A. Personal Statement
I have a long-standing interest in preventing child obesity and have developed a vigorous collaborative research program to study how genetic factors interact with our current obesogenic environment to cause excess weight gain in children. I have a strong history of NIH-funded research and have served as principal investigator on R01 and R21 projects. Through these funded awards, I have developed a collaborative, multidisciplinary research program that utilizes diverse methods to better understand risk factors for child obesity. For example, in a randomized trial of pre-adolescent children, my lab found that children with a common obesity-risk variant in the FTO gene had a greater response to food advertisements in terms of food consumption (Gilbert-Diamond, Int J Obes.) and brain response in dopaminergic reward regions of the brain (Rapuano, PNAS) compared to children with the low risk variant. In close collaboration with Dr. Emond (multi-PI on this proposal), I have further researched the effect of environmental food cues on preschool children using both observational and experimental research methods. In one study of 60 preschool children, we demonstrated that food advertisement exposure leads to excess consumption (Emond, Pediatrics). The fMRI-based physiological markers of food-cue-reactivity that I utilize in my research with older participants are not appropriate for very young children, because they are unable to tolerate the loud noises and small confines of the MRI machine. To address that limitation, our lab has developed a robust eye-tracking methodology for use in young children to measure attentional bias to food cues, a proxy for food-cue-reactivity. Dr. Brand (co-I) Dr. Emond, and I have collaborated extensively on this research. Using these methods, we have observed an association between attentional bias to food cues and current BMI z-score in preschoolers (Brand, Appetite). In addition to experimental methods, I also have extensive experience using observational methods with large genome-wide datasets and prospective cohorts to further interrogate obesity risk factors and have published 13 peer-reviewed journal articles in that examine genetic risk for disease outcomes in respected journals such as American Journal of Human Genetics, Human Genetics, Int J. Obesity and PNAS. I have also worked extensively with longitudinal cohorts and have extensive experience with participant recruitment and retention. My research extends to the Dartmouth-Hitchcock weight and wellness center where I have gained experience with clinical translation of obesity prevention and treatment findings. Through that center, I have collaborated extensively with Dr. Meijer, (co-I) who is a PhD researcher and clinical dietician. Dr. Meijer and I meet weekly for 2 hours to discuss ongoing research projects and we have several publications in progress. The research team on this current proposal has complimentary academic training including Cognitive Psychology, Biostatistics, Public Health, Clinical Dietetics, Epidemiology, and Genetics that will support our vigorous interdisciplinary research. Importantly, our team shares a dedication to conducting research that accounts for inter-individual differences to support future child obesity prevention and treatment interventions that are targeted and effective.
Ongoing and recently completed projects that I would like to highlight include:

R01 HD092604  Gilbert-Diamond (PI)  07/01/2018 – 04/30/2023
Title: The Relation of Genetic Factors, Food Cues, and Self-Regulation with Excess Consumption and Adiposity in Children
Goal: The primary objectives of the study are to evaluate the influence of genetics and television food advertisement exposure on children’s eating patterns after they have eaten to satiety.
Role: Principal Investigator

1R21HD097475  Gilbert-Diamond (PI)  09/21/2018 - 08/31/2020
Title: Media multi-tasking and cued overeating: assessing the pathway and piloting an intervention using an attentional network framework
Goal: This research aims to elucidate attentional mechanisms that may mediate a previously observed association between electronic media multitasking and eating in the absence of hunger. It further aims to test whether an intervention designed to reduce attention to peripheral cues can reduce cued eating.
Role: Principal Investigator

1UH3OD023275  Karagas (PI)  09/2018 - 08/2023
Title: A prospective study of critical environmental exposures in formative early life that impact lifelong health in rural US children: The New Hampshire Birth Cohort Study
Goal: The primary objective of this research is to contribute environmental exposure and child health data to the ECHO – Environmental Influences on Child Health Outcomes Pediatric Cohorts program.
Role: Co-Investigator

5R21HD076097  Gilbert-Diamond (PI)  04/01/2014 – 03/31/2016
Title: Children’s genetic predisposition to eat without hunger after food advertisements
Goal: The primary objectives of the study are to evaluate the influence of genetics and television food advertisement exposure on children’s eating patterns after they have eaten to satiety.
Role: Principal Investigator

Citations:


B. Positions, Scientific Appointments and Honors

Positions and Employment

2020-present  Director, Quantitative Biomedical Sciences Graduate Program
2018-present  Associate Professor, Department of Epidemiology; Community & Family Medicine, Geisel School of Medicine at Dartmouth, Hanover, NH
2018-present  Adjunct Associate Professor, Department of Biology; Dartmouth College, Hanover, NH
2018-present  Member, The Obesity Society
2012-present  Member, Cancer Epidemiology Research Program, Norris Cotton Cancer Center, Lebanon, NH
2018-present  NIDDK Fellowship Review Panel
2012-2018  Assistant Professor, Department of Epidemiology; Community & Family Medicine, Geisel School of Medicine at Dartmouth, Hanover, NH
2012-2018  Adjunct Assistant Professor, Department of Biology; Dartmouth College, Hanover, NH
se cues affect consumption and whether some children are genetically predisposed to eat more in response to those cues. I have successfully run a randomized trial of 200 9-10-year-old children where we found that exposure to food advertisements leads to increased consumption in children (Gilbert-Diamond, *Int J Obes*.). In that study, we found compelling evidence that children with a common variant in the *FTO* gene had a greater response to food advertisements. To further investigate the biological mechanism behind that response, we did a study in which we neuro-imaged a subset of children using fMRI. In that sub-study we found that children with the obesity related *FTO* gene variant had increased response to food advertisements in dopaminergic reward regions of the brain compared to children with the low-risk variant (Rapuano, *PNAS*). In addition, I have completed a study of 60 children ages 2-5 years old where we have also found an effect of food advertisement exposure on excess consumption (Emond, *Pediatrics*). This preliminary work led to my current NIH-funded R01 project that uses fMRI to study the dopaminergic reward response in pre-adolescence using fMRI.


2. While GWAS studies have identified many genetic variants related to obesity, the identified variants only explain a small fraction of the heritability of obesity that has been demonstrated through twin studies. To gain the tools to study this missing heritability, I completed my post-doctoral fellowship with Jason Moore in Bioinformatics. One area of my research focuses on the interactions between multiple genetic loci and the interactions between genetic and environmental factors related to obesity and metabolic disease (De, *Hum Gen*, 2017, De *BioData Min*, 2015, Saxena, *Am J Hum Genet*, 2012). This research uses large genomic data sets from genome-wide arrays and involved collaborators from multiple large institutions in the US and abroad; I was senior author on two papers for which the lead author was my graduate student (De, 2017, De 2015). To support such computationally intensive research on highly multidimensional data, I have also developed a machine learning method to study epistasis for quantitative traits (QMDR: quantitative multidimensionality reduction analysis) in collaboration with a statistician; I was senior author on the resulting publication (Gui, *PLoS One*, 2013).
3. My research also focuses on the development of novel methods to assess food approach and avoidance behaviors in children. In collaboration with Drs. Brand and Emond, I have worked extensively to develop eye-tracking methodologies and evaluate eye-tracking hardware and paradigms to better assess attentional bias and attention to peripheral cues in children. I have also collaborated with computer scientists to pilot a device to assess eating rate through collecting and processing audio sounds picked up from the mandible using a head-worn device. The development of that device is ongoing and presents exciting future opportunities for research on eating rates.


4. My research program has recognized the multi-factorial nature of the development of obesity and has used observational cohort studies to better understand how multiple predictors affect one’s risk for developing obesity. In addition to studying how genetic factors and responsivity to food cues affect excess consumption and obesity, my research has also studied other factors including, but not limited to, electronic media in the bedroom (Gilbert-Diamond, Jama Pediatr, 2014), media multitasking (Lopez, Front Psychol, 2020), and maternal diet (Emond, J of Nutr, 2018).

5. I have been extensively involved in the New Hampshire Birth Cohort Study, a longitudinal pregnancy cohort based in a rural setting. Through working as an investigator in that cohort, I have researched exposure to environmental contaminants, such as arsenic, and how they influence child growth. A major contribution that I’ve made in this area is identifying that dietary sources, such as rice, substantially contribute to arsenic exposure in populations with low levels of arsenic in ground water (Gilbert-Diamond, *PNAS*, 2011). I have also demonstrated that low-dose arsenic exposure affects child growth *in utero* and during the first year of life. I am particularly interested in growth during infancy as it relates to later risk of developing obesity.


**Complete List of Published Work in MyBibliography:**