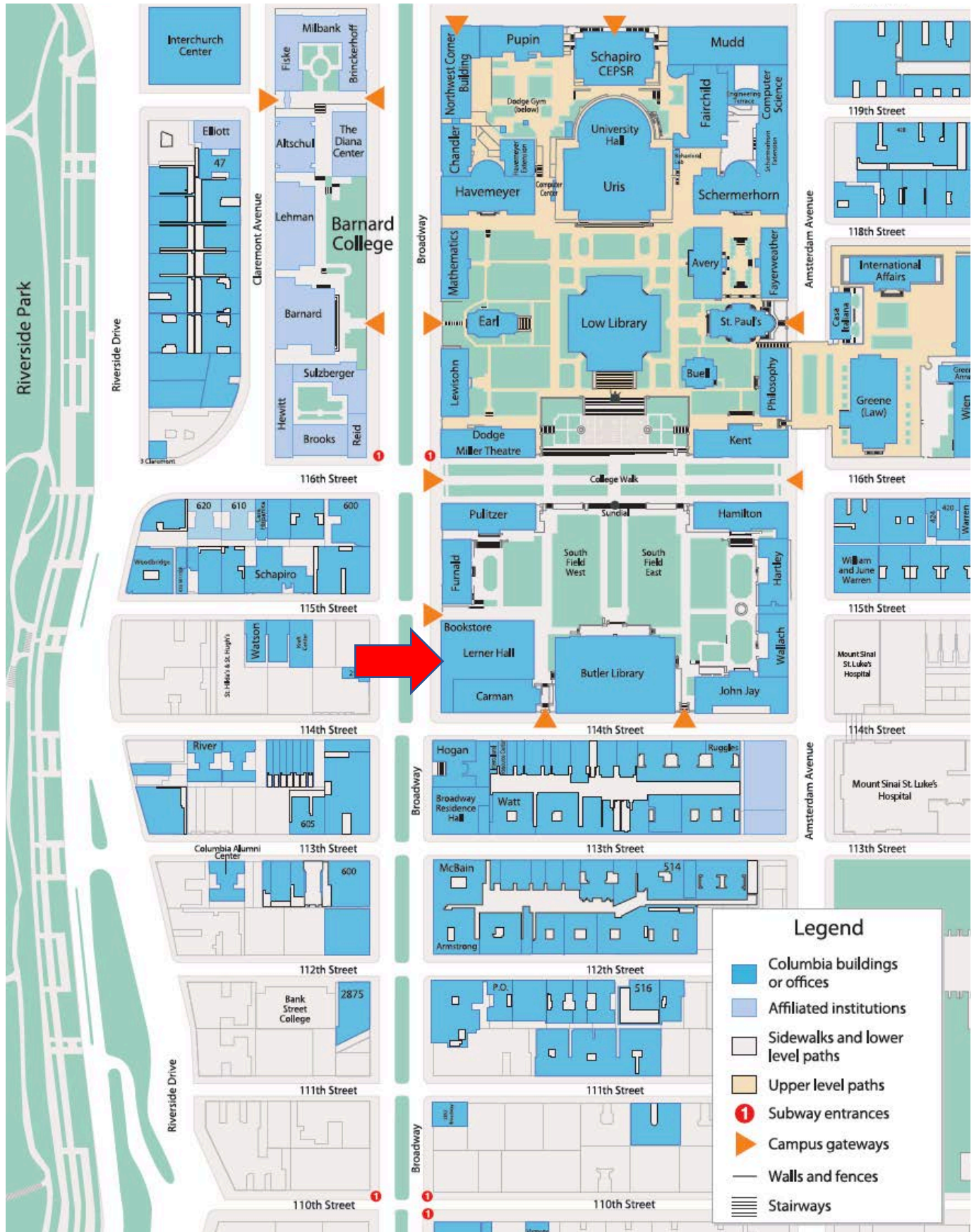
# New York City Regional Obesity Forum

## Wednesday, September 26, 2018
## Columbia University
## Lerner Hall

| Time | Duration | Session |
|---|---|---|
| 9:00-9:30 | 30min | Arrive, coffee, poster set-up |
| 9:30-9:40 | 10min | Welcome remarks |
| | | **Chair:** Elodie Picarda (Albert Einstein College Of Medicine) |
| 9:40-10:00 | 20min | **Short talk 1** - Peristera-Ioanna Petropouloua (Columbia University Medical Center) - "Translational potential of the anorexigenic effects of osteoblast-derived Lipocalin-2 in monkeys and humans" |
| 10:00-10:20 | 20min | **Short talk 2** - Diana Cousminer (Children's Hospital of Philadelphia, University of Pennsylvania) - "Genetic study of longitudinal pubertal height growth describes links with adult health" |
| **10:20-11:10** | **5 intro+45min** | **Keynote 1: Michael Jensen (Mayo Clinic, Rochester, Minnesota) - "Regulation of Adipose Tissue Lipolysis in Obesity - Is There a Role for Inflammation?"** |
| 11:10-11:40 | 30min | Coffee/Networking/Poster viewing |
| | | **Chair:** TBD |
| 11:40-12:00 | 20min | **Short talk 3** - Narges Razavarian (NYU Langone Health) - "Predicting Childhood Obesity Using Electronic Health Records And Publicly Available Data" |
| 12:00-12:20 | 20min | **Short talk 4** - Sandra Steensels (Weill Cornell Medical College) - "Acyl-CoA thioesterase 9 (Acot9) promotes weight gain and hepatic glucose production (HGP)" |
| 12:20-2:00 | 100min | Lunch/Networking/Poster viewing and presentations (between 12:50 and 2:00) |
| | | **Chair:** Troy Roepke (Rutgers University) |
| 2:00-2:20 | 20min | **Short talk 5** - Mary Jo Messito (New York University School of Medicine) - "Primary Care Based Child obesity Prevention Beginning in Pregnancy: Starting Early Program Impacts on Infant Feeding and Child Weight" |
| 2:20-2:40 | 20min | **Short talk 6** - Maria Caterina De Rosa (Columbia University Medical Center) - "Transcription Factors Defined by Single Cell RNA-seq Program Human Stem Cells into PVH Neurons" |
| **2:40-3:30** | **5 intro+45min** | **Keynote 2: Rebecca Haeusler (Naomi Berrie Diabetes Center, Columbia University Medical Center, New York) - "Bile acids in obesity: new tricks for old molecules"** |
| 3:30-4:00 | 30min | Coffee/Networking/Poster viewing |
| | | **Chair:** Ryan Walker (Icahn School of Medicine at Mount Sinai) |
| 4:00-4:20 | 20min | **Short talk 7** - Apurva Khedagi (Columbia University) - "Cardiometabolic risk factors with severity of obesity over childhood" |
| 4:20-4:40 | 20min | **Short talk 8** - Priya Bhardwaj (Weill Cornell Medical College) - "Local factors secreted by obese breast adipose tissue contribute to DNA damage in breast epithelium of BRCA mutation carriers" |
| 4:40-4:50 | 10min | Closing remarks, poster awards |

# Short Talk 1

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Translational potential of the anorexigenic effects of osteoblast-derived Lipocalin-2 in monkeys and humans |
| **Authors** * | Peristera-Ioanna Petropouloua*, Ioanna Mosialoua, Steven Shikhela, Fabiana Bahnab, Norman Simpsonc, Mihran Bakalianc, Suham Kassirc,d, Patrick Carberrye, Chrissy May Longf, Lawrence Shapirob, Akiva Mintze, Matthew Jorgensenf, Mark Underwoodc,d, John Mannc,d, Cyrille B. Confavreuxh and Stavroula Koustenia |

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**Structured Abstract** *

Background

In this study we sought to decipher the properties of a new anorexigenic hormone that we recently discovered, Lipocalin 2 (LCN2). In wild-type (WT) mice, circulating LCN2 levels increase 3-fold within 1 hour of refeeding, due to an increase of Lcn2 expression specifically in osteoblasts. Intraperitoneal administration of recombinant LCN2 to WT and Lcn2osb-/- mice suppresses food intake within 1 hour. Moreover, LCN2 crosses the blood brain barrier, binds to melanocortin 4 receptor (MC4R) in the hypothalamus and activates anorexigenic pathways. These observations suggest that upregulation of Lcn2 after feeding may be a physiological satiety signal and prompted us to examine whether such a mechanism is conserved in monkeys and humans.

Methods

Three male African green monkeys were intravenously injected with saline and 3 with recombinant human LCN2 (rh-LCN2; 0.0375 mg/kg) for 5 days. Food consumption was measured daily. Post-mortem baboon and human brain sections were incubated with 125I-rh-LCN2 to assess binding to the hypothalamus. To examine the relationship between LCN2 and postprandial regulation in humans, young normal weight (mean BMI $20.8 \pm 0.5$ Kg/m$^2$) and obese (BMI $45.6 \pm 1.8$ Kg/m2) women were given a 200kcal mixed meal (55% carbohydrate) following an overnight fast. LCN2 serum levels were measured before and after ingestion of the test meal.

Results

Daily treatment of lean monkeys with recombinant human LCN2 for 5 days showed a 23% decrease in appetite and a subsequent 1% decrease in body weight. Furthermore, autoradiography of baboon and human brain sections incubated with 125I-rh-LCN2, showed specific binding of the ligand in the hypothalamus of both species examined. In humans, LCN2 levels were significantly increased by over 50% within 1 h after meal ingestion in the normal weight group and remained significantly elevated by 40% for at least 2h. In contrast, LCN2 levels in obese women failed to increase in response to the 200 kcal mixed meal.

Conclusions

In summary, we show that rh-LCN2 treatment in monkeys associates with appetite suppression and weight loss. Additionally, rh-LCN2 binds to the hypothalamus of both baboon and human brain sections, suggesting the same anorexigenic mechanism previously observed in mice. Furthermore, our results from the human study provide the first evidence that the anorexigenic function of LCN2 may be conserved in humans and that its postprandial response and function is impaired in obesity. Such findings are extremely important for the development of a novel and much-needed approach to the therapy of obesity.

# Short Talk 2

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|---|---|
| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Genetic study of longitudinal pubertal height growth describes links with adult health |
| **Authors** * | Diana L. Cousminer* on behalf of the Early Growth Genetics Consortium |
| **Institutional Affiliations For Each Author.** * | Children's Hospital of Philadelphia, University of Pennsylvania |
| **Corresponding Author Email** * | cousminerd@email.chop.edu |

**Structured Abstract** *

Background: Distinct growth patterns during puberty correlate with adverse health outcomes such as poor cardiometabolic health; however, the genetic mechanisms mediating differences in growth trajectories remain largely unknown.

Methods: We leveraged >39,000 trans-ethnic samples from 20 cohorts with repeated height measurements across childhood. Longitudinal height was modeled using Super-Imposition by Translation And Rotation (SITAR) growth curve analysis for three parameters determined for each individual's growth curve: a-size, representing taller or shorter than the mean; b-timing, representing timing of the growth spurt earlier or later than the mean; and c-velocity, which is the tempo of the pubertal growth spurt, a property of pubertal growth that has not previously been subjected to genetic analyses. We performed Haplotype Reference Consortium imputation followed by genome-wide association meta-analysis with GWAMA. Independent loci detected with GCTA-COJO were fine-mapped using trans-ethnic data with MR-MEGA, followed by credible set analysis.

Results: We observed six genome-wide significant loci. All were previously reported for related traits, including body size from infancy to adulthood, adiposity, and age at menarche. Next, we used LD score regression to investigate genetic correlations with traits and diseases. a-size strongly correlated with body size traits across the life-course and health traits such as coronary artery disease. b-timing was highly correlated with pubertal timing and body fat/BMI traits, plus adult fasting insulin and 2hr glucose adjusted for BMI. Interestingly, although c-velocity correlated with puberty timing and body size throughout life, these correlations were more reflected in height than adiposity phenotypes. However, c-velocity also correlated with measures of adult health, including glycemic traits (insulin, HOMA-IR), metabolites (HDL, VLDL concentrations), bone (femoral neck bone mineral density), lung function and lung cancer.

Conclusions: Combined, these findings suggest that the tempo of pubertal development (c-velocity), often challenging to assess and regularly overlooked in epidemiological studies, may provide insight into adult health outcomes, and these correlations may be partly independent of adolescent adiposity. Additionally, our results should help in the identification of specific growth trajectories impacting lifelong health.

# Short Talk 3

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Predicting Childhood Obesity Using Electronic Health Records And Publicly Available Data |
| **Authors** * | Robert Hammond, Rodoniki Athanasiadou, Silvia Curado, Yindalon Aphinyanaphongs, Courtney Abrams, Mary Jo Messito, Rachel Gross, Michelle Katzow, Melanie Jay, Narges Razavian*, Brian Elbel |

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**Structured Abstract** *

Background
Because of the strong link between childhood obesity and adulthood obesity comorbidities, and the difficulty in decreasing BMI later in life, effective strategies are needed to address this condition in early childhood. The ability to predict obesity before age five could be a useful tool, allowing prevention strategies to focus on high risk children. The few existing prediction models for obesity in childhood have primarily used data from longitudinal cohort studies, relying on difficult to collect data that are not readily available to all practitioners. We used existing EHR data from a safety net health system in New York City to predict obesity at age five using machine learning approaches.

Methods
We used EHR data from the first two years of life to predict obesity status between the ages of 4.5 and 5.5 years. We used a variety of machine learning algorithms to perform both binary classification (predicting probability of obese/non-obese status between 4.5 and 5.5 years), and regression on the normalized median BMI (predicting the expected median BMI between 4.5 to 5.5 years).

Results
Using our best performing model, LASSO regression, for girls we were able to predict obesity with an Area Under the Receiver Operator Characteristic Curve (AUC) of 81.7% [81.4%, 81.9%] with 34.3% of the variance of BMI at age five being explained; for boys we were able to predict obesity with an AUC of 76.1% [76.0%, 76.3%] with 28.1% of variance being explained from our best multivariate model. For girls LASSO regression with LASSO feature selection was the best performing model, while for boys single feature regression models using the average WFL Z-score between 19 and 24 months performed as well or even better than the full-feature LASSO regression model.

Conclusions
We were able to predict obesity at age five using real-world electronic health record (EHR) data with an AUC comparable to cohort-based studies, reducing the need for investment in additional data collection. Our results suggest that machine learning approaches for predicting future childhood obesity would improve the ability of clinicians and researchers to drive future policy, intervention design and the decision-making process in the clinical setting.

# Short Talk 4

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Acyl–CoA thioesterase 9 (Acot9) promotes weight gain and hepatic glucose production (HGP) |
| **Authors** * | Sandra Steensels, Jixuan Qiao, Corey D. Holman, Curtis J. Bare and Baran A. Ersoy |
| **Institutional Affiliations For Each Author.** * | Department of Medicine, Weill Cornell Medical College, New York, NY, USA |
| **Corresponding Author Email** * | bae2008@med.cornell.edu |

**Structured Abstract** *

Background: Pathogenesis of obesity–induced disorders such as type 2 diabetes and non–alcoholic fatty liver disease are associated with increased lipotoxicity. Acot9 deactivates fatty acyl–CoAs by hydrolyzing them into free fatty acids and CoA in vitro. Suggestive of a pathogenic role, Acot9 expression increases in the livers of mice following high fat diet (HFD, 60% kcal) and inhibits insulin signaling in cultured cells. Aim: This study was designed to explore the role of Acot9 in hepatic lipid and glucose homeostasis in vivo. Methods: Mice with systemic ablation of Acot9 (Acot9−/−) were generated by the insertion of Frt–flanked STOP cassette. Liver-specific rescue of Acot9 (Acot9LWT) was achieved by the adenoviral delivery of Flp recombinase into Acot9−/− mice. Transgenic mice and wild type littermates (Acot9+/+) were weaned to a chow (n=9–12) or HFD (n=10–12) for 11 weeks. Metabolic cages were used to monitor body weight, energy expenditure, physical activity and food intake. Glucose homeostasis was measured by tolerance tests to glucose (GTT), insulin (ITT) and pyruvate (PTT). Body composition was assessed by magnetic resonance imaging. Activity of key regulators for lipogenesis were assessed by immunoblots. Results: When fed a chow diet, Acot9−/− mice did not manifest any metabolic abnormalities compared to Acot9+/+ mice. However, following HFD, Acot9−/− mice exhibited reduced body weight (7%, P<0.01), gonadal fat (17%, P<0.05) and fasting blood glucose (18%, P<0.05). Decreased body weight and adiposity were associated with increased energy expenditure (5%, P<0.05). Acot9−/− mice did not have improved insulin sensitivity as evidenced by unchanged responses to GTT and ITT. Decreased fasting blood glucose was instead associated with reduced HGP (27%, P<0.01) as determined by PTT. The reduction in HGP was neutralized upon rescue of hepatic Acot9 expression in Acot9LWT mice. Further supportive of hepatic contributions, the loss of Acot9 expression blunted the expression of fatty acid synthase (28%, P=0.06) and increased phosphorylation of acetyl–CoA carboxylase (27%, P<0.01) in the livers of Acot9−/− mice, which is indicative of decreased de novo lipogenesis. Conclusions: Our results indicate that Acot9 impairs lipid and glucose homeostasis and contributes to obesity and metabolic disorders in the setting of HFD, at least in part by increasing energy expenditure, decreasing hepatic fatty acid synthesis and increasing HGP. Therefore, Acot9 may represent a novel target in the treatment of metabolic disorders.

# Short Talk 5

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Primary Care Based Child obesity Prevention Beginning in Pregnancy: Starting Early Program Impacts on Infant Feeding and Child Weight |
| **Authors** * | Mary Jo Messito, MD*, Michelle Katzow MD MS, Alan Mendelsohn MD, Rachel Gross MD MS |
| **Institutional Affiliations For Each Author.** * | New York University School of Medicine |
| **Corresponding Author Email** * | mary.messito@nyumc.org |

**Structured Abstract** *

Background: Prenatal and pediatric primary care represent a unique opportunity to reach high-risk families for child obesity prevention. Obesity-promoting diet/activity practices begin during pregnancy. The "Starting Early Program" (StEP) is one of the first comprehensive child obesity prevention programs using primary care to target low-income, Hispanic families.

Objective: to determine StEP impacts on infant feeding and activity practices and infant weight.

Methods: Randomized controlled trial enrolled pregnant women in the 3rd trimester to standard primary care control group vs. the StEP intervention: prenatal and postpartum nutrition counseling and nutrition and parenting support groups coordinated with primary care visits. English or Spanish speaking Hispanic women, ≥ 18 years old with an uncomplicated singleton pregnancy were included. Growth parameters obtained from medical records, weight for age z-scores (W/Agez) based on WHO growth charts, BMI percentiles based on CDC growth charts. Feeding activity practices and styles assessed by 24-hour recalls and validated surveys. Assessments performed at baseline during pregnancy and at infant age 3, 10, and 24 months. Statistical analysis: t-tests, chi square, linear and logistic regression. Within group analyses to explore impact of intervention dose.

Results: Baseline prenatal: (n=566): 80% immigrants, 90% receive WIC, 25% depressive sx, 30% food insecure, 29% with obesity. 24% C-section delivery 50% girls, mean birth weight z =-.02. No differences in baseline characteristics. At infant age 3 and 10 months (n = 456, and 412 respectively) StEP mother infant dyads had higher exclusive breastfeeding, (43% vs. 33% p=.04) daily tummy time, (41% vs. 29% p=.01) not giving juice (39% vs. 24% p=.003), family meals (83% vs. 70% p=.006) and less early introduction of complimentary foods (6% vs. 17% p<.01) than the control group. Intervention dyads also had decreased cereal in the bottle and non-responsive maternal infant feeding styles. StEP intervention infants had lower mean W/age z-scores at 24 months (n=394, 0.84 vs 0.62 p=.05). Median number of sessions attended by age 2 years: 7 out of possible 12 (range 1-12). Within the intervention group, attending ≥ 7 sessions reduced the risk of BMI ≥ 85th% at age 2 by 28%.

Conclusions: Starting Early intervention infants had healthier feeding and activity in infancy and lower weight at 2 years, with a dose dependent reduction in overweight prevalence. Findings demonstrate a scalable system with the potential to augment obesity prevention in primary care for at-risk families

# Short Talk 6

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Transcription Factors Defined by Single Cell RNA–seq Program Human Stem Cells into PVH Neurons |
| **Authors** * | Maria Caterina De Rosa1, Vidhu Thaker1, George Stratigopoulos1, Charles A. LeDuc1, Richard Rausch1, Qi Su2, Yurong Xin2, Jesper Gromada2, Wendy K. Chung1, Judith Altarejos2, Rudolph L. Leibel1, Claudia A. Doege1 |
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**Structured Abstract** *

Background

Body weight is regulated in part by hypothalamic feeding circuits. Neurons of the paraventricular nucleus of the hypothalamus (PVH) are central to these processes. Single cell RNA–sequencing enables identification of molecular signatures of transcription factors specific to PVH neuronal subtypes.

Methods

We subjected single cells isolated from punch biopsies of the PVH of male 5 weeks old C57BL/6Ntac mice to single cell RNA–seq utilizing a 10X Genomics Chromium platform. Cell Ranger v 2.0 was used for sequence alignments, and Seurat v 2.0 for the downstream analyses. Differential expression analysis was utilized to identify cell type–specific transcription factors. These transcription factors were confirmed via fluorescent RNAscope in situ hybridization in the mouse PVH region. To program human pluripotent stem cells into PVH neurons, we overexpressed the transcription factors SIM1 and BRN2 and others identified by RNAseq in the H9 human embryonic stem cell (hESC) line, using a doxycycline–inducible system. Cells were first plated on Matrigel. And after two days cells were re–plated on a layer of mouse astrocytes to undergo neuronal differentiation into PVH neurons.

