A Psychobiobehavioral Expansion of the Cyclic Obesity/Weight-Based Stigma (COBWEBS) Model in Adolescents with Overweight and Obesity

by

Tiwaloluwa A. Ajibewa

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Kinesiology)
in the University of Michigan
2021

Doctoral Committee:

Associate Professor Rebecca E. Hasson, Chair
Associate Professor Alison L. Miller
Professor Leah E. Robinson
Assistant Professor Kendrin R. Sonneville
Assistant Professor Claudia Toledo-Corral, Cal State-Northridge

Tiwaloluwa A. Ajibewa

tajib@umich.edu

ORCID iD: 0000-0001-5756-5975

Dedication

I would like to dedicate this dissertation to my parents, John and Stella Ajibewa. Thank you for your constant encouragement, love, prayers, and support. Your unwavering care and your numerous sacrifices are a shining example for me, my siblings, and to everyone who has gotten a chance to know you.

Acknowledgements

This dissertation work is partly supported by the University of Michigan's Rackham Graduate School Research Grant, Rackham Merit Fellowship, the School of Public Health Momentum Center, and the American Diabetes Association grant given to Dr. Rebecca Hasson.

I would like to acknowledge and thank my dissertation committee: Dr. Rebecca Hasson, Dr. Alison Miller, Dr. Leah Robinson, Dr. Kendrin Sonneville, and Dr. Claudia Toledo-Corral for their time, expertise, and invaluable guidance in putting together this dissertation. This dissertation could not have been completed without each of you and your amazing mentorship. Special thanks to my advisor and graduate mentor Dr. Rebecca Hasson for giving me the opportunity to conduct research as a graduate student in the Childhood Disparities Research Laboratory over these past five years. The opportunity to carry out my research interest with your support and trust was priceless. In addition, I particularly appreciate your patience and encouragement during the good times and the challenging periods of my PhD journey. I am thankful for your consistent motivation and the fact that I could always count on you to look out for my best interest.

I am grateful to the current members and alumni/a of the Childhood Disparities Research Laboratory, who were all instrumental in collecting data for this dissertation. The Childhood Disparities Research Laboratory has been a family for me over the past five years, and I deeply appreciate each member and alumni/a of the lab that I have gotten the chance to know and work with over the past few years. In addition, I am grateful to the School of Kinesiology faculty, staff members, and colleagues, particularly my cohort-mates Dr. Zhanjia Zhang, and Dr. Erica Twardzik. Starting the PhD program with you two was a blessing. Both of you set the mark for being exceptional graduate students, great researchers, and even better friends. To my colleague, friend, and fellow CDRL member Lexie Beemer as well, thank you for being such a wonderful friend and colleague. Your work ethic, intelligence, willing to help attitude, meticulous notetaking, positive attitude, and so much more make it my pleasure to call you my friend and colleague. Thank you so much for making my time in the Childhood Disparities Research Laboratory such a delight. And to all the participants, caregivers, and family members who participated in the studies that comprise this dissertation, thank you!