Results

22,138 sequenced cells resulted in 22 distinct clusters. Sub–clustering of Tubb3–expressing clusters yielded 16 neuronal clusters, with a total of 2,376 cells. In our preliminary single cell analyses, the transcription factors Sim1 and Brn2 had been identified as the major transcription factors for neuropeptidergic PVH subpopulations (OXT, AVP, TRH, CRH); their expression was confirmed in vivo using RNAscope in situ hybridization.

During neuronal differentiation (following overexpression of SIM1 and BRN2) we performed a time course analysis which showed downregulation of the pluripotency master regulator NANOG coinciding with upregulation of neuronal marker TUBB3. In addition, we showed that SIM1 and BRN2 transgene expression activates endogenous SIM1 and BRN2, with consequent expression of OXT, AVP, TRH and CRH after 6 days of differentiation, confirming the single cell RNA–seq findings.

Conclusions

This study defines a strategy for the generation of specific neuronal cell types based on their transcription factors signature identified by single cell RNA–seq. The generation of such specific neuronal sub–populations should enable the investigation of the molecular mechanisms underlying human obesity and the discovery of novel therapeutic targets.

# Short Talk 7

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Cardiometabolic risk factors with severity of obesity over childhood. |
| **Authors** * | Apruva Khadegi*(1), Kun Qian(2), Molly McDonald(3), Benjamin Weaver(4), Sinead Christensen(3), Sara Lopez-Pintado(2), Joel Hirschhorn(3), Vidhu Thaker(5). |
| **Institutional Affiliations For Each Author.** * | 1: Columbia University College of Physicians and Surgeons, 2: Mailman School of Public Health, New York, NY, 3: Boston Children's Hospital, Boston, MA, 4: Boston University School of Medicine, Boston, MA, 5: Columbia University Medical Center |
| **Corresponding Author Email** * | vvt2114@cumc.columbia.edu |

**Structured Abstract** *

Background: The rising trend of severe childhood obesity portends an epidemic of cardiometabolic disease in the future generation(s). We aimed to study the effect of severity of obesity measured longitudinally on the cardiometabolic risk factors (CMRF) in children from underrepresented minorities and identify their co-occurrence over time.

Methods: Laboratory & clinical data was obtained for 702 children screened for Genetics of Early Childhood Obesity study from electronic health records (EHRs) at Boston Children's Hospital. Longitudinal trajectories of body mass index (BMI, min 3 readings, at least 6 months apart) were characterized into Class 1 (95th – 120% of 95th %tile for age), Class 2 (120–140% of 95th) and Class 3 (>140% of 95th) based on their median BMI percentile and > 50% values in the assigned class. The last available laboratory value for lipid profile, HbA1c, BP and ALT/AST were obtained. Prevalence of these variables and relative risk were calculated using generalized linear models controlling for age, sex, race/ethnicity, insurance status and food insecurity indicators. All analyses were performed in R version 3.4.

Results: Our cohort of children is focused on severe obesity: 43% Class 2 & 39% Class 3; 54% Female, 34% African American and 34% Hispanic ethnicity. There was a median of 365 readings of the CMRF/individual ranging from 6 months–21.14 years of age, and the last available value was chosen for analysis. Total CMRF (1: 18.4%, 2: 27.8%, 3: 31.5%, $p < 0.001$), ALT (1: 10.9%, 2: 15.1%, 21.9%, $p < 0.001$), BP (1: 43%, 2: 49.8%, 3: 58.5%, $p = 0.01$) and triglycerides (TG) (1: 36%, 2: 61.5%, 3: 62.3%, $p = 0.04$) were significantly increased with severity of obesity. The risk ratio of abnormal BP was 1.24 (CI 1.04–1.42, $p = 0.02$) and TG was 1.4 (CI 1.1–1.6, $p = 0.01$) for children in Class 3 with reference to Class 1, while other CMRF did not reach statistical significance while controlling for covariates. There was a significant difference in the prevalence of 1–5 CMRF with severity of obesity ($p < 0.001$) with an increasing trend with the obesity class ($p < 0.001$ for 1–5 comorbidities).

Conclusion: Severity of childhood obesity is associated with increase in the risk for cardiometabolic risk factors, especially their co-occurrence. The high prevalence of these comorbidities early in life adds another reason to the call for action. EHRs are a valuable resource to study the natural history of childhood obesity.

# Short Talk 8

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Local factors secreted by obese breast adipose tissue contribute to DNA damage in breast epithelium of BRCA mutation carriers |
| **Authors** * | Priya Bhardwaj*, Heba Zahid, Neil M. Iyengar, Xi Kathy Zhou, Dilip D. Giri, I-Chun Chen, Sofya Oshchepkova, Monica Morrow, Jason Spector, Clifford A. Hudis, Andrew J. Dannenberg, Kristy A. Brown |
| **Institutional Affiliations For Each Author.** * | Weill Cornell Medical College, Hudson Institute of Medical Research, Memorial Sloan Kettering Cancer Center, Weill Cornell Medical College, Memorial Sloan Kettering Cancer Center, Weill Cornell Medical College, Weill Cornell Medical College, Memorial Sloan Kettering Cancer Center, Weill Cornell Medical College, Memorial Sloan Kettering Cancer Center, Weill Cornell Medical College, Weill Cornell Medical College |
| **Corresponding Author Email** * | kab2060@med.cornell.edu |

**Structured Abstract** *

Background: Obesity is a well-established risk factor breast cancer. Studies have reported a greater incidence of breast cancer in obese BRCA mutation carriers compared with lean BRCA mutation carriers. Causative factors that drive the increased penetrance of breast cancer in obese women are poorly characterized. We hypothesized that obesity-associated dysfunctional adipose tissue may cause DNA damage in the breast epithelium of BRCA mutation carriers. If true, this would provide evidence for the initial insults associated with increased risk of tumor formation.

Methods: To assess DNA damage associated with obesity, breast FFPE sections were obtained from BRCA mutation carriers (N=82) with a body mass index (BMI) ranging from 17.7–38.5, as well as wildtype individuals. Immunofluorescence (IF) staining of the DNA damage marker γH2AX was carried out. To test the effect of locally secreted factors on DNA damage induction, breast adipose conditioned media was collected over 24 hours from primary breast adipose explants. Conditioned media was used to treat normal breast epithelial cells (MCF10A) harboring a heterozygous BRCA1 mutation or wildtype for BRCA. To assess the reversibility of obesity-associated DNA damage, a mouse model of caloric restriction was utilized whereby mice were fed a high fat diet for 10 weeks to induce obesity and then randomized to either continue ad libitum feeding or to a 30% caloric restriction diet for an additional 7 weeks until sacrifice. Mammary glands were harvested at sacrifice and FFPE sections were used for IF staining for γH2AX.

Results: We found that BMI was positively correlated with DNA damage in breast epithelium in BRCA mutation carriers. The number of γH2AX foci was also higher in the breast epithelium of obese versus lean women wild type for BRCA. Conditioned media from obese breast adipose tissue stimulated DNA damage in breast epithelial cells, suggesting a role for locally secreted factors in mediating DNA damage. Using a mouse model of obesity, we found that caloric restriction was sufficient to significantly reduce mammary gland DNA damage in association with decreased weight and restoring metabolic function.

Conclusions: These data show for the first time that obesity is associated with increased DNA damage in the breast epithelium of BRCA mutation carriers. Our results suggest that therapies targeting weight may reduce the increased DNA damage observed in obese BRCA mutation carriers and thereby decrease tumor formation in this high-risk population of women.

# Board 1, Poster 1

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|---|---|
| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Candidate gene analyses in families with discordance for extreme obesity. |
| **Authors** * | Michael H. Preuss, Claudia Schurmann, Wei-Dong Li, Richard A. Price, Danielle Reed, Ruth J.F. Loos |
| **Institutional Affiliations For Each Author.** * | The Charles Bronfman Institute for Personalized Medicine – Icahn School of Medicine at Mount Sinai, Regeneron Genetics Center, Department of Genetics – College of Basic Medical Sciences – Tianjin Medical University, University of Pennsylvania, Monell Chemical Senses Center, The Charles Bronfman Institute for Personalized Medicine – Icahn School of Medicine at Mount Sinai; The Mindich Child health and Development Institute – Icahn School of Medicine at Mount Sinai |
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**Structured Abstract** *

Obesity in the general population is a common multifactorial disease that results from an intricate interplay of genes and environment. On the other hand, extreme and early-onset forms of obesity are rare, typically caused by a single gene mutation, and environment has little impact. Recent genome-wide association studies have shown that genes that play a role in monogenic and syndromic obesity, also may play a role in common obesity (e.g. MC4R, SH2B1, POMC).
Here, we examine the role of rare and low frequency coding variants in 119 candidate genes in 393 families (n =1,501 individuals) discordant for extreme obesity. Families had at least one offspring with extreme obesity (BMI ≥ 40 kg/m2), who had at least one sibling or parent with no history of overweight or obesity (lifetime BMI < 25 kg/m2). DNA samples were genotyped using the Illumina MEGA array and the 1000G reference panel was imputed using a pedigree-phased approach. Analyses focused on 119 genes known to play a "definite" or "likely" role in monogenic forms of extreme and early-onset obesity in humans and/or animal models. We performed single-variant (additive/recessive) and gene-based association analyses of rare (MAF < 1 %) and low-frequency (MAF < 5%) missense variants. Case (BMI ≥ 40 kg/m2) vs control (BMI < 30 kg/m2) or extreme control (BMI < 25 kg/m2) status were used as outcomes. All analyses were adjusted for age and sex. Bonferroni corrected P-values at □ = 0.05 correspond to 10-4 for single-variant analyses and 4x10-4 for gene-based analyses.
None of the single-variant or gene-based results reached Bonferroni-corrected significance, but for 15 variants (P<0.01) and 8 genes (P<0.05) associations were promising. For example, missense variants in ALMS1 (rs45608038, p.Asn1786Asp, P=0.002), ATXN2 (rs7969300, p.Ser248Asn, P=0.0015), BBS1 (rs77298332, p.Ser410Phe, P=0.004) and BDNF (rs66866077, p.Glu6Lys, P=0.003) were found to be associated with increased risk of extreme obesity. Gene-based analyses highlighted SH2B1 (Pburden=0.004), GLUCY2C (Pburden=0.01), TLR5 (Pburden=0.01), among others. Despite a family design with discordance in extreme obesity, our study had insufficient statistical power to identify significant associations with rare and low-frequency variation in candidate genes. Nevertheless, our analyses have prioritized a number of genes and variants for further follow up analyses to elucidate the mechanisms through which they affect human obesity.

# Board 1, Poster 2

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Deletion of Myeloid RAGE Does Not Protect Against Insulin Resistance in Mice Fed High Fat Diet |
| **Authors** * | Lakshmi Arivazhagan*1, Evelyn MS Litwinoff 1, Henry R Ruiz 1, Hye Lim Noh 2, Sujin Suk 2, Jason Kim 2, Ravichandran Ramasamy 1 and Ann Marie Schmidt 1 |
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| **Corresponding Author Email** * | AnnMarie.Schmidt@nyumc.org |

**Structured Abstract** *

Background:
Obesity is a major global health problem, with over one third of adults in the US alone classified as obese. Obesity, partially attributed to "western" diets which are high in fats and carbohydrates, coupled with a sedentary lifestyle, often leads to a state of insulin resistance (IR), type 2 diabetes mellitus and its complications. We have previously shown that the receptor for advanced glycation end products (RAGE) and its ligands contribute to the pathogenesis of obesity and IR, as whole body Ager (Receptor for Advanced Glycation End-Products gene encoding RAGE) deleted mice fed a high fat diet (HFD) showed protection from weight gain and IR. Bone marrow transplanted from Ager-deleted into wild type mice fed HFD partially protected them from weight gain and IR. However, the mediating stem cell type has not yet been identified. Hence, we hypothesize that myeloid RAGE could play a major role in exerting protection from IR upon HFD feeding.

Methods:
We generated mice with myeloid-specific deletion of Ager (MDR) LyzMCre (+/+).Ager flox/flox along with LyzMCre (−/−).Ager flox/flox (Flox) and LyzMCre (+/+) (Cre) controls. Mice were fed either standard chow (LFD) or HFD (60% kcal/fat) for 3 months starting at 8 weeks of age. The mice were assessed for body mass and composition, glucose and insulin sensitivity, food consumption, energy expenditure, physical activity, whole body glucose metabolism by hyperinsulinemic-euglycemic clamp studies and hepatic triglycerides.

Results:
After 3 months on LFD or HFD, no significant differences in body mass, body composition, food intake, energy expenditure and physical activity of the MDR mice were observed compared to the respective controls. However, insulin tolerance tests and hyperinsulinemic-euglycemic clamp studies showed decreased insulin sensitivity and decreased insulin action in the livers of MDR mice vs. controls, respectively, indicating that the MDR mice were more insulin resistant. To begin to determine the mechanism for increased IR, we measured the levels of liver triglycerides and report that the HFD-fed MDR mice showed significantly elevated levels of triglycerides in the liver vs. controls, suggesting poorer lipid metabolism in the liver, leading to liver steatosis and increased IR.

Conclusion:
Strikingly, unlike whole body Ager deletion, myeloid-specific deletion of Ager does not protect from HFD-induced IR, despite no differences in body mass or composition. The mechanisms underlying these findings are under active investigation.

# Board 2, Poster 1

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Receptor for Advanced Glycation End Products (RAGE) Modulates Expression of Abcg1 and Glo1 in Bone Marrow–Derived Macrophages in High Fat Feeding |
| **Authors** * | Karan Singh1*, Yuhan Hao2,3, Aristotelis Tsirigos2,3, Richard A. Friedman4, Ravichandran Ramasamy1, Ann Marie Schmidt1 |

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**Structured Abstract** *

Background: Obesity and diabetes are complex disease syndromes resulting from genetic–environmental interactions. The cell surface molecule receptor for advanced glycation end products (RAGE) plays a crucial role in the pathogenesis of obesity and diabetes and its complications. We previously reported that mice globally devoid of Ager (the gene encoding RAGE) display nearly complete protection from high fat diet (HFD)–induced obesity, insulin resistance and diabetes. Reconstitution of lethally irradiated wild type mice with Ager–deficient bone marrow resulted in partial but significant protection from obesity when mice were fed the HFD compared to mice recipients of wild–type bone marrow. To delineate the cell–intrinsic role of RAGE in myeloid cells, we studied bone marrow–derived macrophages (BMDMs) under basal and pathophysiological conditions.

Methods: BMDMs were grown in physiologic levels of glucose from non–diabetic male mice or from mice after four months of HFD. BMDMs from WT and Ager-deficient mice, aged 38 weeks, were subjected to Assay for Transposase–Accessible Chromatin using sequencing (ATAC-Seq) and RNA sequencing (RNA-seq) in N=4–5 mice/group.

Results: In ATAC–Seq and RNA–Seq studies, we identified that the expression of 32 genes is significantly different under the basal condition between Ager–deficient and WT BMDMs. These genes displayed similar patterns at the open–closed chromatin as well as at the transcriptome level. Under pathophysiologic condition (HFD), we identified 16 genes that are significantly different between the Ager–deficient and WT BMDMs. Ager-deficient BMDMs derived from the HFD–fed mice displayed significantly higher expression of the Abcg1 gene, which is involves in the reverse cholesterol transport, compared to the WT BMDMs. In addition, in basal condition and HFD, Glo1, which encodes the protein glyoxalase 1 (GLO1), which detoxifies pre–AGE species, demonstrated significantly more opened chromosome regions across transcription start sites and higher levels of mRNA, and, by Western blotting, protein levels, in Ager deficient vs. WT BMDMs. In parallel, we found that measurement of methylglyoxal (MG), a substrate for GLO1 and a pre–AGE species, was significantly lower in the Ager deficient vs. WT BMDMs.

Conclusions: ATAC–Seq and RNA–Seq studies revealed that RAGE regulates key genes involved in metabolic dysfunction, Abcg1 and Glo1, both of which were higher in BMDMs devoid of RAGE. Ongoing investigation is assessing the mechanisms by which RAGE regulates Glo1 and Abcg1.

# Board 2, Poster 2

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Clinical and functional characterization of Melanocortin 4 Receptor (MC4R) variants in African American and Hispanic children with severe early onset obesity (SECO). |
| **Authors** * | J. Zhou*(1), M.C. De Rosa*(1), A. Chesi(2), S. McCormack(2), B. Weaver(3), M. McDonald(4), K. Liimatta(1), M. Rosenbaum(1), C. Doege(1), J.N. Hirschhorn(4), S. Grant(2), V.V. Thaker(1) |
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**Structured Abstract** *

Background: MC4R mutations are the most common cause of monogenic obesity in children of European ancestry, prevalence 2.56%. Data are limited on the prevalence in children with SECO in African American and Hispanic ancestry/ethnicity. The objective of this study was to identify the prevalence of rare variants in MC4R in this population and perform their functional characterization.

Methods: We assessed the prevalence of rare variants in MC4R in children with SECO, defined as body mass index > 120% of the 95th percentile for age, documented before age 6, and present for at least 6 months. Subjects were recruited from 3 tertiary care U.S. hospitals and identified through clinical encounters or by using a validated algorithm for identification of SECO from Electronic Health Records (PMID: 27452794). Longitudinal analysis of BMI trajectory was performed using Super Imposition by Translation and Rotation (SITAR) to generate size, tempo, and velocity estimates for individual subject (PMID: 20647267). DNA was obtained under institution approved consent from the Biobank, or in person recruitment. MC4R variants
were identified by whole exome or targeted Sanger analysis. For 3 novel variants, we performed functional assessment of cAMP response to its endogenous ligand, α−MSH, and measured expression by surface biotinylation assay in HEK293 cells.

Results: In our cohort of 298 children, 86% were African American and 20% Hispanic by self−reported ancestry/ethnicity; 51% female. We identified 8 children with heterozygous rare variants (MAF < 1%, R7S, F202L (n=2), M215I, G252A, V253I, I269N, F284I) and 15 with common variants (V103I, I198=, Q156=, I251L). Three of these were novel (M215I, G252A, F284I). In an in vitro system of MC4R overexpression in HEK293 cells, the initiation of cAMP response to α−MSH required 10−fold higher concentration in M215I & G252A, and 1000−fold higher in F284I. The peak cAMP response of WT protein was not seen in the tested variants with increasing concentrations of α−MSH. Surface expression of the receptor detected on Western blot showed diminished levels for the mutants as compared to WT, with the least protein detected in G252A amongst the new variants. All of the identified variants were deemed pathogenic based on our functional studies and/or prior literature reports.

Conclusions: In our cohort of children with severe, persistent early onset obesity from underrepresented minorities, 2.7% of the children had rare, pathogenic heterozygous variants in MC4R. We established the pathogenic nature of 3 novel variants in MC4R in our cohort by in vitro functional studies.

# Board 3, Poster 1

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| **Abstract Topic Category** * | Genetics/Epigenetics/Early Determinants of Obesity |
| **Abstract Title** * | Harnessing Single Cell technology to understand the biology of Rpgrip1l on obesity. |
| **Authors** * | Sohum Gala*(1), Maria Caterina De Rosa*(1), Qi Su (2), Charles A. LeDuc (1), Rick Rausch (1), Yurong Xin (2), Jesper Gromada (2), Judith Altarejos (2), Rudolph L. Leibel (1), Claudia A. Doege (1), Vidhu Thaker (1), George Stratigopoulos (1) |
| **Institutional Affiliations For Each Author.** * | 1) Columbia University Medical Center, New York, NY, 2) Regeneron Pharmaceuticals, Tarrytown, NY |
| **Corresponding Author Email** * | gs2172@cumc.columbia.edu |

**Structured Abstract** *

Background: Previous work has shown that congenital arcuate–specific hypomorphism of Rpgrip1l, a gene adjacent to FTO obesity–associated SNPs, causes neurodevelopmental defects and obesity in mice. Congenital Rpgrip1l deletion in the anorexigenic MC4R–expressing neurons of the paraventricular hypothalamus (PVH) also results in increased adiposity in mice. We sought to use single cell RNA sequencing (SC–RNA seq) to identify the impact of Mc4r–specific Rpgrip1l deletion in the development of the PVH.

Methods: We subjected single cells isolated from PVH punches of 5–weeks old WT (n=33) and Mc4R–specific Rpgrip1l hypomorphic (MUT, n=27) C57BL/6Tac male mice to single cell sequencing utilizing a 10X Genomics Chromium platform. Cell Ranger 2.0 was used for alignment and Seurat 2.3 to perform initial clustering analysis on each of the datasets using previously published methods (PMID: 26000488). We utilized the neuronal clusters from each group to perform an integrated analysis of the two datasets to identify the common and different cell types, as well as differences in counts and cellular gene expression profiles. We used the top non–overlapping 1000 genes with highest dispersion (variance/mean) from both datasets after controlling for the known relationship between mean expression and variance. This was followed by canonical correlation analysis (CCA) based on the above genes. In a single integrated analysis on the combined neuronal cells, we performed clustering and comparison of the differences in proportion, and differential gene expression across the two conditions. Additionally, we made a group–wise comparison of the average expression profile of the genes for each cluster.

Results: After quality control, we obtained 22,138 cells (17,516 genes) from WT and 11,173 cells (17,022 genes) from MUT animals. A total of 4,454 neuronal cells (2,394 WT and 2,060 MUT) were used for CCA. We identified 17 clusters in the integrated analysis labeled by PVH–specific canonical genes (Avp/Rora, Avp, Bdnf/Trh, Cck, Cck/Avp, Oxt, Crh, Sst, Trh, Gal, Ngrn, Apoe/Gnas1, Apoe/Gnas2, Kiss1 and 3 unknown clusters). There was a significant difference in the proportion of cells seen in Avp/Rora, Avp, Cck/Avp, Apoe/Gnas1 & 2, Gal, and Kiss1 clusters between the 2 groups. The analysis of differential gene expression is ongoing.

Conclusions: Integrated analysis of SC–RNA seq of multiple cell groups is a valuable tool for understanding molecular genetic changes and effects of interventions at the cellular level.

# Board 3, Poster 2

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The Peer Assisted Lifestyle (PAL) intervention protocol: A technology-assisted weight-loss intervention within Patient Aligned Care Teams at the VA |
| **Authors** * | Cerveny, S; Hall, E; Zahur, R; Fronshtein, M; Ajenikoko, AK; Wittleder, S; Orstad, SL; Fang, Y; Jay, M |
| **Institutional Affiliations For Each Author.** * | NYU School of medicine, VA NY Harbor, New Jersey Institute of Technology |
| **Corresponding Author Email** * | melanie.jay@nyumc.org |

**Structured Abstract** *

Background/Rationale:
Veterans shoulder a disproportionate burden of obesity and its co-morbidities, and modest weight loss improves health outcomes. The Veterans Affairs (VA) New York Harbor Healthcare System offers the MOVE! program, but only 8% of eligible patients attend. However, Veterans see their primary care providers 3.6 times per year, supporting the importance of developing primary care-based interventions. The United States Preventive Services Task Force (USPSTF) recommends the use of the 5As framework (Assess, Advise, Agree, Assist, Arrange) for counseling patients about weight. Interactive behavior change technologies may facilitate delivery of the 5As in primary care. The PAL protocol was developed from a pilot RCT (Randomized Control Trial; n=45) conducted at the Manhattan VA that showed feasibility for short-term weight loss.

Methods:
Up to 520 primary care patients at the Brooklyn VA will be recruited from the 17 patient-aligned care teams (PACT) randomized into the PAL intervention or the Enhanced Usual Care (EUC) arm. The PAL intervention includes: 1) an online tool based on the 5As, 2) health coaching and goal setting with a Veteran peer coach, 3) addressing barriers to attending MOVE! and similar intensive weight management programs, and 4) telephone health coaching. Providers in the PAL arm receive training in counseling and support. Patients in the EUC arm will receive weight management handouts and information about MOVE! programs. Baseline data will be collected via surveys, chart review, basic physical measurements, and a routine blood draw. All participants will come to 6 and 12 month study visits to evaluate intermediate, behavioral, and weight outcomes. Eligibility criteria include: age 18-69 years, obese or overweight with a comorbidity (e.g., arthritis, sleep apnea, hypertension), at least one PCP visit in the past two years, and no physical, mental health, or other contraindications.

Results:
We predict patients in the PAL intervention arm will lose 2.2 kg more weight than EUC patients after 12 months of treatment (Primary Outcome) and that a higher percentage will have clinically significant weight loss ($\geq$5%). We will also evaluate clinical (e.g., reduced blood pressure and waist circumference) and behavioral (e.g., increased physical activity, improved dietary intake, and intensive weight management program engagement) outcomes.

Conclusions:
The PAL intervention utilizes the patient-aligned care team (PACT) model while linking patients to existing evidence-based programs. This RCT will determine the effectiveness of the PAL intervention in urban VA patients with obesity.

# Board 4, Poster 1

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Do goal-directed or outcome-based financial incentives promote weight loss among primary care patients with obesity? Protocol for the Financial Incentives foR Weight Reduction (FIReWoRk) RCT |
| **Authors** * | Stephanie L. Orstad, PhD; Joseph A. Ladapo, MD, PhD; Christina Hernandez, MPH; Susan Parraga, BA; Tiffany R. Martinez; Eduardo Corona, BS; Riana Liang Chen, BS; Miguel A. Cuevas, BS; Victoria Sweat, MA; Sandra Wittleder, PhD; Melanie Jay, MD, MS |
| **Institutional Affiliations For Each Author.** * | NYU School of Medicine, David Geffen School of Medicine at UCLA |
| **Corresponding Author Email** * | melanie.jay@nyumc.org |

**Structured Abstract** *

Background: Obesity is a major public health challenge and exacerbates economic disparities through employment discrimination and increased personal health expenditures. Financial incentives for weight management may intensify individuals' utilization of evidence-based behavioral strategies while addressing obesity-related economic disparities in low-income populations. Trials have focused on testing incentives contingent on achieving weight loss outcomes. However, based on social cognitive and self-determination theories, providing incentives for achieving intermediate behavioral goals may be preferable to incentivizing outcomes if they enhance an individual's skills and self-efficacy for sustaining long-term weight loss. The objective of this paper is to describe the rationale and design of the Financial Incentives foR Weight Reduction (FIReWoRk) study, a randomized controlled trial to test the comparative- and cost-effectiveness of two financial incentive strategies for weight loss (goal-directed vs. outcome-based) among low-income adults with obesity, as compared to the provision of health behavior change resources alone.

Methods: We are recruiting 795 adults, 18-70 years-old with a body mass index $\geq 30$ kg/m2, from three primary care clinics serving residents of socioeconomically disadvantaged neighborhoods in New York City and Los Angeles. All participants receive a one-year commercial weight loss program membership, self-monitoring tools (bathroom scale, food journal, and Fitbit Alta HR), health education, and monthly check-in visits. In addition to these resources, those in the two intervention groups can earn up to $750 over 6 months for 1) participating in an intensive weight management program, self-monitoring weight and diet, and meeting physical activity guidelines (goal-directed arm); or 2) a $\geq 1.5\%$ to $\geq 5\%$ reduction in baseline weight (outcome-based arm). To maximize incentive efficacy, we incorporate concepts from behavioral economics, including immediacy of payments and framing feedback to elicit regret aversion. We will use generalized mixed-effect models for repeated measures to examine intervention effects on weight at 6, 9, and 12 months.

Discussion: This study addresses an important gap in obesity research by comparing goal-directed and outcome-based incentives for weight loss. We anticipate that the results will inform the design of scalable financial incentive programs to address obesity in public and private health systems.

# Board 4, Poster 2

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Prevent Age-related Weight Gain by Daily Self-weighing in Workplace Employees |
| **Authors** * | Zhong, Y*.; Barre, L.K.; Levitsky, D.A. |
| **Institutional Affiliations For Each Author.** * | Division of Nutritional Science Sciences at Cornell University,Division of Nutritional Science Sciences at Cornell University,Division of Nutritional Science Sciences and the Department of Psychology at Cornell University |
| **Corresponding Author Email** * | dal4@cornell.edu |

**Structured Abstract** *

Background: Age-related weight gain refers to the phenomenon where adults between twenty and forty years of age gain an average of one pound per year. Our previous studies demonstrate that daily self-weighing combined with graphic feedback is effective in promoting weight maintenance among students and health club members. Our research question is whether daily weighing can prevent age-related weight gain in a more representative population: workplace employees.

Method: We initiated a two-year randomized controlled trial in summer 2017. The one-year data from the intervention group is presented. Participants were recruited and randomized into the intervention group (n=151) or no-treatment control group (n=137). Weight and height of both groups were measured at the beginning of the study and will be taken again at the end of the two-year period. The intervention group participants were provided an electronic scale and instructed to weigh themselves daily in the morning. The weight data from the scales were electronically uploaded to a server that could be accessed by the researchers. Reminder emails are sent to participants who do not weigh themselves for over three consecutive days. The weight change was calculated as the difference between the average weights of the first eight days and the last eight days of the 12-month period. The weighing frequency was defined as percent of the days the participants weighed themselves out of the 12-month period. The association between the weight change percent and the weighing frequency was explored using nonparametric correlation test.

Results: At baseline, the intervention group participants were 41.7±11.1 (mean, sd) years old, mostly female (75.5%) and White (86.8%)), with at least a college education (71.5%). The median baseline BMI was 25.7 kg/m2 (IQR=23.2 to 29.8). During the 12-month study period, 8 participants dropped out of the study (attrition=5.3%) while the 143 remaining participants had a median weighing frequency of 86.3% of the days (IQR=77.5 to 91.5%). Overall 79.7% maintained their body weight within ±5% of their original weight, 11.2% (n=16) lost 5% or more and 9.1% (n=13) gained over 5% of their original weight. The median percent weight change for the year was −0.37% (IQR= −2.49 to 2.57%), which was not significantly associated with weighing frequency (Spearman r=−0.06, p=0.5).

Conclusion: Workplace employees can adhere to a daily weighing regimen with reminders for one year. The data also suggests that daily self-weighing with graphic feedback on weight may help people resist age-related weight gain.

# Board 5, Poster 1

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| Abstract Topic Category * | Interventions and Clinical |
| Abstract Title * | Self–efficacy as a predictor of weight loss in veterans with obesity participating in medical weight management. |
| Authors * | Parziale A1,2,3, Vallejo–García F1,2,3, McCloskey K2,3, Callanan M4, Tenner C4, Alemán JO1,2,3* |
| Institutional Affiliations For Each Author. * | 1. Division of Endocrinology, Department of Medicine, Manhattan Campus of the New York Harbor VA Healthcare System; 423 East 23rd St, New York, NY 10010<br>2. Division of Endocrinology, Department of Medicine, New York University Langone Medical Center, 435 East 30th St; New York, NY 10016<br>3. Laboratory of Translational Obesity Research, New York University School of Medicine; 435 East 30th St, 6th Floor; New York, NY 10016<br>4. Department of Medicine, Manhattan Campus of the New York Harbor VA Healthcare System; 423 East 23rd St, New York, NY 10010 |
| Corresponding Author Email * | jose.aleman@nyumc.org |

## Structured Abstract *

Background: Overweight and obesity are major health burdens in veterans, occurring at higher rates than general population (78% vs 69%) and associating with chronic disease development. Self–efficacy is a person's ability to overcome the difficulties inherent in performing a specific task in a particular situation such as smoking cessation. We evaluated if a simple self–efficacy score may serve as a predictor of weight control in de–identified records of veterans with obesity undergoing a weight loss intervention consisting of lifestyle modification with pharmacotherapy through a VA weight management clinic.

Methods: We reviewed deidentified records for of 22 participants (17 males, 5 females, age range = 43–69, race/ethnicity 45% Black, 32% White, 18% Latino, and 5% Asian) in the weight management clinic at the Manhattan VA hospital. As part of the initial intake assessment, we used 2 validated questions to assessed participants' confidence in their ability to lose weight. Individuals were asked: "How ready are you to commit time, energy and resources to weight loss therapy?" and "How confident are you in your ability to lose weight?". Answers were rated from 1 (not ready or not confident) to 3 (ready or confident). Self–efficacy was scored as high (5-6), intermediate (3–4) or low (0–2).

Results: Participants were on average 56 years old with weight of 275±49 lb at baseline. Eight records were classified as having high self–efficacy, 7 had moderate self–efficacy, and 3 low self–efficacy. After 3 months of lifestyle plus pharmacotherapy intervention, weight loss in the high self–efficacy group was $8.8 \pm 4.6$ lb vs $-0.4 \pm 1.2$ lb (mean±SE, p = 0.088) in the non–high self–efficacy group. Excess body weight in high vs non–high self–efficacy groups at 3 months was $3.08\% \pm 0.02\%$ vs $-0.26\% \pm 0.01$ (p = 0.094).

Conclusions: Our data suggests that veterans with obesity and high self–efficacy may lose more weight than those with lower self–efficacy. This simple tool may serve as an important first screen for weight management clinical practice.

References:
●Diabetes Prevention Program Research, G., et al., 10–year follow–up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. Lancet, 2009. 374(9702): p. 1677–86.
●Tsai et al. Readiness Redefined: A Behavioral Task during Screening Predicted 1–Year Weight Loss in the Look AHEAD Study. Obesity (Silver Spring). 2014 April ; 22(4): 1016–1023. doi:10.1002/oby.20648.

# Board 5, Poster 2

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The protective effect of social support on the intergenerational transmission of obesity in a low–income, Hispanic sample. |
| **Authors** * | Michelle Katzow, MD MS*, Rachel Gross, MD MS, Alan Mendelsohn, MD, Lauren Thomas Berube, MS RDN, Mary Messito, MD |
| **Institutional Affiliations For Each Author.** * | NYU School of Medicine, Department of Pediatrics, NYU School of Medicine, Department of Pediatrics, NYU School of Medicine, Department of Pediatrics, NYU Steinhardt Department of Nutrition and Food Studies, NYU School of Medicine, Department of Pediatrics |
| **Corresponding Author Email** * | michelle.katzow@nyumc.org |

**Structured Abstract** *

Background: Prepregnancy overweight and obesity (OW/OB) is a strong predictor of child obesity, and is common among low–income, Hispanic women. Social support may protect against obesity in women of child–bearing age but the study of its effect on the inter–generational transmission of obesity has been limited.

Methods: We performed a longitudinal analysis of 523 low–income, Hispanic mother–child dyads in the "Starting Early" program, a primary–care based randomized–controlled obesity prevention trial. Anthropometric data were obtained by medical record review. Prepregnancy OW/OB was defined as maternal BMI ≥25kg/m2 and child BMI z–score (BMIz) was calculated based on WHO reference standards. Maternal social support was measured using a subscale of the Maternal Social Support Index, which asks, "How many people can you count on in times of need?" Responses were dichotomized as low (0–1) and high (≥2). We used multilevel linear growth curve modeling to determine the effect of prepregnancy OW/OB on child BMIz from birth through age 3 years, and if that effect was moderated by maternal social support. Analyses were adjusted for maternal age, birth country, education, child sex and group intervention status.

Results: 64% of women had prepregnancy OW/OB and 20% had low social support. Prepregnancy OW/OB status was positively associated with child BMIz (B=0.17, 95%CI 0.03, 0.31) and maternal social support was negatively associated with child BMIz from birth through age 3 years. The interaction between maternal social support and prepregnancy OW/OB was significantly and negatively related to child BMIz score (interaction term B=−0.38, 95%CI −0.75, −0.01). Maternal OW/OB status was associated with a 0.50 kg/m2 (95% CI 0.18, 0.83) increase in child mean BMIz among those with low social support and was not significantly associated with child mean BMIz among those with high social support (B=0.10, 95%CI −0.05, 0.26).

Conclusions: Maternal social support was associated with lower child BMIz score from birth through age 3 years and may be protective against the inter–generational transmission of obesity in low income, Hispanic families.

# Board 6, Poster 1

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Weight loss outcomes in patients using BMIQ: a review of patients treated at the Comprehensive Weight Control Center |
| **Authors** * | Sarah Barenbaum, Sherin Mathews, Katherine H. Saunders, Guadalupe Minero, Alpana P. Shukla |
| **Institutional Affiliations For Each Author.** * | All authors(except Guadalupe Minero): Comprehensive Weight Control Center Division of Endocrinology, Diabetes, & Metabolism Weill Cornell Medicine Guadalupe Minero: BMIQ |
| **Corresponding Author Email** * | aps2004@med.cornell.edu |

**Structured Abstract** *

Background:

Many online food and weight tracking tools have been developed to help users lose weight; however, very few have been systematically studied to ascertain their efficacy. This study was a retrospective chart review assessing the implementation and utility of BMIQ, a novel web-based weight management program offered to patients treated at the Comprehensive Weight Control Center (CWCC) at Weill Cornell Medicine. BMIQ is a customizable cloud-based platform used to deliver evidence-based behavioral management. It incorporates educational materials, meal plans, and tracking tools for patients, along with guidance for the healthcare provider to manage patients.

Methods:

All new patients seen at the CWCC between 9/1/2016- 6/1/2017 were identified by an Epic query, and the electronic medical records of patients enrolled in BMIQ were reviewed. In BMIQ, the following data were queried: total number of enrolled patients who activated their account, login rates, and use of BMIQ sessions content. Data pertaining to demographics, medical history, medication history and weight changes during 6-month follow-up were recorded by reviewing electronic medical records.

Results:

495 new patients at the CWCC were enrolled in BMIQ during the study period, of which 217 had 6-month follow-up. Of these, 64% (n=138) activated their BMIQ account and viewed ≥ 1 BMIQ session; 48% (n=66) viewed ≥ 4 BMIQ sessions. Mean age was 50, and 72% were female. The average number of MD and RD visits was 3.5 (±1.1) and 1.9 (±1.6) respectively. At 6 months the patients were taking an average of 1.6 weight-loss medications; metformin was the most commonly prescribed (76%). Mean weight loss across all 217 patients at 6 months was 7% (±6%); 59% achieved ≥5% weight loss and 28% achieved ≥ 10% weight loss. Patients who viewed ≥ 4 BMIQ sessions averaged 8.6% weight loss at 6 months; 70% reached ≥5% weight loss and 41% achieved ≥10% weight loss. Patients who did not use BMIQ or viewed <4 sessions had a mean weight loss of 6.3%; 54% achieved 5% weight loss and 22% achieved 10% total weight loss (p<0.05 for all results).

Conclusion:

Patients with limited in-clinic MD and RD follow-up who also utilized BMIQ experienced superior weight-loss outcomes. This retrospective review suggests the feasibility and utility of this unique online behavioral program as part of medical weight management at a tertiary weight management center.

# Board 6, Poster 2

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Effect of Food Order on Postprandial Glucose Excursions in Prediabetes |
| **Authors** * | Alpana P. Shukla*, Morgan Dickison, Natasha Coughlin, Ampadi Karan, Anthony Casper, Wanda Truong, Ana Emiliano, Katherine Saunders, Rekha B Kumar, Leon Igel, Louis J Aronne |
| **Institutional Affiliations For Each Author.** * | All authors: Weill Cornell Medicine |
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## Structured Abstract *

Background: It has been previously demonstrated that food order during a meal has significant impact on postprandial glucose excursions in type 2 diabetes; consuming vegetables and protein before carbohydrate attenuates post-meal glucose spikes compared to eating the same meal components in the reverse order. In this study, we sought to assess the generalizability of these findings to individuals with prediabetes using a meal with a different macronutrient ratio, and explored the glycemic impact of a third meal order of vegetables first followed by protein and carbohydrate.

Methods: We used a within-subjects crossover design in which all participants consumed isocaloric meals with exactly the same composition, on three separate days, after a 12 hour overnight fast. Each meal was consumed in 30 min, under the following experimental conditions that were randomly assigned:
1. Carbohydrate first (CF) (ciabatta bread) over 10 min, a 10 min rest interval, and then protein (grilled chicken breast) and vegetables (lettuce, tomatoes, bell peppers, red cabbage, with balsamic vinegar and olive oil) over 10 min.
2. Protein and vegetables first (PVF) over 10 min, a 10 min rest interval, and then carbohydrate over 10 min.
3. Vegetables first (VF) over 10 min, a 10 min rest interval, and then protein and carbohydrate together over 10 min.
Glucose was measured at baseline (just before meal ingestion) and at 30 min intervals up to 180 min.

Results: Baseline fasting glucose concentrations were similar in the three meal conditions. Postprandial glucose levels were significantly decreased at 30 and 60 min and the iAUC0–180 was 38.8% lower (2067.8 ± 411.7 vs 3379.5 ± 554.2 mg/dlx180min; p=0.008 min) following the PVF meal order, compared with CF, the reverse meal order. The VF meal pattern also showed reduced postprandial glucose levels at 30 and 60 min compared with CF and a decrease in iAUC0–180 of 23.4%(2588.3±621.7 vs 3379.5 ± 554.2 mg/dlX180min, p=0.205). Mean glucose concentrations were significantly lower in the CF meal condition compared to both PVF and VF at 120, 150 and 180 mins. Incremental glucose peaks were lower by 46% and 43% following the PVF and VF meals respectively compared to CF .The carbohydrate-first meal pattern demonstrated marked glycemic variability whereas glucose levels were stable in other meal conditions.

Conclusions: Food order presents a novel, simple behavioral strategy to attenuate glycemic excursions in prediabetes. Prospective studies are needed to assess its feasibility and effectiveness as a diabetes prevention tool.

# Board 7, Poster 1

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Portion creation tasks in persons undergoing bariatric surgery are both sensitive and predictive of weight loss outcome |
| **Authors** * | Jeon D. Hamm, Musya Herzog, Shoran B. Tamura, Ari Shechter, Jeanine Albu, Blandine Laferrère, Jeff M. Brunstrom, F. Xavier Pi-Sunyer, & Harry R. Kissileff |
| **Institutional Affiliations For Each Author.** * | Columbia University Medical Center, Mount Sinai–St. Luke's Hospital, University of Bristol |
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**Structured Abstract** *

Background:
Tasks that used computerized adjustment of portion sizes to assess planned food intake and expected satiety were sensitive to expectations about foods (Forde et al, 2015). We therefore expanded these tasks to assess how bariatric surgery affects expectations about foods and whether these expectations can predict weight loss outcome.

Methods:
53 patients undergoing bariatric surgery (PBS) and 31 age- and race-matched controls (CT) (BMI between 18.5 and 24.9 kg/m2) adjusted virtual portions of twelve foods varying in flavor and energy density under five theoretical eating situations. Participants were told to create portions 1) eaten typically (typical), 2) eaten to feel satisfied (satisfied), 3) representing the most that they could tolerate eating (maximum), 4) eaten if nothing was limiting them (desired), and 5) eaten to stay healthy (healthy). PBS completed the tasks and were weighed pre- and three-months post-surgery, while CT completed tasks at a baseline visit and a three-month follow-up. Differences across four factors (group, time, food, question) were tested with planned comparisons by means of Tukey's adjusted $p < .05$. Rank difference in weight loss at three months was correlated with rank baseline portions. Results are presented for the foods that generated the largest effects (grapes and potato chips).

Results:
There was a significant four-way interaction. Significant Tukey-adjusted differences among foods and situations did not change between visits in CT, but in PBS portion sizes of all foods and situations, except healthy, decreased post-operatively. Portions for desired and maximum were significantly larger than for the other three situations in CT (at both visits) and in PBS (at baseline). Portions of grapes were higher than most foods for most questions, and differed between groups and visits. Pre-operative maximum ($r2=.1033$, $p=.0190$), and desired ($r2=.1295$, $p=.0081$) portions of grapes and desired portions of potato chips ($r2=.0740$, $p=.0487$) positively predicted three-month weight loss. Additionally, pre-operative desired/healthy portion difference (desired – healthy) of grapes ($r2=0.13$, $p=0.0077$) predicted three-month weight loss.

Conclusions:
This task was not only sensitive to group, food, question, and changes due to bariatric surgery, but also predicted weight loss. Portion size tasks are effective for assessing eating expectations that predict clinical outcomes.

# Board 7, Poster 2

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The Acute Effects of Yoga on Mood and Body Connection among Participants in a Weight Loss Program |
| **Authors** * | Corinne Grady*, Richard Weil |
| **Institutional Affiliations For Each Author.** * | Institute of Human Nutrition Columbia University, Mt Sinai St Luke's Hospital Weight Loss Program |
| **Corresponding Author Email** * | cg3048@cumc.columbia.edu |

**Structured Abstract** *

Background

Obesity poses a significant physical and emotional burden on individuals and many seek to lose weight through lifestyle changes. Yoga has become increasingly popular and may provide physical and psychological benefits in individuals with obesity. However, the effects of yoga on psychological factors such as mood and body connection have not been examined in this population.

Methods

Free one-hour yoga sessions were offered to women (n=17) and men (n=2) with class 2 and 3 obesity (avg BMI =39.7) currently enrolled in the 52-week, out-patient, lifestyle-change Weight Loss Program at Mount Sinai St. Luke's Hospital. Yoga poses were modified to be accessible for participants, through variations in chairs and on the floor if tolerated. Participants completed a survey before and after each class including two items from each subscale of the Abbreviated Profile of Mood States and five items from the Scale of Body Connection, which measures body awareness and body dissociation. Participants were asked how anxious they were about taking yoga prior to the class. Hunger pre- and post-session was determined with a 5-point Likert scale from 1 to 5. Paired t-tests were used to evaluate changes pre- and post-session. Linear regression was used to determine associations between BMI, absolute scores, and changes in score.

Results

The majority of participants had taken yoga before (83%). Fifty percent were "Not at All anxious", 44% were "A little," and 6% were "Moderately" anxious about taking yoga. There was a non-significant decrease in hunger after the class from 2.16 to 1.89 (p=0.23). There were significant decreases (p<0.05) in the following mood items: angry, annoyed, confused, discouraged, exhausted, fatigued, restless, sad, tense, and uncertain. There was a significant increase in the active mood item (p=0.009), and non-significant increases in competent, confident, and energetic items. There was a non-significant increase in body awareness from 3.3 to 3.5 on the 5-point Likert scale (p=0.18) and a non-significant decrease in body dissociation from 2.2 to 1.9 (p=0.12). Pre-session body dissociation was significantly associated with BMI (R2=0.34, p=0.01). No other significant associations between body connection and BMI were found.

Conclusions

Sessions of appropriately modified yoga positively impacted mood in women and men with class 2 and 3 obesity. BMI did not predict changes in body connection after a yoga session. We recommend that women and men in this population participate in modified yoga classes. More research is needed to determine if regular yoga practice conveys greater benefit.

# Board 8, Poster 1

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The CHANGE Challenge: A Pediatric Obesity Group Intervention |
| **Authors** * | Julia Galasso2, Donna Martinez1*, Tiffany Diaz1*, Alexandra Aarons1, Monalisa Ghose1, Onjona Hossain3, Migdalia Morel1, Sandra Arevalo4, , Margaret Walsh1*, Laura Kaplan-Weisman1 |
| **Institutional Affiliations For Each Author.** * | 1. Institute for Family Health<br>2.The Ohio State University<br>3.Fordham University<br>4.Montefiore Medical Center |
| **Corresponding Author Email** * | mwalsh@institute.org |

**Structured Abstract** *

Background
Pediatric obesity is a significant public health problem in the United States. There is 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 family medicine practice can help obese children improve their BMI percentiles.

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 for 12 weeks at the Walton Family Health Center in four cohorts between September 2016–June 2018. Using a culturally sensitive, interactive curriculum, each session incorporated hands-on activities conducive to promoting healthy diet and lifestyle modifications, including nutrition education, physical fitness, and group preparation of a healthy snack.
For each cohort, the experimental group was comprised of patients who attended at least 50% of the group sessions, control group 1 of patients who attended less than 50% of sessions, and control group 2 of outreached age-matched controls who did not attend and received routine care. BMI percentile changes were calculated at the end of the group and then 6 and 12 months from the conclusion of the group from baseline BMI percentiles.

esults
Fifty unique children aged 5–12 years with a BMI greater than the 85th percentile attended one or more cohorts, with a total of 16 experimental group patients and 34 control group 1 patients. Families were actively engaged in group sessions and discussions in shared medical visits showed that they had increased knowledge about obesity and had begun dietary and lifestyle changes. Attendance was lower than desired to achieve statistical significance attendance and we allowed participation in more than one cohort and obese siblings outside the targeted age range to attend. We also had difficulty recalling patients for BMI checks at the follow-up intervals. Due to our small sample size and limited follow-up BMI percentiles, it is difficult to draw a conclusion about the impact of our intervention on decreasing BMI percentiles.

Conclusion
Though participation was low and we cannot fully assess if our intervention reduced BMI percentiles, the CHANGE Challenge has been a successful first step in addressing pediatric obesity at the IFH. Going forward, we plan to change the group schedule to improve attendance and focus on systematizing follow-up outreach.

# Board 8, Poster 2

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|---|---|
| Abstract Topic Category * | Interventions and Clinical |
| Abstract Title * | Role of Ethnicity on Weight Loss and Attrition After Bariatric surgery |
| Authors * | King R*, Levesque K, Patel K, Mark V, Shah A, McGinty J, Laferrère B |
| Institutional Affiliations For Each Author. * | Department of Medicine, Obesity Research Center, Columbia University; Department of Medicine, Obesity Research Center, Columbia University Department of Medicine, Obesity Research Center, Columbia University Department of Medicine, Obesity Research Center, Columbia University Department of Medicine, Obesity Research Center, Columbia University Bariatric Surgery Center, Englewood Hospital Department of Medicine, Obesity Research Center, Columbia University |
| Corresponding Author Email * | bbl14@columbia.edu |

### Structured Abstract *

Background: The variability of weight loss after bariatric surgery may be explained by ethnicity. Studies suggest that Hispanics (H) or African Americans (AA) lose less weight and are less likely to follow up than Caucasians (C). Our goal was to assess the role of ethnicity on weight loss and attrition rate after either Roux-en-Y gastric bypass (RYGB) or adjustable gastric banding (AGB). We hypothesized that Hispanics and African American lose less weight and have higher attrition than Caucasians, regardless of surgery type. Methods: This is a retrospective analysis of observational longitudinal data collected in 570 adults who underwent either RYGB or AGB at St. Luke's Roosevelt Hospital (NY). BMI, percent total weight loss (%TWL) and attrition rate were collected at 3, 6, and 12 months after surgery. Comparison between ethnic groups was made by ANOVA. Results: Patients were predominantly Hispanic (45% H; 32% AA; 23% C; $p<0.001$), women (86.7%), had mean BMI of 47.3 $\pm7.9$ kg/m$^2$ (H: 46.8$\pm8.0$ kg/m$^2$; AA: 48.4$\pm8.4$ kg/m$^2$; C: 46.7$\pm7.0$ kg/m$^2$; $p=0.073$); mean age was 39.8 $\pm11.6$ years, and Hispanics were younger (H:38.1 $\pm11.0$ years, AA: 41.4 $\pm11.1$ years; C: 41.1 $\pm12.8$ years; $p=0.004$). There was no effect of ethnicity on gender distribution ($p=0.774$). Hispanics were more likely to get RYGB (53% H, 30% AA, 17% C; $p<0.001$) than AGB (30% H, 37% AA, and 33% C; $p=0.520$) and, as a result, had the greatest one year %TWL (H: 29.1%$\pm12.0$, AA: 24.7%$\pm14.0$, C: 21.6%$\pm14.6$; $p<0.001$). RYGB and AGB groups did not differ in age ($p=0.171$) or gender distribution (women 85.9% in RYGB and 88.1% in AGB; $p=0.540$). RYGB had greater pre-surgery BMI (48.3$\pm8.4$ kg/m$^2$; AGB 45.2$\pm6.6$; $p<0.001$). As expected, RYGB resulted in greater one year %TWL than AGB (34.5%$\pm8.9$ vs. 13.1%$\pm8.5$; $p<0.001$). Ethnicity had no effect on %TWL after RYGB (H: 35.4%$\pm6.9$; AA: 34.1%$\pm10.2$; C: 32.3%$\pm11.0$; $p=0.263$) or AGB (H: 14.0%$\pm7.5$; AA: 12.5%$\pm7.2$; C:12.9%$\pm10.8$; $p=0.693$). The overall attrition was 38% at 1 year, significantly greater after RYGB than AGB (48% vs. 32% dropout, respectively; $p<0.001$), and not affected by ethnicity ($p=0.498$). Conclusion: Contrary to previous reports, ethnicity did not affect weight loss after either RYGB or AGB. The apparent greater weight loss amongst Hispanics was driven by the greater weight loss after RYGB, a preferred surgery for Hispanics in our cohort. Attrition at 1 year was equally high in all ethnic groups. Future research will study the role of ethnicity on potential bias of surgery selection.

# Board 9, Poster 1

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Sweet beverage inclination predicts 3-month weight loss after Roux-en-Y Gastric Bypass but not Sleeve Gastrectomy |
| **Authors** * | Shoran Tamura, Jeon D Hamm, Musya Herzog, Ari Shechter, Jeanine Albu, Blandine Laferrère, F. Xavier Pi-Sunyer, & Harry R Kissileff |
| **Institutional Affiliations For Each Author.** * | Columbia University Medical Center, Mount Sinai-St. Luke's Hospital |
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**Structured Abstract** *

Background:
Many physical and physiological changes, such as weight loss and accelerated nutrient transit, occur after Roux-en-Y gastric bypass (RYGB) and vertical sleeve gastrectomy (VSG) surgery. Gastrointestinal discomfort with or without dumping syndrome, common after consumption of sugary items, can occur after bariatric surgery (Ramadan, et al, 2016). Because postoperative gastrointestinal discomfort could change sweet taste perception and consumption of sweet liquids, our study aimed to test the hypothesis that a greater decrease in inclination (i.e. liking and wanting) to consume sweet liquids postoperatively, driven by high preoperative inclination, leads to greater weight loss in patients undergoing RYGB compared to those undergoing VSG.

Methods:
Seventeen RYGB and 38 VSG patients rated their liking (GVAS scale) (Hayes, et al, 2013) after drinking 5mL cooled (47°F) portions of eight beverages: distilled water, sucrose solutions (6.1% and 34%), chocolate beverages (chocolate shake and chocolate Ensure), and Cherry Kool-Aid either unsweetened or sweetened with aspartame to 10% and 20% sucrose equivalency, 1-2 weeks before and three months after surgical intervention, with body weight recorded at each visit. Using a cup-shape scale (Hogenkamp, et al, 2017), participants also indicated how much of the beverage they would want to drink with a horizontal line across the cup (0-935mL). For each group, three-month weight loss was regressed from preoperative liking and wanting as well as from liking and wanting differences (preoperative-postoperative). Results presented are for the sweetest beverage, 34% sucrose solution, which generated the largest effects.

Results:
RYGB patients preoperative liking positively predicted three-month weight loss ($r2=.2495$, $p=.0412$), whereas liking difference RYGB did not correlate with weight loss ($r2=.0241$, $p=.5517$). RYGB patients preoperative wanting ($r2=.4135$, $p=.0054$) and RYGB wanting difference ($r2=.3225$, $p=.0174$) positively predicted weight loss. Neither preoperative liking, wanting, nor pre/postoperative differences significantly correlated with three-month weight loss in the VSG group.

Conclusion:
The reduction in wanting of highly sweet solutions after RYGB may be due to a learned aversion to sweet beverages elicited by symptoms of gastrointestinal discomfort and/or dumping syndrome. This reduction may result in a decrease in sugar consumption, which could lead to greater weight loss. However, VSG patients also experience these symptoms, so further examination into the physiological differences between RYGB and VSG patients is needed to understand the discrepancy between their inclination for sweet beverages. Tests of liking and wanting of highly sweet beverages could be clinically useful in assessing which operation will most benefit a candidate of bariatric surgery.

# Board 9, Poster 2

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Factors in virtual portion size creation that predict weight loss after bariatric surgery |
| **Authors** * | K Ku1, JD Hamm1, S Tamura1, A Shechter1, J Albu2, B Laferrère1, J M Brunstrom3, FX Pi–Sunyer1, & HR Kissileff1 |
| **Institutional Affiliations For Each Author.** * | 1Columbia University Medical Center, 2Mount Sinai–St. Luke's Hospital, 3University of Bristol; 1150 St. Nicholas Ave. New York, NY 10032, USA; kk3242@cumc.columbia.edu |
| **Corresponding Author Email** * | kk3242@cumc.columbia.edu |

**Structured Abstract** *

Background: The size of the portion of food that healthy individuals created in a computerized task (virtual portion size creation) predicted amounts of food actually eaten. (Appetite, 59:933, 2012). Changes in food consumption correlate with weight loss. Therefore, virtual portion size creation could predict weight loss. The hypothesis was that portions of sweet foods created by patients before surgery could predict weight loss after bariatric surgery.

Methods: 53 candidates for bariatric surgery (BMI greater than 35 kg/m2) created virtual portions of eight snack foods that represented four categories (high and low energy–dense crossed with sweet and salty): apples, grapes, edamame, olives, milk chocolate, donuts, potato chips, and pretzels. The four different eating situations were: Typically eaten (typical), the most that they could tolerate eating (maximum), eaten if nothing was limiting them (desired), and eaten to stay healthy (healthy) as a contrasting condition. This task was completed 1–2 weeks pre–operatively and three months post–operatively. Weight was recorded at both visits. Principle component analysis by principal (SAS9.4) was used to create factors that grouped the portions created into combinations of foods and eating situations. Three–month weight loss was then regressed from all these factors included in a multiple regression.

Results: The combinations loaded on to three factors which explained 49% of the total variance, and two more explained 70%. Maximum and desirable portions of donuts, chips, grapes and apples positively loaded onto factor 1. Factor 2 loadings were positive for maximum, desirable, and typical portions of pretzels and healthy portions of chips, donuts and chocolate and negative for healthy portions of apples and edamame. Milk chocolate for all questions and apples for 'typical' loaded positively onto factor 3 and the two low energy–dense salty snacks for all questions except 'healthy' loaded on to factors 4 and 5. Factor 1 positively predicted weight loss but none of the other factors made a significant prediction.

Conclusions:. Optimally preferred sweetness, like that found in grapes, apples and donuts, could reflect actual consumption, reduction of which could lead to greater weight loss as consumption increased. Consequently, the factor structure of the portion size creation task can be used to identify those individuals at risk for poorer weight loss after bariatric surgery.

# Board 10, Poster 1

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| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | The Goals for Eating and Moving (GEM), a technology-assisted weight-loss intervention in primary care setting: Preliminary overview of baseline characteristics in a cluster-randomized control trial |
| **Authors** * | Velastegui, L*; Wittleder, S; Ajenikoko, AK; Grullon, R; Ngo, V; Cotumaccio, D; Bogan, E; Narala, D; Cerveny, S; Orstad, SL; Fang, Y; Rehm, C; Wylie-Rosett, J; Jay, M |
| **Institutional Affiliations For Each Author.** * | Albert Einstein College of Medicine, NYU School of Medicine, New Jersey Institute of Technology |
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**Structured Abstract** *

Introduction

Over one-third of American adults have obesity and are at increased chronic disease risk. Although Medicare reimburses physicians' 5As (Assess, Advise, Agree, Assist, Arrange) counseling patients with obesity, barriers to providing counseling include competing demands and low perceived competency. A technology-assisted, 5A-based weight management intervention to engage patients in setting behavioral goals may facilitate integration of obesity counseling in primary care. In this analysis, we include a preliminary overview of recruitment, retention and baseline data for the first 159 participants enrolled in the GEM study so far.

Method

We developed the Goals for Eating and Movement (GEM) protocol and algorithms for 5As weight management and conducted a pilot RCT (N=45) to evaluate the feasibility of implementing a technology-assisted GEM intervention. The GEM intervention includes: 1) the online GEM tool via tablet, 2) health coach counseling and goal setting, 3) addressing barriers to participating in intensive weight management programs (e.g., the Veteran's Affairs MOVE! Program, Telephone Lifestyle Coaching and the Diabetes Prevention Program), and 4) follow-up telephone coaching.

In a cluster-randomized control trial, we are evaluating the GEM intervention in two NYC healthcare systems (the VA New York Harbor Healthcare System Manhattan Campus) and Montefiore Health System Medical Groups (4 Bronx, NY sites) among both English and Spanish-speaking patients. Primary care (PC) teams were randomized (19 teams, 59 providers) to the GEM intervention or EUC (enhanced usual care). Providers in the GEM arm receive training in brief counseling and support. Patients (N=512) are recruited via mail and phone calls. Eligibility criteria include: age 18 - 69 years, obese or overweight with a comorbidity (e.g., arthritis, sleep apnea or hypertension), PC physician visit in the last two years, and no physical/mental health or other contraindications.

Results

We hypothesize that GEM patients will lose 2.2 kg more than EUC patients and that a higher percentage will have clinically significant weight loss (>5%). We will also examine whether the intervention improves clinical outcomes (e.g., blood pressure and waist circumference), improves behavioral outcomes (e.g., physical activity and dietary intake), and increases engagement in intensive lifestyle programs. We present a preliminary overview of recruitment, retention, and baseline characteristics of enrolled patients (N=159) to indicate the effectiveness of our randomization strategy.

Conclusion

The GEM intervention utilizes the patient-centered medical home (PCMH) model of care while linking patients to existing evidence-based programs. This RCT will determine the effectiveness of the GEM intervention in a diverse patient population.

# Board 10, Poster 2

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|---|---|
| **Abstract Topic Category** * | Interventions and Clinical |
| **Abstract Title** * | Functional Magnetic Resonance Imaging (fMRI) and Meal–Related Hormone Concentrations Pre and Post Roux–en–Y gastric Bypass (RYGB) Surgery in Individuals with and without baseline Binge Eating (BE) |
| **Authors** * | Shaunte Baboumian* <br> Samia Mirza <br> Samuel Tweardy <br> Allan Geliebter |
| **Institutional Affiliations For Each Author.** * | Mount Sinai St. Luke's Hospital, and Department of Psychiatry at Mount Sinai Icahn School of Medicine, NY, NY <br> Touro College and University System, NY, NY <br> Mount Sinai St. Luke's Hospital, and Department of Psychiatry at Mount Sinai Icahn School of Medicine, NY, NY <br> Mount Sinai St. Luke's Hospital, and Department of Psychiatry at Mount Sinai Icahn School of Medicine, NY, NY |
| **Corresponding Author Email** * | shaunte.baboumian@mountsinai.org |

**Structured Abstract** *

Background: Although recently recognized by the DSM–5, the biological basis of binge eating disorder (BED) is not well studied. Similarly, the mechanisms underlying bariatric surgery are not well understood. fMRI is a tool to study neural activation in response to stimuli. We used fMRI and measured meal–related hormones pre and post RYGB to investigate those with baseline binge eating (BE) vs those without (NB).

Methods: In a prospective cohort study of females with obesity, 15 RYGB surgery (S): 8 BE, 7 NB and 13 non–treatment (NT): 7 BE, 6 NB underwent brain scans pre and 4–mo post–surgery, while viewing high and low energy–dense (HED and LED) food (F) and non–food (nF) visual stimuli. BE and NB subjects were similar in age, body mass index (BMI), and % body fat. Regions of interest (ROI) fMRI analyses used peak coordinates for nine regions reported by our group in Ochner et al. (2011).

Results: Post–surgical wt loss was similar for the BE groups, and those with BE no longer engaged in binge eating. At baseline and post–surgery in response to F > nF, BE (vs. NB) showed greater precuneus and thalamic activation post–surgery, all p's < .01. At post–surgery BE (vs. NB) had less middle occipital gyrus (p < .001) and inferior frontal gyrus activation (p < .004) to HED vs. LED cues. Overall S (vs. NT) had a greater reduction in dorsomedial prefrontal cortex (dmPFC) activation to HED and had increased activation to LED (p < .006). At 4 mo, BE (vs. NB) had greater thalamic activation in response to F vs nF cues (p < .014). Postprandial plasma concentrations of GLP–1 and leptin increased post–surgery (p = 0.036, p = 0.035, respectively). Post–surgery, those with BE had higher GLP–1 concentrations vs NB (p = 0.042).

Conclusion: Post–surgically, those with BE appear to pay less attention to HED vs. LED food cues and require less inhibition to control their eating behavior. Post–surgically, F vs nF stimuli continued to be more salient and relevant for BE vs. NB, whereas HED vs. LED food cues remained equally salient and relevant for BE and NB. While both GLP–1 and leptin concentrations increased post–surgically, the effect was greater for BE, suggesting greater increases in satiety. The findings suggest that the neural BE phenotype persists post–surgery, even after losing BE status.

# Board 11, Poster 1

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Regulation of ZNF638 expression in response to thermogenic signal in brown adipocytes is mediated by cAMP response element binding protein (CREB) |
| **Authors** * | Luce Perie*, Narendra Verma & Elisabetta Mueller |
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| **Corresponding Author Email** * | elisabetta.mueller@nyumc.org |

**Structured Abstract** *

Background: Zinc fingers proteins are a large family of transcription factors implicated in diverse processes including development and differentiation. A number of them, including Prmd16 and Zfp516, have been recently shown to play major roles in brown fat tissues by regulating differentiation, thermogenesis and energy expenditure. We have previously shown that the zinc finger protein ZNF638 is an early regulator of adipogenesis, in vitro, acting as transcriptional coactivator. Methods: We performed RNA and protein analyses to assess ZNF638 expression in fat depots such as brown adipose tissue (BAT) and subcutaneal (scWAT) and epididymal (eWAT) white adipose tissue isolated from male mice at 8 weeks of age. In brown adipocytes, we investigated in vitro whether ZNF638 is induced by stimuli that regulate brown fat functionality such as inducers of cAMP via forskolin treatment. To determine whether cAMP response element binding protein (CREB) regulates ZNF638 at the transcriptional level, we performed a preliminary in silico analysis of the ZNF638 promoter. We also investigated by chromatin immunoprecipitation (ChIP) assays if CREB is present in the ZNF638 promoter. To determine if CREB is necessary and sufficient to activate the ZNF638 promoter in response to forskolin treatment, we cloned 1kb of ZNF638 promoter containing the CRE elements identified and performed luciferase assays. Results: Here we present new data demonstrating that ZNF638 is highly expressed in vivo in thermogenic tissues, selectively expressed in fully differentiated brown adipocytes and that its RNA levels are elevated in response to increased cAMP levels. Detailed in silico analysis of the ZNF638 promoter revealed the presence of CRE responsive elements, and ChIP assays demonstrated that the transcription factor CREB binds to the promoter of ZNF638. Functional assays using a luciferase promoter reporter demonstrated that CREB is required for ZNF638 expression in response to forskolin treatment. Conclusion: Taken together, these results suggest that ZNF638 is highly expressed in thermogenic tissues in vivo and activated in brown adipose tissues in response to classic pro-thermogenic signals such as those activating cAMP pathways.

# Board 11, Poster 2

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Allograft Inflammatory Factor-1 suppresses thermogenesis by limiting norepinephrine bioavailability and promotes high fat diet-induced obesity and diabetes |
| **Authors** * | Chinnasamy P 1*, Srinivasan A 1, Casimiro I 1, Riascos-Bernal DF 1, Tarabra E 3, Zheng W 2, Zong H 3, Jayakumar S 1, Singh R 3, Pessin JE 3, Sibinga NES 1. |
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**Structured Abstract** *

The burgeoning epidemics of obesity and associated metabolic syndromes have major negative effects on the well-being of populations in the US and around the world, so understanding new pathways that contribute to the development of these conditions is a public health imperative. Interventions that augment energy expenditure by enhancing β3-adrenergic receptor (AR) signaling on brown and beige adipocytes have therapeutic potential to limit obesity and associated glucose intolerance and insulin resistance. Recent reports link a specific population of CD45+ F4/80+ sympathetic neuron-associated macrophages in white adipose tissue (WAT) to uptake and degradation of norepinephrine (NE), while levels of Cx3CR1+ MHCII+ macrophages in brown adipose tissue (BAT) correlate with reduced sympathetic innervation and affect NE levels and signaling; these findings suggest that distinct macrophage populations in adipose tissue affect energy utilization.

Interestingly, human population studies link sequence variants at the allograft inflammatory factor-1 (AIF1) locus to obesity, adipose inflammation, and diabetes. To test potential causal connections suggested by these findings, we inactivated the Aif1 locus in mice. Remarkably, deficiency of AIF1 in mice confers resistance to diet-induced obesity, restrains white adipose depot expansion and inflammation, decreases lipid content of brown adipose tissues, reduces hepatosteatosis, and improves glucose handling. These protections reflect elevated core energy expenditure, with increased expression of thermogenesis-related genes and increased NE levels in brown and white adipose tissues. They also correlate with increased beiging of white adipose tissue, plus enhanced β3-AR signaling and higher oxygen consumption in brown adipose tissues. In mechanistic terms, these last effects were reversible by β-AR antagonist, and we observed that AIF1 promotes polarization of a specific macrophage (CD45+ F4/80hi Ly6Chi MHCIIhi CX3CR1lo) subset and supports expression of bioamine degradation pathway genes, including monoamine oxidase-A, in bone marrow derived macrophages and in brown and white adipose tissues and their stromal vascular fractions. Together, our findings suggest that AIF1-mediated polarization of specific macrophage subsets might be critical for NE uptake and/or degradation in adipose tissues, which in turn reduces energy expenditure and promotes obesity and insulin resistance.

# Board 12, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Retinol as a regulator of energy homeostasis during embryogenesis |
| **Authors** * | Youn-Kyung Kim, Elena Giordano, Ulrich Hammerling, and Loredana Quadro |
| **Institutional Affiliations For Each Author.** * | Department of Food Science and Rutgers Center for Lipid Research, Rutgers University, New Brunswick, NJ 08901, USA |
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**Structured Abstract** *

Vitamin A is an essential regulator of mammalian embryogenesis, mainly owing to the transcriptional regulatory action of its active form, retinoic acid. Retinol (vitamin A alcohol) instead has long been thought to lack essential biological activities and serve exclusively as a precursor of bioactive vitamin A forms. However, evidence exists in the literature indicating that retinol per se, not retinoic acid, also regulates mammalian development, even though the mechanism(s) underlying this function has never been clarified[1,2]. We identified a novel protein kinase Cδ (PKCδ) signaling pathway that requires retinol (vitamin A) as a cofactor and is involved in the regulation of fuel utilization in mitochondria. The mitochondria-localized PKCδ forms a signaling complex (PKCδ signalosome) with the adapter p66Shc, cytochrome c and retinol that stimulates the conversion of pyruvate to acetyl-coenzyme A by the pyruvate dehydrogenase complex (PDHC) and regulates glucose flux, ultimately modulating mitochondria respiration and ATP production[3,4].

To investigate the potential role of the PKCδ signalosome in maintaining energy homeostasis during embryogenesis, we generated a new mouse strain in which only the retinol-binding site of PKCδ was mutated (PKCδki-/-). PKCδki-/- mice display early embryonic lethality. Specifically, a severely reduced Mendelian ratio of PKCδki-/- mice from PKCδki+/- crosses was observed after gestational day 10. This finding strongly supports the involvement of retinol and PKCδ as regulator of mitochondrial energy metabolism, given the high-energy requirement during organogenesis and the dependence of the developing embryo on oxidative phosphorylation upon establishment of the placenta (i.e. around day 9-10 of gestation).

Seahorse analysis of mouse embryo fibroblast (MEF) derived from PKCδki+/+ and PKCδki+/- embryos showed that the respiration rates of PKCδki+/- MEF cells were reduced compared to the PKCδki+/+ MEFs. This reduction was not due to the mitochondrial numerical deficiency. Moreover, acute production of reactive oxygen species (ROS) measured by fluorescence spectroscopy was attenuated in PKCδki+/- MEF compare to WT cells. Overall, our preliminary findings indicate that inactivation of the PKCδ retinol-binding site compromises mitochondrial energy metabolism, already in heterozygosity. They support our hypothesis that retinol and PKCδ may have an important role during embryogenesis in regulating respiration and ATP production.

# Board 12, Poster 2

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Intestinal fat sensing for satiety and satiation through 12-hydroxylated bile acids |
| **Authors** * | Sei Higuchi1*, Chen Zhao1, Tiara R. Ahmad1, Enrico Bertaggia1, Kristian K. Jensen2, Liangsu Wang2, Nicholas V. DiPatrizio3, Rebecca A. 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<br>3. Division of Biomedical Sciences, School of Medicine, University of California, Riverside |
| **Corresponding Author Email** * | rah2130@cumc.columbia.edu |

**Structured Abstract** *

Background: Excessive food intake is an important risk factor for obesity. Feeding behavior is governed partly by lipid mediators in the gastrointestinal tract, which regulate satiation and satiety. Bile acids (BAs) regulate the absorption, localization, and metabolism of dietary lipid in the intestine, but their effects on lipid-regulated satiation and satiety are completely unknown. We focused on a subset of BAs- the 12-hydroxylated BAs (12-OH BAs) – which are positively correlated with BMI in humans. We now show that deficiency of the essential enzyme to produce 12-OH BAs, Cyp8b1, reduces food intake and body weight in mice, and this effect is dependent on intestinal fat. We tested two hypotheses: 12-OH BAs affect food intake by (1) regulating production of the bioactive lipid oleoylethanolamide (OEA) , which enhances satiety (increasing inter-meal interval); or (2) regulating the quantity and localization of hydrolyzed fat in distal small intestine, which slows gastric emptying and induces satiation (reducing meal size).
Methods and Results: To determine the direct effects of 12-OH BAs, we used Cyp8b1 null mice. These mice showed low OEA in jejunum after refeeding. Because low OEA is expected to reduce satiety, these data do not support a role for this pathway in the hypophagia of Cyp8b1-/- mice. We evaluated liquid-phase and solid-phase gastric emptying, and both tests showed that Cyp8b1 deficiency decreased gastric emptying. Furthermore, dietary fat was necessary and sufficient for this phenotype. We hypothesized that this effect was mediated by the the G protein-coupled receptor GPR119, which is enriched in enteroendocrine cells of the ileum and colon. Concomitant knockout of GPR119 normalized the slow gastric emptying, reduced food intake and low body weight of Cyp8b1 deficient mice.
Conclusions: These data demonstrate that eliminating 12-OH BAs slows gastric emptying and induces satiation by allowing hydrolyzed fats to access the distal small intestine, where they activate GPR119. This suggests that enhanced satiation signaling by GPR119 overrides OEA signaling in food intake suppression.

# Board 13, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | A Novel Therapeutic Target for Obesity and Metabolic Impairment: Regulation of Advanced Glycation Endproduct (AGE) Signaling Via the RAGE/DIAPH1 Axis. |
| **Authors** * | Henry H. Ruiz*, Juan Francisco Aranda, Carmen Hurtado del Pozo, Akash Gujral, Joseph Boroda, Ann Marie Schmidt |
| **Institutional Affiliations For Each Author.** * | 1New York University Langone Medical Center, Diabetes Research Program |
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**Structured Abstract** *

Background: The epidemic of obesity continues unabated; significant consequences of obesity include the development of insulin resistance (IR) and type 2 diabetes (T2D) and its complications, which increase morbidity and mortality, and lead to significant reduction in quality of life. Advanced glycation endproducts (AGEs) are the products of non–enzymatic binding of sugars and/or lipids to proteins. AGEs form highly stable molecules, which are elevated in the circulation and accumulate in key metabolic tissues during obesity, insulin resistance and diabetes. Highly processed foods (e.g. Western diets) are a major source of exogenous AGEs, which may be a contributing factor to the increasing incidence of obesity and IR. Notably, restriction of dietary AGE intake improves IR and overall metabolic health in obese and diabetic patients. We have previously demonstrated that deletion of Ager (gene encoding the receptor for AGEs, RAGE) is protective from diet–induced adipose tissue inflammation, obesity (DIO) and insulin resistance. Our laboratory found that AGE–RAGE signaling requires the interaction of the RAGE cytosolic tail with diaphanous 1 (DIAPH1). Diaph1 is a member of the formin family which plays important roles in immune cell motility, cytoskeletal remodeling, phagocytosis and Rho GTPase signal transduction. Since lack of RAGE was protective from DIO and metabolic impairment, we reasoned that 1) AGE signaling via the RAGE/DIAPH1 axis can alter insulin action independent of obesity and that 2) DIO is, at least in part, driven by the overactivation of the RAGE/DIAPH1 signaling axis due to the excessive intake of AGEs from Western diets. Methods: To test these hypotheses, we administered three daily intraperitoneal doses of 10mg/kg carboxymethyl lysine (CML–AGE), a prototypical AGE, to lean, healthy wild type (Wt) mice and assessed glucose and insulin tolerance. We then fed Wt and global Diaph1 knock out (gD1KO) mice either a standard chow or a high fat diet (60% kcal/fat) for 12 weeks to induce metabolic impairment. Results: Our data suggest that acute exposure to AGEs decreases insulin sensitivity and impairs the ability to effectively clear glucose from the circulation without effects on body mass, and for the first time, we report that Diaph1 is required for the development of DIO and its associated metabolic sequelae. Conclusion: These findings are of significant translational relevance in light of our laboratory's current efforts to develop a small molecule inhibitor of the RAGE/DIAPH1 axis which may represent a therapeutic approach to treat obesity and IR.

# Board 13, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Overfeeding-induced obesity to uncover novel determinants of β cell mass adaptation |
| **Authors** * | Alberto Bartolomé*, Yann Ravussin, Molly Gallop, Anthony W. Ferrante Jr., Utpal B.Pajvani |
| **Institutional Affiliations For Each Author.** * | Department of Medicine – Columbia University Medical Center (all authors) |
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**Structured Abstract** *

Background
Both physiologic (pregnancy, aging) or pathologic (obesity, insulin resistance) stimuli increase overall insulin demand. β cell mass must expand accordingly in order to maintain euglycemia – failure to do so leads to overt Type 2 Diabetes (T2D). But despite decades of study, molecular mechanism(s) that regulate the adaptive response of β cell mass remain elusive. As β cells have limited proliferative capacity, we hypothesized that diet-induced obesity may not induce robust and/or simultaneous β cell proliferative response to uncover these mechanisms, knowledge of which could yield novel T2D therapeutic targets.

Methods
We utilized a novel model of overfeeding-induced obesity (OIO) by forced intra-gastric delivery of a high-calorie liquid diet in wildtype mice. Control mice received intra-gastric saline infusion. With OIO, mice gain substantial (~50%) body weight, with weight gain over 2 weeks comparable to that observed with 24-week ad libitum high-fat diet (HFD) feeding. This model was used to study compensatory β cell hyperplasia using transcriptomic and imaging techniques.

Results
In 2 weeks of OIO, β cell mass was increased by 50% as compared with saline-infused controls, matching the cell expansion observed after or 24 weeks HFD feeding. With the acuity of the stimulus, the number of proliferating β cells was 6-fold enriched in the OIO model, compared to either HFD-fed or saline-infused controls. The magnitude of β cell proliferative response prompted a transcriptomic profile of OIO islets, which revealed increased expression of gene components of pathways previously associated with β cell compensatory hyperplasia, such as the circadian pathway and Foxm1 targets. But as hypothesized, OIO islets showed a unique "cell proliferation" signature in comparison with previously published transcriptomic data of other β cell mass compensation models, including profound gene expression changes of cell replication machinery components, as well as an array of other genes not thought to regulate cell proliferation or β cell biology.

Conclusion
OIO is an excellent model to induce rapid β cell adaptive proliferation, and presents a unique platform for the discovery of new factors involved in β cell mass adaptation.

# Board 14, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Enhanced mitochondrial DNA repair confers protection against obesity and metabolic syndrome by altering white adipose tissue energetics |
| **Authors** * | Sai Santosh Babu Komakula*1,2, Jana Tumova1, Hong Ye1, Vladimir Vartanian3, Agnieszka Dobrzyn2, R. Stephen Lloyd3, Deeptha Kumaraswamy1, Harini Sampath1,4. |
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| **Corresponding Author Email** * | harini.sampath@rutgers.edu |

**Structured Abstract** *

Background:
Oxidative lesions in mitochondrial DNA (mtDNA) are repaired via the base excision repair (BER) pathway. BER is initiated by DNA glycosylases such as 8-oxoguanine DNA glycosylase (OGG1), which is localized to the nucleus and the mitochondria. We have reported that mice deficient for both the nuclear and mitochondrial forms of OGG1 are prone to diet-induced obesity and metabolic syndrome, indicating a role for this enzyme in the maintenance of cellular energy status. In the present study, we are trying to understand the role of mitochondrial OGG1 in the whole-body energy homeostasis.

Methods:
We have used the novel transgenic animals with targeted overexpression of only mitochondrial OGG1 (Ogg1Tg mice) and fed them with high fat diet for 12 weeks. We have analyzed these mice for the changes in the baby composition, respiration, markers of metabolic health and mitochondrial function.

Results:
We report here that mitochondrial OGG1 alone is sufficient to significantly protect animals from high-fat diet-induced obesity, insulin resistance, fatty liver, and adipose tissue inflammation. These favorable metabolic phenotypes are mediated by an increase in whole body energy expenditure that is in turn driven by specific metabolic adaptations in white adipose tissue. Adipose tissue from Ogg1Tg mice display marked increase in the levels of PGC-1α and Sirtuin 1 and significantly increased basal oxygen consumption and mitochondrial uncoupling. Additionally, markers of mitochondrial fusion, including Mitofusin-2, are significantly increased in adipose tissue from Ogg1Tg animals. Electron microscopy studies further confirm that mitochondria from adipose tissue of Ogg1Tg mice are longer and more electron dense than mitochondria of age- and diet-matched wild-type counterparts.

Conclusions:
These data indicate a novel and critical role for mtDNA repair in modulating energy homeostasis by enhancing adipose tissue mitochondrial respiration. These findings also underscore the importance of white adipose tissue mitochondrial structure and function to whole body energy balance.

# Board 14, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Phenolic-enriched raspberry fruit extract (Rubus idaeus) reduces body weight gain, adiposity, and decreases ghrelin in male mice fed a high-fat diet |
| **Authors** * | Dushyant Kshatriya, Xinyi Li, Gina M. Giunta, and Nicholas T. Bello |
| **Institutional Affiliations For Each Author.** * | Nutritional Sciences Graduate Program Rutgers University, Nutritional Sciences Graduate Program Rutgers University, Department of Animal Sciences Rutgers University, Department of Animal Sciences Rutgers University |
| **Corresponding Author Email** * | ntbello@rutgers.edu |

**Structured Abstract** *

Background: Red raspberries (Rubus idaeus) contain numerous phenolic compounds with purported health benefits. One naturally occurring phenolic, raspberry ketone (4-(4-hydroxyphenyl)-2-butanone), is the primary flavor component of red raspberries. The objective of this study was to compare the metabolic signatures associated with a phenolic-enriched raspberry extract and raspberry ketone in preventing high-fat diet-induced weight gain.

Methods: Male C57BL/6 mice (8 weeks old) received a daily oral dose of vehicle (Veh; 50% propylene glycol, 40% water, and 10% DMSO), raspberry extract low (REL; 0.2 g/kg), raspberry extract high (REH; 2 g/kg) or raspberry ketone (RK; 0.2 g/kg). Coincident with daily dosing, mice were placed on a high-fat diet (45% fat kcal).

Results: After 4 weeks, REH and RK reduced body weight gain (approximately 5-9%) and white adipose mass (approximately 20%) compared with Veh. There were also reductions (approximately 20-25%) in retroperitoneal, epidydimal, and inguinal fat mass in the RK and REH compared with REL and Veh groups. Terminal plasma ghrelin levels were lower (approximately 45%) in REL, REH, and RK compared with Veh, whereas corticosterone and insulin levels were elevated in the REL compared with Veh. Indirect calorimetry indicated that respiratory exchange ratio (RER; $v.CO_2/v.O_2$) was lower (approximately 5%), suggesting an increase in fat oxidation, in REL and RK compared with Veh. REH treatment increased total ambulatory behavior. Overall, the changes in RER were related to the differences in lean mass between groups. There were not any treatment differences in cumulative intake or in meal patterns.

Conclusion: Raspberry ketone and phenolic-enriched extracts of red raspberries have the potential to prevent diet-induced weight gain by altering metabolism.

# Board 15, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | "Reuniting Overnutrition and Undernutrition, Macronutrients and Micronutrients" |
| **Authors** * | Syed F. Mehdi, Miji Kim, Sun K. Yoo, Ramchandani Santosh, Zanali Razvi, Barbara Lowell, Jesse Roth. |
| **Institutional Affiliations For Each Author.** * | Laboratory of Diabetes and Diabetes-Related Disorders, The Feinstein Institute for Medical Research, Northwell Health, Manhasset, New York. |
| **Corresponding Author Email** * | drfaizee83@gmail.com |

**Structured Abstract** *

Over-nutrition and its late consequences are a dominant theme in medicine today. In addition to the health hazards brought on by over-nutrition, the medical community has recently accumulated a roster of health benefits with obesity, grouped under "obesity paradox".Throughout the world and throughout history until the 20th century,undernutrition was a dominant evolutionary force. Under-nutrition brings with it a mix of benefits and detriments that are opposite to and continuous with those of over-nutrition. This continuum yields J-shaped or U-shaped curves relating body mass index to mortality. The overweight have an elevated risk of dying in middle age of degenerative diseases while the underweight are at increased risk of premature death from infectious conditions. Micronutrient deficiencies, major concerns of nutritional science in the 20th century, are being neglected. This "hidden hunger" is now surprisingly prevalent in all weight groups, even among the overweight. Because micronutrient replacement is safe, inexpensive, and predictably effective, it is now an exceptionally attractive target for therapy across the spectrum of weight and age.

# Board 15, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Effect of Food Order on Ghrelin Suppression |
| **Authors** * | Alpana P Shukla*, Elizabeth Mauer, Leon I Igel, Wanda Truong, Anthony Casper, Rekha B Kumar, Katherine H Saunders, Louis J Aronne |
| **Institutional Affiliations For Each Author.** * | All authors: Weill Cornell Medicine |
| **Corresponding Author Email** * | aps2004@med.cornell.edu |

**Structured Abstract** *

Background: Data suggest that the temporal sequence of carbohydrate ingestion during a meal has a significant impact on postprandial glucose , insulin, and GLP-1 excursions in type 2 diabetes (T2DM), while the effects on ghrelin suppression and satiety have not been reported.

Methods: Using a crossover design, 16 subjects with overweight/obesity and metformin-treated T2DM were assigned to consume the same meal on 3 days in random order:

• Carbohydrate-first: carbohydrate (bread and orange juice), followed 10 minutes later by protein (chicken) and vegetables

• Carbohydrate-last: protein and vegetables, followed 10 minutes later by carbohydrate

• Sandwich: all meal components together, each half consumed over 10 min with a 10 min interval in between

Blood was sampled for glucose and total ghrelin measurements at baseline (just before meal ingestion) and at 30 min intervals up to 180 min. Participants rated their hunger and fullness levels using a visual analog scale (VAS) at the same time points.

Results: Baseline glucose and ghrelin concentrations, as well as hunger and satiety scores were similar in the three meal conditions. At 180 min, ghrelin levels remained suppressed following the carbohydrate-last meal order, while the carbohydrate-first meal led to a rebound in ghrelin to pre-prandial levels (% ghrelin change from baseline to 180 min: $-11.45 \pm 3.86$ % vs $4.13 \pm 4.38$ %; p=0.003). Decremental areas under the curve (dAUC 0-180) were similar in the three meal conditions. There was an inverse correlation between % change in ghrelin and % change in glucose from baseline when assessing all participants in the three meal conditions at the evaluated time points (r=-0.204; p < 0.001). We did not observe a significant effect of food order on subjective VAS appetite measures.

Conclusions: To our knowledge, this is the first study to demonstrate that manipulation of macronutrient order can impact ghrelin excursions. Further study with extended observation period, assessment of other gut hormones using meals with different macronutrient composition, and more objective measures of satiety is needed to assess the clinical implications of these findings.

# Board 16, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Unacylated ghrelin inhibits the growth of breast cancer cells via effects on MAPK signaling |
| **Authors** * | Au, CC.*1,2, Britt, K3, Ladumor, H1, Gerard, C2, Callaghan, B4, Cain, JE2, Inghirami, G5, Furness, JB4, Brown, KA1,2 |
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| **Corresponding Author Email** * | kab2060@med.cornell.edu |

**Structured Abstract** *

Background:

Numerous obesity–associated factors, including estrogens, glucose and insulin, have been shown to stimulate tumor cell proliferation in vitro and in vivo (Cohen et al., 2012). A gut–derived peptide hormone, unacylated ghrelin (UAG), has been shown to be involved in restoring energy homoeostasis, improving insulin sensitivity, reducing fasting glucose (Benso et al., 2012, Delhanty et al., 2010, Delhanty et al.,2013), as well as suppressing inflammation and estrogen biosynthesis (Docanto et al., 2014, Au et al., 2016). The cyclic UAG analog, AZP531, is currently progressing into type II diabetes clinical trials (Allas et al., 2016, Granata et al., 2012). We recently discovered that UAG, hypothesized to act at a yet unidentified G protein–coupled receptor (GPCR), inhibits hormone–dependent breast cancer cell growth. The current study aimed to characterize the breadth of breast cancer subtypes that are responsive to UAG/AZP531 and to characterize the mechanism of action.

Methods:
For in vitro assays, luminal A, luminal B, HER2+, TNBCs, endocrine therapy–resistant cells, and patient–derived breast cancer cells, were cultured in 3D using matrigel. Effects on breast cancer cell growth were quantified by measuring EdU incorporation and cell number. Effects on second messenger systems (cAMP, $Ca^{2+}$) was measured and involvement of G$\square$i was confirmed in assays using pertussis toxin (PTX). Western blot analysis was used to characterize effects on cell cycle, apoptosis and downstream signaling. Effects of UAG and AZP531 on breast cancer cell growth in vivo were characterized in xenografted balb/c nude mice and a syngeneic breast cancer model.

Results:

In vitro, UAG significantly inhibited the growth of all breast cancer subtypes, except those carrying RAS/RAF mutations. Resistance was further confirmed in BRAF–transfected MCF7 cells, and KRAS– and BRAF–mutated and wild–type colon cancer cells. Effects were shown to be mediated via inhibition of cell cycle. UAG had no effect on the release of intracellular $Ca^{2+}$, but inhibited the forskolin-stimulated production of cAMP. This effect was prevented in the presence of G$\alpha$i inhibitor, PTX, suggesting that UAG mediates its effects via a G$\alpha$i–coupled GPCR. UAG inhibits the phosphorylation of ERK1/2 and downstream MAPK targets. In vivo, UAG or AZP531 suppressed tumor growth compared with vehicle control.

Conclusion:

Our findings provide evidence for the potential of UAG and its analogs as novel breast/colorectal cancer therapeutics and suggest that patients likely to benefit will have tumors wild type for RAS/RAF.

# Board 16, Poster 2

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|---|---|
| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | A lipase–independent pathway of lipid release and immune modulation by adipocytes |
| **Authors** * | Stephen E Flaherty III*, Ambar Grijalva, Xaioyuan Xu, Eleanore Ables, Alireza Nomani, Anthony Ferrante Jr. |
| **Institutional Affiliations For Each Author.** * | Columbia University, Columbia University, Columbia University, Columbia University, Rutgers University, Columbia University |
| **Corresponding Author Email** * | awf7@columbia.edu |

**Structured Abstract** *

BACKGROUND:
To meet systemic metabolic needs, adipocytes release fatty acids and glycerol through the action of neutral lipases. Lipids are also key local regulators of immune function. During obesity, triglycerides accumulate in adipose tissue macrophages; this accumulation is associated with systemic metabolic complications, including insulin resistance and hepatic steatosis.

METHODS:
Bone-Marrow-Derived Macrophages (BMDMs) were co-cultured with murine adipose tissue with a genetic deficiency of adipocyte Atgl or with the presence of lipase inhibitors. These cells and tissues were analyzed for lipid content using confocal microscopy and commercial assays.
Exosomes from adipose tissue were isolated and characterized using electron microscopy, mass spectroscopy, nanoparticle tracking analysis, western blot, quantitative PCR, and lipid content assays. Exosomes were labeled with PKH26, or collected from animals with tdTomato-expressing adipocytes, and added to BMDMs or injected into mouse fat pads. These tissues and cells were examined using confocal microscopy, western blot, flow cytometry, and quantitative PCR.

RESULTS:
BMDMs cultured with lipase-deficient adipose tissue (as compared to WT) accumulate similar levels of neutral lipid, despite a marked decrease in free fatty acid release from these tissues. Adipocytes release exosome-sized vesicles (AdExos) that contain high levels of neutral lipid. Adipose tissue from lean animals releases ~ 1% of their lipid content per day via exosomes; this rate more than doubles with obesity. AdExos are taken up almost exclusively by local macrophages. AdExos are sufficient to induce the lipid-loading seen in BMDMs co-cultured with adipose tissue. Unexpectedly AdExos and associated factors are sufficient to foster differentiation of bone marrow precursors into adipose tissue macrophage-like cells by inducing multinucleation, increased lysosomal program, increased ATM-associated gene expression, and increased lipid content.

CONCLUSION:
Our findings suggest both a novel pathway of local lipid release and a mechanism by which parenchymal cells can modulate tissue resident macrophage differentiation and function.

# Board 17, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Hepatocyte-specific depletion of nuclear membrane proteins LAP1 and torsinA causes abnormal lipid droplet accumulation and nonalcoholic fatty liver disease in chow fed mice |
| **Authors** * | Ji-Yeon Shin*1,2, Antonio Hernandez-Ono1, Tatyana Fedotova1, Michael J. Lee2, Cecilia Östlund1,2, William T. Dauer3, Henry N. Ginsberg1 and Howard J. Worman1,2 |
| **Institutional Affiliations For Each Author.** * | 1Department of Medicine, 2Department of Pathology and Cell Biology, College of Physicians & Surgeons, Columbia University, New York, NY 10032, USA,3Department of Cell and Developmental Biology and Neurology, University of Michigan Medical School, Ann Arbor, MI 48109, USA |
| **Corresponding Author Email** * | hjw14@cumc.columbia.edu |

**Structured Abstract** *

Background: A major complication of obesity is nonalcoholic fatty liver disease (NAFLD). NAFLD encompasses a spectrum of liver pathologies that begins with lipid droplet (LD) accumulation in hepatocytes, which can progress to nonalcoholic steatohepatitis (NASH). The nuclear envelope (NE) is a specialized subdomain of the endoplasmic reticulum (ER), which is involved in lipid synthesis and the formation and trafficking of lipid droplets. Lamina-associated polypeptide 1 (LAP1) is an integral protein of the inner nuclear membrane of the NE. LAP1 interacts with torsinA, an ER resident ATPase that requires LAP1 to activate its enzyme activity. Given the role of the ER and emerging role of the NE in steatosis, we have examined the effect of LAP1/tosinA complex on the hepatic lipid metabolism and LD biogenesis.

Methods: We generated hepatocyte-specific LAP1 conditional knockout (CKO) mice and torsinA CKO mice and used various in vivo analyses to assess hepatic glucose and lipid metabolism including very low-density lipoprotein (VLDL) secretion experiments in these mice. Histological and imaging analysis including electron microscopy (EM) were used to examine liver sections and isolated hepatocytes. We also used viral-vector mediated gene knockdown techniques for in vivo and in vitro studies.

Results: Hepatocyte-specific LAP1 CKO mice on a chow diet had steatosis starting at 16 weeks of age, progressing to NASH after 1 year of age. Hepatocyte-specific torsinA CKO mice developed striking steatosis at early ages and displayed abnormal ER LD accumulation. Neither CKO model was obese or insulin resistant. Histological and EM studies on livers from torsinA CKO mice demonstrated abnormalities of ER structure. In contrast, LAP1 CKO mice had nuclear as well as cytoplasmic LDs. Both CKO models had significantly reduced secretion rates of VLDL triglycerides and apolipoprotein B100. Defective secretion of apolipoprotein B was also observed in isolated hepatocytes from each CKO model. We have developed cellular models in which either LAP1 or torsinA are knocked-downed to study the molecular mechanisms of LAP1-torsinA complex on LD biogenesis and VLDL secretion in hepatocytes.

Conclusion: Our results demonstrate critical roles of LAP1 and torsinA in the assembly and secretion of VLDL and the normal trafficking of ER-derived LDs. They describe a novel protein complex at NE involved in hepatic lipid metabolism and steatosis development. Clarifying the role of a novel protein complex at the NE in hepatic lipid metabolism will lead to information on its potential role in the development of NAFLD and NASH.

# Board 17, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Adipocyte-Specific Tribbles1 Regulates Plasma Adiponectin Levels and Lipid Metabolism |
| **Authors** * | Elizabeth E. Ha*, Ruifeng Ling, Jian Cui, Robert Bauer |
| **Institutional Affiliations For Each Author.** * | Columbia University, Columbia University, Columbia University, Columbia University |
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**Structured Abstract** *

Background:

Genome-wide association studies (GWAS) have identified single nucleotide polymorphisms (SNPs) near the gene Tribbles-1 (TRIB1) that significantly associate with coronary artery disease (CAD) and plasma lipid traits, suggesting a role for the pseudokinase TRIB1 in lipid metabolism and disease. Additionally, the same SNPs are genome-wide significantly associated human plasma adiponectin levels, suggesting that TRIB1 has a role in adipose tissue. Functional studies in mice have identified roles for hepatic Trib1 and its interaction with the transcription factor Cebpα in lipid metabolism, but few studies have investigated adipocyte-specific Trib1.

Methods:

We developed adipocyte-specific Trib1 knockout (Trib1_ASKO) mice by crossing Trib1-floxed mice with AdipoQ-Cre mice. Plasma cholesterol and triglycerides in both chow- and high fat diet-fed mice were measured after a 4hr fast, and plasma adiponectin was measured by ELISA (n = 7/group). Plasma glucose and glucose tolerance was measured after a 16hr fast (n = 7/group). RNA-seq of subcutaneous white adipose tissue (scWAT) adipocytes was performed (n = 4/group), and differentially expressed genes were identified using DESeq2 analysis and annotated by DAVID pathway analysis. To measure adiponectin secretion, scWAT explants were placed in Opti-MEM for 24hr, and secreted adiponectin was measured via ELISA (n = 4/group). Histology, qPCR, and immunoblotting were performed on whole scWAT.

Results:

Trib1_ASKO mice had decreased plasma triglycerides (>20%) and cholesterol levels (~15%) as well as increased plasma adiponectin levels (>20%) compared to their wild-type counterparts. Additionally, high fat diet-fed Trib1_ASKO mice exhibited improved glucose tolerance. There was no difference in adipose burden, adipocyte morphology, or Cebpα protein levels, suggesting that adipose Trib1's mechanism is Cebpα-independent. RNA-seq of adipocytes from wild-type and ASKO mice revealed 5,436 differentially expressed genes, many of which were annotated as secreted, glycosylated, or containing disulfide bonds; this suggests that Trib1 modulates the adipocyte secretory pathway, perhaps via the endoplasmic reticulum. Indeed, adipose explant studies suggest that Trib1 KO adipocytes have increased adiponectin secretion. Meanwhile, hepatic cholesterol synthesis genes were downregulated in Trib1_ASKO mice, suggesting that adipose-hepatic crosstalk may be involved in regulating cholesterol levels.

Conclusions:

TRIB1 is a recently identified novel regulator of plasma lipid metabolism, and our data suggests that this regulation is in part accomplished in an adipocyte-specific manner. Our data suggests that Trib1 regulates the adipocyte secretome, which may regulate lipids via extra-adipose tissues. Our ongoing studies are aimed at elucidating the cellular and physiologic mechanisms of adipose-specific Trib1.

# Board 18, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Disruption of insulin stimulated Glut4 translocation in brown adipose tissue induces systemic insulin resistance |
| **Authors** * | Picatoste B, Soares D, Alves–Bezerra M, Cohen P, Cohen DE, McGraw TE. |
| **Institutional Affiliations For Each Author.** * | Department of Biochemistry, Weill Cornell Medical College, Department of Biochemistry, Weill Cornell Medical College, Joan & Sanford I. Weill Department of Medicine, Weill Cornell Medical College, Laboratory of Molecular Metabolism, The Rockefeller University, Joan & Sanford I. Weill Department of Medicine, Weill Cornell Medical College, Department of Biochemistry, Weill Cornell Medical College |
| **Corresponding Author Email** * | map2094@med.cornell.edu |

**Structured Abstract** *

Background:
Defective GLUT4 translocation to the plasma membrane is a hallmark of insulin resistance and type 2 diabetes. We have shown that knockout of Rab10 severely inhibits translocation of GLUT4 to the plasma membrane of white and brown adipocytes, resulting in a blunting of insulin–stimulated glucose uptake. Our studies of an adipose–specific (white and brown fat) Rab10 knockout mouse (aRab10KO) revealed systemic insulin resistance on a normal chow diet, primarily due to hepatic insulin resistance (Vazirani R et al, Diabetes 2016). The main function of brown adipose tissue (BAT) is to dissipate energy in the form of heat. However, numerous studies support a role for BAT in metabolic regulation beyond thermogenesis.

Methods:
To determine the impact on whole body metabolism of solely silencing Rab10 in BAT, we generated a BAT–specific Rab10 knockout mouse (bRab10KO mouse).

Results: Rab10 protein expression was lost in brown adipose tissues of bRab10KO mice, while remaining unchanged in white adipose tissue. The bRab10KO mice are insulin resistant on a normal chow diet. bRab10KO mice are glucose intolerant and have elevated plasma insulin levels during a glucose tolerance test compared to control mice. However, fasting blood glucose and plasma insulin levels are unchanged. The body composition is different in the bRab10KO mouse: Visceral and inguinal fat are heavier in the KO mice compared to controls, suggesting that the disruption of BAT metabolism induced by deletion of Rab10 indirectly affects white adipose tissue.

Conclusion: Disruption of insulin stimulated Glut4 translocation in brown adipose tissue induces systemic insulin resistance. Analyses of bRab10KO mouse demonstrate the importance of the insulin–regulated glucose flux into BAT for whole body metabolic regulation.

# Board 18, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Reversal of obesity and diabetes by ketogenic diet: Orle of epigenetic and bistability mechanisms |
| **Authors** * | Fumiko Isoda, Cesar Moreno, Charles V. Mobbs |
| **Institutional Affiliations For Each Author.** * | Department of Neuroscience, Icahn School of Medicine |
| **Corresponding Author Email** * | charles.mobbs@mssm.edu |

**Structured Abstract** *

Background: Ketogenic diets reverse obesity and obesity, but the mechanisms mediating these effects remain unknown. We have previously shown that inhibition of hypothalamic expression of the histone acetytlase Creb-binding protein (Cbp) induces robust obesity and diabetes, in association with induction of hypothalamic Cpt1a, a rate-limiting step of oxidation.

Methods: Inhibition of hypothalamic Cbp by the Cre-lox approach, and expression of hypothalamic gene expression by qPCR across. We have now shown that inhibition of hypothalamic CBP leads to elevated Cpt1a and induces Ppar-alpha, which in turn produces Cpt1a, leading to a bistable state pro-beta oxidation, pro-obese state, difficult to reverse. This bistable state may explain the difficult of reversing obesity and diabetes. However, the key ketone produced by the ketogenic diet is 3-hydroxybutyrate, which is known to act as an HDAC inhibitor. Similar HDAC inhibitors such as NaButyrate reverse obesity, presumably by reversing the elevated HAT produced by CBP. Furthermore most strains with the highest hypothalamic expression levels of CBP exhibit the highest weight gain on a high-fat diet, and live the longest on a standard low-fat diet. Conclusions: HDAC inhibitors including phenylbuytrate may be useful to treat obesity and diabetes.

# Board 19, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Serotonin and dopamine mimic glucose-induced reinforcement in C. elegans: Potential Role of NSM neurons and the ser-4 receptor |
| **Authors** * | Elizabeth Schwart, Charles Mobbs |
| **Institutional Affiliations For Each Author.** * | Dept. of Neuroscience, Icahn School of Medicine |
| **Corresponding Author Email** * | charles.mobbs@mssm.edu |

**Structured Abstract** *

Background. Food produces powerful reinforcement that can lead to overconsumption and likely contributes to the obesity epidemic. Methods. The present studies examined molecular mechanisms mediating food-induced reinforcement in the model system C. elegans. Results. After a 1-hour training session during which food (bacteria) is paired with the odorant butanone, odor preference for butanone robustly increased. Glucose mimicked this effect of bacteria. Glucose-induced odor preference was enhanced similarly by prior food withdrawal or blocking glucose metabolism in the presence of food. Food- and glucose-induced odor preference was mimicked by serotonin signaling through the ser-4 receptor. Dopamine (thought to act primarily through a D1-like receptor) facilitated, whereas the D2 agonist bromocriptine blocked, food- and glucose-induced odor preference. Furthermore, prior food withdrawal similarly influenced reward produced by serotonin, dopamine, or food, implying post-synaptic enhancement of sensitivity to serotonin and dopamine. Conclusions. These results suggest that glucose metabolism plays a key role in mediating both food-induced reinforcement and enhancement of that reinforcement by prior food withdrawal, and implicate serotonergic neurons in these processes.

# Board 19, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Regulation of de novo Lipogenesis in the Enterocyte |
| **Authors** * | Tasleenpal Akal* <br> Sai Komakula <br> Hong Ye <br> Deeptha Kumaraswamy <br> Harini Sampath |
| **Institutional Affiliations For Each Author.** * | Department of Nutritional Sciences, Rutgers University, New Brunswick, New Jersey |
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**Structured Abstract** *

Background: Stearoyl–CoA Desaturase 1 (SCD–1) is an enzyme that resides in the endoplasmic reticulum. Its primary role is catalyzing the formation of monounsaturated fatty acids (MUFAs) from saturated fatty acids. Specifically, it converts palmitoyl–CoA (16:0) and stearoyl–CoA (18:0) to palmitoleoyl–CoA (16:1) and oleoyl-CoA (18:1), respectively, by inserting a double bond at the delta 9 position. These MUFAs are the primary substrates for triglyceride synthesis. As such, excess triglyceride synthesis and storage can contribute to lipid induced disorders such as, obesity and Type 2 diabetes. Previous studies have shown SCD–1 knockout mice have increased energy expenditure and are resistant to obesity. The role of SCD–1 has been extensively explored in different tissues such as liver, adipose, and skin, through tissue–specific knockout models. However, little is known about the role of SCD–1 in the intestine.

Methods: Previous studies have shown that after fasting, refeeding a high carbohydrate (fat–free) diet increases the expression of SCD–1 as well as other lipogenic genes. Since little is known about regulation of lipogenesis in the intestine, we sought to explore the regulation of SCD–1 as well as other genes involved in lipogenesis and fatty acid oxidation within the Duodenum, Jejunum, and Ileum. We utilized a fasting refeeding protocol to acutely modulate lipogenesis. In this study, wild type mice were fasted for 24 hours and then refed a High Sucrose Very Low Fat (HSVLF) Diet for 16 hours. We measured gene expression of acetyl CoA–Carboxylase (Acc), fatty acid synthase (Fas), stearoyl CoA–Desaturase 1 (Scd–1), Sterol Regulatory Element Binding Protein (Srebp–1c) , acyl–CoA synthase (Acsl),carnitine palmitoyl transferase I (Cpt–1), acyl–CoA Oxidase (Aox), and peroxisome proliferator–activated receptor alpha (Ppar–a).

Results: Our results show an increase in SCD–1 expression as well as other genes involved in lipogenesis after re–feeding in the duodenum, jejunum and ileum. There is also an upregulation of genes involved in fatty acid oxidation in the fasted state in the duodenum, jejunum, and ileum.

Conclusions: Regulation of SCD–1 and other genes involved in lipogenesis as well as fatty acid oxidation in the Duodenum, Jejunum, and Ileum is similar to what we have seen in previous literature regarding liver and adipose tissue.

# Board 20, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Allograft inflammatory factor–1–like (AIF1L) is not essential for the pathogenesis of obesity but in certain settings such as presence of Ella–Cre–transgene– it confers resistance to obesity. |
| **Authors** * | Dippal H. Parikh*, Dario F. Riascos bernal, Lander Egana Gorrono, Prameladevi Chinnasamy, 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 |
| **Corresponding Author Email** * | nicholas.sibinga@einstein.yu.edu |

**Structured Abstract** *

Background: Allograft inflammatory factor–1 (AIF1) is a pro–inflammatory molecule that has been linked to body mass, obesity, and diabetes in various human populations. Interestingly, a paralog of AIF1 called AIF1–like (AIF1L) was identified by the human genome project but has not been linked to metabolism. Compared to AIF1, AIF1L has sequence identity of 63% and similarity of 80% but has not been well studied, and little is known about its physiological function. Based on known AIF1 associations with metabolism, the high homology of these proteins, and expression of AIF1L in adipose depots, we hypothesized that AIF1L might play a functional role in diet–induced obesity (DIO).

Methods: The mating strategy employed generated WT and AIF1L–deficient mice –– with and without the presence of Ella–Cre transgene. After we confirmed similar growth and weight on chow diet at the age of 8 weeks, all 4 groups of mice were subjected to HFD for 16–18 weeks.

Results: We observed that the Ella–Cre transgene suppressed HFD–induced weight gain relative to mice lacking the transgene. Remarkably, loss of AIF1L revealed an opposite effect of the transgene, as Ella–CreTg+, AIF1L–deficient mice weighed significantly more than Ella–CreTg+, AIF1L–replete mice. These findings suggest a genetic interaction between the transgene and Aif1L loci. The differences in body weight were attributable to an increase in adipose tissue mass in Ella–CreTg+, AIF1L–deficient mice, while lean mass in mice of both sexes was preserved. In females, both subcutaneous and visceral fat depots increased in mass. In males, on the other hand, increased adiposity was accompanied by larger subcutaneous fat depots and fatty–appearing livers, while visceral fat depots weighed less when compared to WT. These findings indicate a differential depot– and sex–dependent role for AIF1L. Also, metabolic profiles after 18 weeks of HFD – with clear differences in adipose burden – showed decreased physical activity in AIF1L–deficient mice, without differences in energy expenditure. Energy expenditure analysis prior to and early in HFD feeding will give us more insight into the source of the phenotype. Interestingly, WT mice and AIF1L–deficient mice without the Ella–Cre transgene gained weight similarly in response to HFD, with no differences in their body weight curves, fat and lean mass, or metabolic profiles.

Conclusions: Taken together, these results show that AIF1L is not essential for the pathogenesis of obesity; however, in some settings - such as the presence of the Ella–transgene - it confers resistance to DIO in a sex–dependent manner.

# Board 20, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Co-localization of neuronal nitric oxide synthase (nNOS) and bone morphogenetic protein receptor 1a in the ventromedial hypothalamus (VMH) |
| **Authors** * | Pallabi Sarkar*, Hamad Wajid*, Kevin B. Knapp, Vishwendra Patel and Vanessa H. Routh |
| **Institutional Affiliations For Each Author.** * | Department of Pharmacology, Physiology and Neuroscience, Rutgers University. |
| **Corresponding Author Email** * | routhvh@njms.rutgers.edu |

**Structured Abstract** *

•Background:

The role of the ventromedial hypothalamus (VMH) in glucose homeostasis is complex. VMH activation increases blood glucose following insulin-induced hypoglycemia. Conversely, the VMH is also part of the neurocircuitry which lowers blood glucose and combats obesity. This neurocircuitry mediates increased brown fat thermogenesis and the browning of white fat in response to estrogen. Glucose decreases the activity of VMH glucose-inhibited (GI) neurons. We have shown that AMP-activated protein kinase (AMPK) activation is required for activation of VMH GI neurons in low glucose. In addition, the majority of VMH GI neurons express the neuronal nitric oxide synthase (nNOS). Both VMH AMPK and nNOS activation are needed to raise blood glucose during hypoglycemia. On the other hand, estrogen directly reduces the activation of VMH GI neurons in low glucose. This suggests that VMH GI neurons must be inactivated in order for estrogen to increase energy expenditure. The thermogenic effect of estrogen in the VMH is mediated by bone morphogenetic protein 8B (BMP8B). Interestingly, VMH AMPK activation opposes the effect of BMP8B and BMP8B inhibits VMH AMPK. Thus, we hypothesize that BMP8B mediates the inhibitory effect of estrogen on GI neurons. To test this hypothesis we first determined whether nNOS and the BMP8B receptor, BMPR1a, co-localize in the same cells in the VMH.

•Methods:

Wild-type adult c57bl/6 mice (n=3) were anaesthetized and transcardially perfused with 4% paraformaldehyde. Their brains were cryosectioned for immunohistochemical detection of nNOS and BMPR1a expression. We then counted the total number of cells (nuclear staining), nNOS+ cells and BMPR1a+ cells in the dorsomedial (dm-VMH), central (cVMH) and ventrolateral (vlVMH) subdivisions of the VMH. Co-localization was expressed as cells exhibiting both nNOS and BMPR1a immunoreactivity.

•Results:

As reported previously, we observed intense immunoreactivity of nNOS in both dm- and vlVMH. However in the dmVMH, a dense network of nNOS+ fibers made delineation of the cellular outline difficult and therefore this region was not considered for further quantification. Central VMH showed a paucity of nNOS+ cells. In constrast the vlVMH had large, clearly-defined nNOS+ cell bodies and a robust expression of BMPR1a. We observed a larger percentage of cells that were BMPR+ ($61.7 \pm 10\%$ of total) compared to nNOS+ cells ($46.5 \pm 5\%$). BMPR1a and nNOS co-localization was observed in 21.72% of total cells in the vlVMH.

• Conclusion:

The co-localization of nNOS and BMPR1a is consistent with the hypothesis that BMP8B mediates the effect of estrogen on GI neurons.

# Board 21, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | The immune checkpoint B7-H3 (CD276) plays a novel role in adipose tissue homeostasis |
| **Authors** * | Elodie Picarda*, Haihong Zong, Elena Tarabra, Rajat Singh, Jeffrey Pessin and Xingxing Zang |
| **Institutional Affiliations For Each Author.** * | Albert Einstein College of Medicine, Bronx, NY 10461, USA |
| **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. However, its role in adipose tissue biology is unknown.

Methods
B7-H3 expression in mouse adipose tissue and 3T3-L1 preadipocytes was assessed by qPCR, western blot, immunofluorescence and flow cytometry. To investigate its role in adipogenesis, mouse adipocyte progenitors and 3T3-L1 were differentiated in vitro into mature adipocytes and analyzed for B7-H3 expression. Mice knock-out for B7-H3 and age-matched WTs were fed a regular chow diet and changes in body weight, fat mass and histology were assessed. Indirect calorimetry, measures of activity, food intake, insulin and glucose sensitivity were performed. Fat immune cells were analyzed by flow cytometry. Seahorse respirometry allowed to assess basal oxygen consumption rate in fat and liver. Adipocyte fatty acid uptake was measured in vitro with a fluorescent analog.

Results
We found B7-H3 gene expression in mouse gonadal and sub-inguinal fat pads. While absent in mature adipocytes, the protein was detected on non-immune cells of the stromal vascular fraction. Over 95% of the adipocyte progenitor cells, defined as CD45-CD31-CD29+Sca1+PDGFR1a+, express B7-H3, both intracellularly and on their surface. B7-H3 is also highly expressed by the 3T3-L1 cell line. We found it is downregulated in the early stage of differentiation, suggesting a role in regulating adipogenesis. Interestingly, B7-H3KO mice developed spontaneous obesity, being 165% heavier than their WT counterparts after 4 months. They displayed increased fat mass, hypertrophic adipocytes, 'whitening' of brown fat and a fatty liver. Although glucose sensitivity was normal, the KO mice had increased fasting glucose and a lower insulin sensitivity. These metabolic alterations were associated with adipose tissue inflammation through the recruitment of proinflammatory M1 macrophages and T cells. B7-H3KO mice respiratory exchange ratio was higher suggesting a preference for glucose over lipids as energy source. Fatty acids oxidation was reduced in white fat from KO mice compared to WTs, explaining in part the increased adiposity of KO mice. Furthermore, fatty acid uptake was increased in insulin-stimulated KO adipocytes, which may contribute to sustaining the obese phenotype.

Conclusions
Our study helps 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.

# Board 21, Poster 2

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Mapping the Developing Beige Fat by Single Cell Analysis |
| **Authors** * | Kosaku Shinoda |
| **Institutional Affiliations For Each Author.** * | Department of Medicine and Molecular Pharmacology, Albert Einstein College of Medicine, Bronx, New York 10461 |
| **Corresponding Author Email** * | Kosaku.Shinoda@einstein.yu.edu |

**Structured Abstract** *

Brown adipose tissue (BAT) is a specialized adipose tissue that dissipates energy for heat generation, whereas white adipose tissue (WAT) functions as storage of excess energy. Studies suggest that loss of BAT is linked to decreased energy expenditure and obesity in humans; thus increasing energy expenditure through regeneration of BAT could be effective to counteract obesity. Certain physiological cues, such as chronic cold exposure, convert WAT into mitochondria-rich, energy consuming BAT-like adipocyte. This browned adipocyte is referred to as a beige adipocyte and recent studies including ours indicate that adult human BAT is mostly composed of beige adipocytes. The objective of the proposed research is to determine all subtypes of cells that give rise to beige adipocyte by single-cell RNA sequencing.

# Board 22, Poster 1

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| **Abstract Topic Category** * | Metabolism and Integrative Physiology |
| **Abstract Title** * | Obesity reduces vitamin D but does not increase parathyroid hormone (PTH) in the elderly unlike younger individuals. |
| **Authors** * | Hao L, Carson J, Amariti J, Shapses SA. |
| **Institutional Affiliations For Each Author.** * | Rutgers University and Rutgers–Robert Wood Johnson Medical Center |
| **Corresponding Author Email** * | shapses@rutgers.edu |

**Structured Abstract** *

Background: There is a classic inverse relationship between PTH and vitamin D, and the relationship is attenuated by obesity (Shapses 2011). In addition, it is well established that serum 25–hydroxyvitamin D (25OHD) is decreased in the obese and is associated with higher levels of PTH. With advances in medicine, overweight and obese individuals are living to older ages, and it is unclear how aging affects the vitamin D–PTH axis in this population.

Methods: In this study, we examined 514 persons who categorized by age: 50–69 y (n=252) or elderly, ages 70+ y (n=262) and by 3 BMI categories were: < 25 (normal weight; n=192); 25–29.9 (n=178); and obesity $\geq$ 30 kg/m2 (n=149). Study participants were either healthy individuals in the community or patients scheduled for hip surgery. Fasting blood was analyzed for 25OHD and PTH.

Results: As expected, the younger group (58.4 $\pm$ 5.2 y) compared to elderly (83.1 $\pm$ 6.4 y) had higher circulating 25OHD (23.6 $\pm$ 7.6 vs 21.8 $\pm$ 9.2 ng/mL) ($p < 0.01$), and lower PTH (44.9 $\pm$ 30.7 vs 54.5 $\pm$ 40.5 pg/mL). The mean BMI was 22.1 $\pm$ 1.9, 27.5 $\pm$ 1.5, and 34.8 $\pm$ 4.5 kg/m2 in the 3 BMI categories, respectively. Circulating 25OHD was lower in the obese (22.2 $\pm$ 8.1 ng/mL) compared to the normal weight (23.0 $\pm$ 9.3 ng/mL) in both age groups. For PTH, there was an age by BMI interaction ($p < 0.05$), indicating that PTH was higher in obesity (49.0 $\pm$ 26.5) compared to normal weight (35.0 $\pm$ 17.8 pg/ml) ($p < 0.05$) in the 50–69 y group, but not in the elderly (44.3 $\pm$ 26.1 in the obese vs. 55.2 $\pm$ 42.0 pg/ml in the normal weight). In addition, there was the expected inverse relationship between 25OHD and PTH in the entire population ($p < 0.01$), but the relationship only remained significant in the 50–69 y group (r= –.206; $p < 0.01$). Conclusion: These findings show the expected decline in 25OHD and rise in PTH due to aging and increasing BMI. However, the normal inverse relationship between obesity and higher PTH is attenuated in the elderly. Since high PTH is associated with higher mortality, especially CVD mortality, it would be interesting to determine if the lower PTH is a protective factor in the elderly obese population.

# Board 22, Poster 2

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| Abstract Topic Category * | Neurological |
| Abstract Title * | Repeated Hypoglycemia causes functional impairment to Growth-Hormone Releasing Hormone Neurons: Implications for Post-Bariatric Surgery Populations |
| Authors * | Mitchell Bayne1, Alexandra Alvarsson1, Kavya Devarakonda2, Jennifer Nam2, Maria J. Gonzalez1, Chis Jewell1, Merina Varghese2, Patrick Hof2, Sarah A Stanley1,2* |
| Institutional Affiliations For Each Author. * | 1-Diabetes, Obesity and Metabolism Institute, Icahn School of Medicine at Mount Sinai<br>2-Department of Neuroscience, Icahn School of Medicine at Mount Sinai |
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**Structured Abstract** *

Maintenance of normal blood glucose levels is a complex process involving coordinated action of the central nervous system (CNS) and peripheral organs. Those who undergo bariatric surgery have seven times the risk of hypoglycemia compared to those without surgery which may lead to cognitive and metabolic defects. The cause of this side effect is controversial but may be due in part to a blunted, centrally-coordinated counter-regulatory response. There are several counter-regulatory responses to hypoglycemia including increased growth hormone. While the brain monitors and adjusts blood glucose levels constantly, the neuronal adaptations to repeated hypoglycemic episodes are poorly understood. In previous work, we found neurons expressing growth hormone releasing hormone are highly responsive to low glucose. Therefore, this hypothalamic population of neurons provides a model to study how glucose-responsive neurons respond and adapt to hypoglycemia. In vitro studies using N38 mouse hypothalamic cells, which express GHRH, show changes in activity and gene expression in response to altered glucose. In vivo, hypoglycemia increases plasma growth hormone and expression of the early immediate gene c-fos in GHRH neurons in GHRH-GFP mice. In addition, GHRH neurons are synaptically connected to peripheral organs regulating glucose metabolism suggesting they may regulate peripheral glucose via mechanisms other than growth hormone. We also examined the effects of repeated hypoglycemia on the activity, gene expression and neuroanatomy of N38 cells in vitro and GHRH neurons in vivo. These studies suggest GHRH neurons may contribute to the counter-regulatory response to hypoglycemia and to the blunted counter-regulation with repeated hypoglycemia.

# Board 23, Poster 1

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Structural and functional brain differences in children with the FTO obesity risk allele |
| **Authors** * | Claudia Lugo–Candelas*, Yajing Pang, Lisa Ranzenhofer, Rachel Korn, Haley Davis, Hailey McInerny, Janet Schebendach, Wendy K. Chung, Rudolph L. Leibel, B. Timothy Walsh, Jonathan Posner, Michael Rosenbaum, Laurel Mayer |
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**Structured Abstract** *

Background: Brain mechanisms underlying observed associations among the FTO gene (intronic SNP rs1421085), food intake, and obesity are not fully understood. Studies in adults suggest structural and functional brain differences by genotype. Studies in children are few and methodologically limited. Method: We examined associations between FTO genotype at rs1421085 and brain structure and function in a sample of healthy children who were not obese (body fat %ile for age and sex ≤ 95%). DNA extracted from saliva was used to genotype (C/T alleles) at rs1421085. Grey matter (GM) morphology, white matter (WM) fiber density, and resting-state functional connectivity (rsfMRI), were assessed using deformation–based GM morphometry, fixel–based morphometry, and seed–based analyses, respectively. Results: Ninety-three children (age= 9.12±1.17 years) were studied (genotypes: 15 CC, 31 CT, 47 TT). Age [F(2,90)=0.38, p=0.69], sex [χ2(2)=0.37, p=0.83], and body mass index (BMI) [F(2,90)=0.47, p=0.63] did not vary across genotypes. C/X individuals showed greater GM volume bilaterally in the cerebellum, temporal gyrus and right occipital cortex (whole brain corrected p < 0.05; adjusted for sex, age, BMI), altered WM fiber density in the inferior cerebellar peduncle tract (FWE corrected p < 0.05, adjusted for sex, age, BMI), increased positive rsfMRI between the L cerebellum, L superior frontal gyrus, and R thalamus as well as increased negative rsfMRI between the cerebellum and the temporal fusiform cortex and lateral occipital cortex (thresholded at voxel level p < 0.001 (uncorrected) and at cluster level p < 0.05 (FDR corrected); adjusted for age, sex, BMI). Conclusions: Our multimodal analyses suggest prominent differences in cerebellar structure/function related to FTO genotype. As the cerebellum has been implicated in reward–based learning and cerebellar activity has been related to BMI, future research is needed to explore the effects of the FTO genotypes on brain development, food intake, and obesity susceptibility.

# Board 23, Poster 2

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Hypothalamic–Habenular–Midbrain Communication Regulates Food Preference in Lean and Obese Rodents |
| **Authors** * | Richard M. O'Connor*, Maria V. Micioni Di Bonaventura, W. Matthew Howe, |
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**Structured Abstract** *

Background
Rates of obesity are on the rise worldwide, resulting in a growing threat to public health. Pharmacotherapies that safely reduce body weight in obesity remain elusive, partially due to our incomplete knowledge of the complex neuronal mechanisms that control food choice (palatable high–calorie versus less palatable low–calorie food). The lateral hypothalamus (LH) is considered a critical node in the maintenance of energy homeostasis. A major output of the LH terminates in the lateral habenula (LHb) which has been described as a "preference center" and exerts a negative influence over motivated behaviors through inhibition of midbrain dopamine neurons. We tested the hypothesis that LH projections to LHb play an important role in food preference and food–related motivation through downstream influences on midbrain dopamine neurons.

Methods
Circuit mapping: Glycoprotein–deleted rabies and cre–dependent TVA–mCherry/glycoprotein viruses were injected into LHb. Retro–AAV–iCre was delivered to the ventral tegmental area (VTA).
Recording neuronal activity: retro–AAV–iCre was delivered to VTA and cre–dependent GCaMP was injected into LHb. A fiber optic was implanted in LHb.
Manipulation of LH inputs to LHb neurons: Retrograde AAV2/5–Cre–eYFP was delivered to LHb and Cre–inducible diphtheria toxin (DTA) or Cre–inducible (hM3Dq) DREADD was delivered to LH.

Results
Using monosynaptic rabies tracing we found prominent innervation of ventral tegmental area (VTA) projecting LHb neurons originating in LH. Using fiber photometry, we found neuronal activity of these VTA projecting LHb neurons decreased in hungry animals during the retrieval of regular rodent chow rewards (homeostatic feeding) and in sated animals during palatable food consumption (hedonic feeding). In lean animals the magnitude of decreased neuronal firing from baseline was greatest for homeostatic food seeking compared to hedonic. Interestingly, this pattern switched when animals became obese through exposure to a cafeteria style. DREADD–mediated stimulation of the LH inputs to the LHb decreased consumption of palatable energy–dense food, whereas ablation of this pathway increases consumption of the palatable food; opposite effects of these manipulations were observed when only standard (less palatable) chow was made available.

Conclusions
These findings identify the LH–LHb–VTA pathway as an important brain circuit involved in feeding and deficits in this circuit may emerge during weight gain and contribute to obesity–associated behavioral abnormalities. Modulation of activity at this circuit may represent a promising therapeutic strategy for reversing hyperphagic feeding patterns that generate and maintain obesity.

# Board 24, Poster 1

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| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Obesity with and without food addiction differ in depressive symptoms, emotional eating, attention bias to food, and hemispheric brain asymmetry |
| **Authors** * | Aviram-Friedman, R., Kafri, L., Alyagon, U., Avinoah, E., Zangen, A. |
| **Institutional Affiliations For Each Author.** * | Zlotowski center for neuroscience: Ben-Gurion University (Israel), Soroka University Medical Center (Israel) |
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**Structured Abstract** *

Background: Obesity with symptoms of addictive eating (OBAE) is a clinically significant condition, which can be measured with the Yale Food Addiction Scale (YFAS). However, the causes and implications of this condition are poorly understood. Methods: As part of a larger clinical trial, the present study sought to compare between obese adults (N = 23) with a similar BMI (range 28–42), but who differ in their YFAS scores [symptoms (YFAS-S) and clinical diagnosis (YFAS-D)]. Participants arrived at the lab in the morning, following a 24-hr dietary preparation and a 12-hr overnight fast. They were weighted, received a standardized breakfast, completed questionnaires [YFAS, Beck depression inventory (BDI), and the three-factor eating questionnaire (TFEQ)], a Food Stroop neurocognitive task, and a 5-min electroencephalographic (EEG) recording at rest. Results: Two groups with distinct profile were found: OBAE (N = 15; M/F:3/12), and obese with no addictive eating (OB; N = 8; M/F:4/4). The level of their depressive symptoms predicted YFAS-D and YFAS-S scores (F = 5.18, p = .04 and F =5.65 p = .03, respectively), and mean BDI score was greater in the OBAE compared with the OB (12.21 Vs. 5.29, respectively; t = −2.14, p = .05). Similarly, symptoms of emotional eating (EE; TFEQ) predicted YFAS-S scores (F = 12.14 p = .002). YFAS-D predicted greater EEG laterality in the left inferior frontal gyrus (IFG; F = 5.33 p = .03), the supramarginal gyrus (F = 5.78 p = .03), and visual association areas (F = 5.11 p = .04). Greater YFAS-S predicted greater left EEG laterality in the post-central gyrus as well (PCG; F = 4.94 p = .04). Lastly, mean reaction time to the Stroop stimuli, following the presentation of high- vs. low-calorie food images, differed between the groups (F = 5.6, p = .03). Discussion: despite no differences in BMI, the obese adults in our sample differed in their psycho-neuro-cognitive profile, based on their addictive eating diagnosis and symptoms. Replication of our findings in a larger sample of obese adults may provide a novel insight into different obesity subgroups, to whom different clinical intervention may be indicated.

# Board 24, Poster 2

| Abstract Topic Category * | Neurological |
|---|---|
| Abstract Title * | Obesity propensity differentially influences morphine withdrawal behaviors in male mice |
| Authors * | Xinyi Li*, Dushyant Kshatriya, Gina Giunta, Nicholas T. Bello |
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Structured Abstract *

Introduction

Individual differences contribute to the varying susceptibility for weight gain and opioid use. The divergence and convergence nature of these individual susceptibilities has not been examined. Therefore, the objective of this study was to determine the influence of obesity propensity on morphine withdrawal behaviors.

Methods

Obese-prone (OP) and obese resistant (OR) male C57BL/6J mice were identified on an 8-weeks high-fat diet (45% Fat). Following identification, OP and OR mice were fed a 5-weeks control diet (10% Fat) and randomized to receive 7-day (twice daily) injections of saline or escalating doses of morphine (20-100 mg/kg/day, i.p.). Following a 7-day withdrawal period, open field exploration test, elevated plus maze, and pre-pulse inhibition were performed sequentially on three succeeding days.

Results

During the dosing and withdrawal period, there were treatment effect ($p<0.05$) and treatment x time ($p<0.05$) effect on % body weight change. Morphine administration resulted in a significant decrease in % body weight change in both OP and OR mice (n=14-15). Following the 7-days withdrawal period, open field exploration test revealed a greater number of line crossings in morphine- treated OR mice (n=14) in comparison to saline-treated OR mice (n=14) ($p<0.05$). OP mice exhibited no such differences (saline-OP, n=15; morphine-OP, n=14). No group or treatment effects were observed in elevated plus maze. Morphine-treated OP mice displayed attenuated pre-pulse inhibition. With a 74 dB pre-pulse intensity, morphine-treated OP mice (n=14) exhibited lower % pre-pulse inhibition in comparison to morphine- treated OR mice (n=11) ($p<0.05$). In response to 90 dB pre-pulse intensity, % pre-pulse inhibition exhibited by morphine-treated OP mice were lower than that of saline controls (n= 15 for both OR and OP) and morphine-treated OR mice ($p<0.05$ for all).

Conclusion

Individual differences culminating in the spectrum of lean and obese phenotypes may contribute to differences in morphine withdrawal.

# Board 25, Poster 1

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Effects of a 12-week high fat diet on brain, behavior, and perception in healthy humans |
| **Authors** * | AG. DiFeliceantonio, S. Edwin Thanarajah, K. Albus, O. Cornely, M. Hess, JC. Bruening, M., DM. Small, M. Tittgemeyer |
| **Institutional Affiliations For Each Author.** * | Yale School of Medicine, MPI for Metabolism Research, CECAD, CECAD, MPI for Metabolism Research, Yale School of Medicine, MPI for Metabolism Research |
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**Structured Abstract** *

Background: There is clear causal evidence from animal studies that a high fat diet can alter dopamine signaling and impair dopamine-dependent functions. Emerging work in humans also supports a causal relationship; though few studies collect comprehensive measures of diet, metabolism, and adiposity, which may all contribute to brain alterations.
Methods: Here, we provide a preliminary report of an ongoing study where perceptual, metabolic, neural, and behavioral measures are collected before and after healthy-weight participants are assigned to consume either a high protein yogurt (~50% kcal from protein) or an equicaloric high fat (~40% kcal from fat) yogurt twice daily for 12-weeks.
Results: Despite no change in adiposity or metabolic measures, diet influenced fat preference and brain response to a high fat/high sugar milkshake. More specifically, liking and perception of fattiness in fat containing stimuli was altered in the high-fat, but not high-protein group. Liking of sweet stimuli was also altered. Functional magnetic resonance imaging revealed increased brain response to a high-fat high-sugar milkshake in the high fat group in the amygdala and a decrease in the ventral striatum, but no change in the high-protein group (trend, preliminary analysis). We also observed an improvement in impulsivity as measured by stop signal reaction time.
Conclusions: This preliminary analysis supports a causal influence of a high fat diet on fat preference and on neural circuits responding to palatable food cues.

# Board 25, Poster 2

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Physical Activity, Food Environmental Cognition and Regional PFC Activation During Ice Cream Intake |
| **Authors** * | Lisa Lanza, Eram Albajri, Hasan Ayaz, Sinclair Smith, Angelo Del Parigi, Jennifer A Nasser |
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**Structured Abstract** *

Background:

In a study of protein preloads, brain activation (measured by fNIRS) and ice cream consumption, participants (15M, 13F) were characterized by habitual physical activity (PA), cognitive restraint (CR), and food environmental cognition (FC). Post Hoc analysis of the fNIRS and food intake data was conducted to determine potential interactions of brain activation correlates by PA, CR, and FC.

Methods:

Participants attended two randomly assigned sessions, consumed a high (HP, 28 grams) or low protein (LP, 2 grams) equicaloric 120 kcal preload, rested for 15 minutes, then were scanned with fNIRS during eating of ad libitum ice cream. FC was measured by the Power of Food Scale (PFS), CR by Three Factor Eating Questionnaire (TFEQ), and habitual PA by the PAQ-M questionnaire, completed at the end of session 2. Regression analysis was used to determine relationship of PA, PFS, and CR to food intake and regional PFC activation.

Results:

Ice cream intake (under LP preload) was positively correlated with PFS 1,2,3 subscales ($p < 0.05$). mPFC activation (LP) was negatively correlated with PFS-3 ($p= 0.025$), PAQ-M ($p= 0 .018$) and CR ($p= 0.005$). All of these correlations were disrupted under the HP condition. A 26 % reduction in ice cream intake was observed under HP compared to LP when CR was controlled ($p= 0.055$).

Conclusions:

Under LP preload, ice cream intake was affected by FC. This may have real world significance as most commercially available snack foods are low in protein (~2 grams/serving). Increasing the preload protein content disrupted the effect of food environment cognition on ice cream intake. PA and CR possibly exert a health promoting effect by reducing mPFC activation under LP condition. However, the most effective way to reduce ice cream intake and medial PFC activation was through intake of HP preload. Further use of fNIRS to study human eating behavior seems warranted.

# Board 26, Poster 1

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|---|---|
| **Abstract Topic Category** * | Neurological |
| **Abstract Title** * | Binge Eating (BE) and Associated Eating Behaviors in Relation to Regional Brain Volume and Cortical Thickness |
| **Authors** * | Nadia Abdo*, Emily Boyd*, Spiro Pantazatos, Shaunte Baboumian, Allan Geliebter<br>*These authors contributed equally to this work. |
| **Institutional Affiliations For Each Author.** * | Mount Sinai St. Luke's Psychiatry Department, Mount Sinai St. Luke's Psychiatry Department, Columbia University Psychiatry Department, Mount Sinai St. Luke's Psychiatry Department, Mount Sinai St. Luke's Psychiatry Department |
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**Structured Abstract** *

Background: Binge eating disorder (BED) was formally recognized by the DSM-5 in 2013 and is the most common eating disorder. BED is characterized by consuming an abnormally large amount of food, accompanied by a feeling of loss of control. Only a few studies have investigated structural brain MRI scans in relation to binge eating (BE) and none in relation to BE and associated eating behaviors. We examined the presence of BE and three related eating behaviors and their relationship with regional volume and cortical thickness in a large community sample.

Methods: We analyzed structural MRI brain scans from the Nathan Kline Institute (NKI) to compare regional volumetric and cortical thickness among 466 healthy individuals (160 m, 306 f; age 18–85, mean 47.2; BMI 28.0 ± 8.4 SD). We investigated volume and thickness among those with and without BE and in their relation to each of the scores from the Three Factor Eating Questionnaire (TFEQ): dietary restraint (0–20), disinhibition (0–16), and hunger (0–15) as well as the interaction between BE status and the scores. BE status was defined as eating unusually large meals accompanied with loss of control for at least 3 days over the past 4 weeks as reported on the Eating Disorder Examination Questionnaire (EDE–Q). Cortical reconstruction and volumetric segmentation were performed with the Freesurfer image analysis suite (Reuter et al., 2012) controlling for sex, age, BMI, and total intracranial volume. We first ran a region of analysis (ROI) analysis using a priori regions associated with BE in previous literature. An exploratory threshold of p–value <0.001 was applied to run a false discovery rate (FDR) test for BE and the eating behaviors in relation to regional volume and cortical thickness. We also tested for differences the three scores from the TFEQ between those with and without BE.

Results: Those with BE had greater left–accumbens volume (p = .008) compared to those without in the ROI analysis. Higher disinhibition scores were correlated with increased left–accumbens volume (r = .13, p = .00028) in an exploratory analysis. No interactions survived the exploratory threshold or the FDR test. Those with BE had higher scores for disinhibition (p = 1.22E–07) and for hunger (p = 5.88E–07).

Conclusions: Our findings show that BE and the associated behavior of disinhibition were associated with greater volume in the left–accumbens, a reward region, consistent with a greater drive for food.

# Board 26, Poster 2

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Bullying Perpetration and Victimization among Adolescents with Overweight and Obesity in a Nationally Representative Sample |
| **Authors** * | Kristie Rupp, PhD*<br>Stephanie M. McCoy, PhD, MPH |
| **Institutional Affiliations For Each Author.** * | 1 Department of Kinesiology, Brooklyn College of the City University of New York, 435 West Quad Building, 2900 Bedford Avenue, Brooklyn, NY 11210, USA. Kristie.Rupp@brooklyn.cuny.edu<br><br>2 School of Kinesiology, University of Southern Mississippi, 118 College Drive #5142, Hattiesburg, MS 39406, USA. Stephanie.McCoy@usm.edu |
| **Corresponding Author Email** * | Kristie.Rupp@brooklyn.cuny.edu |

## Structured Abstract *

Background: Adolescents with obesity are more likely to experience bullying in comparison to their normal weight peers. However, it is unknown whether adolescents with obesity are more likely to perpetuate bullying or be both a bully perpetrator and bully victim in comparison to their normal weight peers. The purpose of this analysis was to examine differences in bully perpetration, bully victimization, and both (bully perpetration and victimization) by weight classification in a nationally representative sample of adolescents.

Methods: Analyses included 22,048 adolescents (50.2% male) aged 10–17 (mean $13.9 \pm 2.3$ years) from the 2016 National Survey of Children's Health. Adolescents were grouped into categories: normal weight, overweight, and obese. Outcome variables included bullying, difficulty making new friends, excessive arguing, depression, and behavioral conduct problems. Logistic regression models, adjusted for age, sex, race, household income, highest level of education in the household, and ADHD assessed the odds of each outcome comparing normal weight to adolescents with overweight and obesity.

Results: Approximately 15% of adolescents were overweight and 13% were obese. Adolescents with overweight and obesity were more likely to experience bullying behaviors: bully victim only (OR=1.32 and 2.05) and be both a bully perpetrator and bully victim (OR=1.40 and 1.94) (p's <0.001), respectively, in comparison to normal weight peers. Adolescents with overweight and obesity within all bully classifications were more likely to experience behavioral issues and depression, as well as to argue excessively and have difficulty making friends compared to adolescents who are neither a bully perpetrator nor bully victim (p's <0.001).

Conclusions: To promote overall health and well–being among adolescents with overweight and obesity, effort should be made to mitigate engagement in and/or victimization from bullying and associated behavioral or depressive symptoms.

# Board 27, Poster 1

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| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Examination of trends in sugary drink consumption among NYC public high school students: Youth Risk Behavior Survey 2013–2017 |
| **Authors** * | Tamar Adjoian*, Divya Prasad, Amaka Anekwe |
| **Institutional Affiliations For Each Author.** * | Affiliation for all three authors:<br>New York City Department of Health and Mental Hygiene, Bureau of Chronic Disease Prevention and Tobacco Control |
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**Structured Abstract** *

Background: Adolescents are often targeted by industry marketing, may have more discretionary spending money than younger children and have high rates of sugary drink consumption. Adolescence is an important developmental period for promoting interventions that reduce the purchase and consumption of sugary drinks, as these beverages are associated with increased chronic disease risk, including obesity, in adults. This analysis examines sugary drink consumption trends among New York City (NYC) youth from 2013-2017.

Methods: The NYC Youth Risk Behavior Survey (YRBS) is a cross-sectional population-based biennial survey representative of public high school students grades 9-12. It is jointly conducted by the NYC Departments of Health and Education and the Centers for Disease Control and Prevention. Data from three YRBS cycles was used in this analysis (2013-2017). Participants were asked two questions about sugary drink consumption over the past week, one about soda and another about other sugary drinks, like iced tea, sports, energy and fruit drinks. For both questions, respondents were instructed to exclude diet/sugar-free drinks. Response options were: 0, 1-3/week, 4-6/week, 1/day, 2/day, 3/day, or 4+/day; these were subsequently assigned midpoint values, respectively: 0, 2/7, 5/7, 1, 2, 3, or 4 drinks/day. Responses were combined to calculate total sugary drinks/day, then dichotomized to <1 and 1+ sugary drink/day. The prevalence of 1+/day was analyzed each year overall and by sex and race/ethnicity. T-tests compared 2015 to 2017, and trends were assessed from 2013-2017.

Results: Daily sugary drink consumption overall decreased from 41.5% to 34.6% from 2013-2017 (p for trend <.001). In each year, males were more likely to drink sugary drinks daily than females (39.1% vs. 30.0% in 2017, respectively, p<.001), but both sexes saw decreases over time. Black and Latino students had significantly higher rates of daily consumption than White or Asian/Pacific Islander (PI) students in each year, but White, Black, and Latino students all saw significant decreases between 2013-2017. Additionally, between 2015-2017, only Latino and Asian/PI saw significant decreases (Latino: 42.5% to 38.3%, p=.028; Asian/PI: 31.1% to 21.4%, p=.004).

Conclusions: Our results show a decrease overall and within some subgroups. Despite this, overall daily sugary drink consumption among public high school students remains higher than any other age group in NYC. Efforts to further reduce consumption should be prioritized by public health practitioners and clinicians, in order to prevent future chronic disease development.

# Board 27, Poster 2

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|---|---|
| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Nutritional quality for food–insecure individuals: A quantitative analysis of the nutritional quality of food available at urban food pantries |
| **Authors** * | Alexander D. Bryan*, Zoë A. Ginsburg, Ellen B. Rubinstein, Hilary J. Frankel, Andrew R. Maroko, Clyde B. Schechter, Kristen Cooksey Stowers, Sean C. Lucan |
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**Structured Abstract** *

Background: Individuals with food insecurity frequently suffer from diet–related chronic diseases such as obesity, hyperlipidemia, diabetes, and poor glycemic control. Such individuals would benefit from improved nutrition, but often rely on food pantries that provide foods of uncertain nutritional quality. Our objective was to assess the nutritional quality of foods and beverages available from food pantries in the Bronx, NY.

Methods: Using a convenience sample of 50 food pantries throughout the South Bronx, we performed a cross–sectional assessment of pantry foods and beverages with interviews of pantry workers. Data on food/drink type (fresh, shelf–stable, refrigerated/frozen), sourcing (food bank or other), and distribution method (prefilled bags versus client choice, plus client position in line) were linked to nutritional quality using validated NuVal scoring (range 1–100; higher scores indicate better–nutrition items; higher overall scoring diets associated with lower BMI, lower risk of chronic disease, and reduced mortality in cohort studies where highest quintiles were about 40).

Results: Only 21 of 50 visited food pantries were open as scheduled and had food available; 12 used prefilled bags (traditional pantries), 9 allowed for client choice. Mean NuVal scores were higher for items available through client–choice pantries than through traditional pantries (69.3 vs. 57.4), driven mostly by higher percentages of fresh items (28.3% vs. 4.8%). For a hypothetical 'balanced basket' of one of each fruit, vegetable, grain, protein, and dairy item from client–choice pantries vs. traditional pantries, the mean NuVal score varied considerable from best items (98.8 vs. 96.6) to worst items (16.4 vs. 35.4), and by whether persons might be first or last in line for food. Pantry workers reported that lower–scoring items (e.g., white rice) were more popular––appeared in early bags or were selected first––leaving higher–scoring items (e.g., brown rice) for clients later in line.

Conclusions: When food pantries were open and had foods/beverages to offer (<50% of the time), the nutritional quality of available items varied by item type, sourcing, distribution method, and client position in line. Given the association between food–insecurity and diet–related chronic disease such as obesity, this creates important opportunities for education and advocacy.

# Board 28, Poster 1

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|---|---|
| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Associations between maternal pre-pregnancy weight and pregnancy-related factors and child birth weight |
| **Authors** * | Chrisa Arcan*, Janos Hajagos, Wei Hou, Marianne Lawrence, James Bernasko |
| **Institutional Affiliations For Each Author.** * | Dept of Family Population and Preventive Medicine, Dept of Biomedical Informatics, Dept of Family Population and Preventive Medicine, Regional Perinatal Center, Department of Obstetrics, Gynecology and Reproductive Endocrinology Stony Brook University<br><br>Stony Brook University |
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**Structured Abstract** *

Abstract

Purpose: There is increasing evidence that maternal pre-pregnancy weight and weight gain during pregnancy are associated with offspring weight and childhood health. The goal of this study was to examine associations between maternal demographic, body mass index (BMI), gestational diabetes, maternal pre-pregnancy weight, and gestational weight gain with offspring birth weight/length among a racially and socioeconomically diverse population.

Methods: Cross-sectional analysis of maternal factors and offspring weight/length. Child births were extracted from Stony Brook Medicine's Health Intent platform from January 2015 to April 2017. Data from electronic health records (EHR) included mother's pre-pregnancy weight, mother's final weight (kg), and diagnoses coded during pregnancy. After implementing the exclusion parameters, a total of 3441 mother/offspring dyads were analyzed (mean pre-pregnancy weight: 69.35 kg; mean BMI: 26.9; mean age: 30 years; white: 84%). Multivariate regression was used to examine association between independent variables and child birth weight/length. All the variables were simultaneously included in the model and adjusted for age, race, smoking status (yes/no), gestational diabetes, and Medicaid participation (yes/no).

Results/Findings: Based on pre-pregnancy weight, there were 46% normal weight, 27% overweight, and 26% obese mothers; 12% were diagnosed with gestational diabetes. Pre-pregnancy BMI of ≥ 30 was associated with greater offspring weight (p=0.000) in a dose response manner; greater offspring weight at maternal Class III vs. Class I obesity categories. Greater gestational weight gain was also associated with offspring weight (p=0.000). Being Black or a smoker was associated with lower offspring birth weight. Gestational diabetes or insurance type were not significantly associated with offspring weight.

Conclusions: There is clear indication that maternal prepregnancy weight and gestational weight gain are important factors in offspring birth weight. Our findings support implementing nutrition and physical activity interventions to prevent overweight and obesity during preconception and pregnancy, as well as understanding the long term effects of these interventions on child weight trajectory.

# Board 28, Poster 2

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|---|---|
| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | Mediterranean Diet Adherence among Greek Americans |
| **Authors** * | Shamima Khan*<br>Victoria Fischer<br>Maria Hassapidou |
| **Institutional Affiliations For Each Author.** * | CRE Services, Inc.,<br><br>Hunter College, School of Urban Public Health, CUNY,<br><br>Department of Nutrition and Dietetics<br>ATEI of Thessaloniki, P.O.Box 141, 57400 Thessaloniki, Greece |
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**Structured Abstract** *

Background/ Aims:
To determine the degree of adherence of Mediterranean Diet (Med–Diet) among health care professionals of Greek ancestry. In addition to observe the association between Med–Diet and obesity, health related behaviors, and health parameters among health care professionals of Greek ancestry

Methods:
A survey instrument was developed based on previously validated questions, which was used to survey members of two Hellenic Medical Societies between April 2015 and January 2016. Questions were asked regarding consumption of various components of the Med–Diet, anthropometric data, health related behaviors, and medical conditions/ medical history and demographics.

Results:
The main outcome variable (Med–Diet score) was between 33 to 53 (mean 40.6 ±4.3, median 40), 55 being the maximum attainable score. . Of the total 40 respondents who were classified as being normal weight (BMI ≤ 24.9), 61.5% were also classified as being more adherent to the Med–Diet ($X2 = 7.82$, $p = 0.005$).Two respondents had a diagnosis of diabetes and none ever experienced a stroke or heart attack.

Conclusions:
Members of these medical societies displayed formidable adherence to Med–Diet. Overall, the sample engaged in healthful life-style choices, and possibly as a result, exhibited relatively lower risk factors for cardio vascular diseases.

# Board 29, Poster 1

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|---|---|
| **Abstract Topic Category** * | Population Health and Epidemiology |
| **Abstract Title** * | A 10 year follow-up of adiposity and dementia in Swedish adults aged 70-years and older |
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**Structured Abstract** *

Background: Adiposity measured in mid- or late-life, and estimated using anthropometric measures such as body mass index (BMI) and waist-to-hip ratio (WHR), or metabolic markers such as blood leptin and adiponectin levels, is associated with late-onset dementia risk. However, during later life this association may reverse and aging- and dementia-related processes may differentially affect adiposity measures. We explored associations of concurrent BMI, WHR, and blood leptin and high molecular weight adiponectin levels with dementia occurrence.

Methods: 924 Swedish community-dwelling elderly without dementia, aged 70 years and older, systematically-sampled by birth day and birth year population-based in the Gothenburg city region of Sweden. The Gothenburg Birth Cohort Studies are designed for evaluating risk and protective factors for dementia. All dementias diagnosed after age 70 for 10 years were identified. Multivariable logistic regression models were used to predict dementia occurrence between 2000-2005, 2005-2010, and 2000-2010 after excluding prevalent baseline (year 2000) dementias. Baseline levels of BMI, WHR, leptin and adiponectin were used.

Results: Within 5 years of baseline, low BMI (<20 kg/m2) increased the odds of dementia and intermediate leptin levels decreased the odds of dementia in women (p<0.05).

Conclusions: In late-life, anthropometric and metabolic adiposity measures appear to be differentially associated with dementia risk. While BMI and leptin levels are highly positively correlated, our results show that their association with dementia at age ≥ 70 years, is asynchronous. These data suggest that with aging, the complexity of the adiposity exposure may increase and suggests metabolic dysregulation. Additional studies are needed to better understand this complexity.