I would like to also acknowledge my family, friends, and mentors outside of the School of Kinesiology. I want to first and foremost appreciate my parents, John and Stella. I've dedicated this dissertation to you both, because of your unwavering support and prayers throughout my academic journey. Thank you both for your encouragement and love. To my siblings Bayo and Tolu Ajibewa. You two have been my closest friends, and biggest allies. Role models to me every step of the way and in every facet of life. To my fiancé Dr. Janelle Goodwill, thank you for your care and support. For inspiring me with the manner by which you balance all your responsibilities, for your drive to help the marginalized, for your continual encouragement and prayers, and for making my grad school experience beautiful. To my various housemates/roommates and friends including Daniel Vignon, Dr. Jubilee Adeoye, Conner VanderBeek, thank you for the late night discussions, basketball and running workouts, and for always brightening my day with jokes. To my RCF (James Akinola, Dr. Samuel Ayinde, Ronke Olawale, and others), H2O (Andrew Muyanja, Joyce Lee, Daniel Bai, Chiamaka, and others), and Vineyard (Nosa, LaTricia, Mr. Obi, and others) church family, thank you for your encouragement, prayers, and time spent together studying the word. I am also thankful for Donnell and Maria Wyche, Kevin and Maria Kummer, for their prayers, wise counsel, and encouragement as spiritual mentors in my life. To Dr. Joseph Alia, Dr. Rich Hardy, and Dr. Sarah Buchanan, my undergraduate professors and mentors at the University of Minnesota-Morris who all gave me an opportunity to do research with and alongside them during my time at Morris; thank you all for allowing me to pursuit research interests across the fields of Chemistry, French, and Athletic training. To Grace Adofoli and Ms. Njia Franklin-Porter, both mentors of mine who with their guidance and wisdom informed me about the possibility of going to grad school and of doing research; without your guidance, I would have never even thought of having a career that revolves around research. Last but most importantly, I would like to thank my Lord and Savior Jesus Christ, without whom none of this would have been possible—to Him alone be all the glory.

Table of Contents

List of Tables

List of Figures

Abstract

The Cyclic Obesity/Weight-Based Stigma (COBWEBS) model is an original, theory-based framework that describes how stress from obesity- and weight-stigma promotes weight gain among individuals of higher weight. Though the COBWEBS model advances our understanding of how stress promotes weight gain, gaps within the model related to physical activity (PA), metabolic risk, and the psychological constructs underlying PA and eating behaviors remain. Using primary and secondary data analysis, this dissertation expands the COBWEBS framework to address these gaps.

Study 1 of this dissertation examined the association between stress, objectively measured PA, and the moderating effect of PA enjoyment among adolescents with overweight/obesity. Greater stress was found to be associated with lower enjoyment of PA. However, stress was not associated with objectively measured PA, nor was PA enjoyment a significant moderator. Findings from study 1 suggests that increased stress may exert more immediate effects on factors preceding PA, which may ultimately lead to decreased participation in PA among adolescents with overweight/obesity.

Study 2 of this dissertation examined the association between acute stress, caloric intake, and the moderating effect of disordered eating behavior in adolescents with overweight/obesity. Acute daily stressors were associated with greater caloric intake. Disordered eating behavior moderated the association between acute stress frequency and caloric intake, but not between acute stress sum and caloric intake. Findings from study 2 suggests that greater exposure to acute

daily stressors may increase food intake in adolescents with overweight/obesity, with greater susceptibility among those with high levels of disordered eating.

Study 3 of this dissertation examined biobehavioral pathways linking stress to metabolic risk in adolescents with overweight/obesity. Stress was not association with metabolic risk, nor was PA, caloric intake, or cortisol significant mediators. However, racial differences in metabolic risk was observed. Specifically, higher levels of stress was associated with lower insulin sensitivity (Si) among Black adolescents, whereas the inverse association was observed for White adolescents. Study 3 findings suggest that stress may uniquely influence racial differences in metabolic risk among Black and White adolescents with overweight/obesity.

Study 4 of this dissertation examined the association between weight stigma exposure and health-promoting intentions and behaviors among older adolescents. It was observed that prior weight stigma experiences were associated with PA avoidance. However, prior weight stigma experiences was not associated with positive PA or eating intentions, nor were there any differences in positive PA intentions, PA avoidance, or eating intentions and behaviors among participants. Study 4 findings indicate that greater weight stigma experiences may lead to higher avoidance of PA.

Together, findings from these studies suggests that psychological stress affects behavioral antecedents such as physical activity enjoyment, and that greater susceptibility to the effects of stress on eating behavior is pronounced among those with disordered eating behavior. Additionally, the stressor of weight stigma leads to the habitual avoidance of engaging in PA. Upstream, preventative approaches addressing these behavioral precursors may help interrupt the cycle of stress and weight gain among adolescents with overweight/obesity.

Chapter 1

Introduction

Childhood obesity is a national public health epidemic. Presently, obesity is estimated to affect nearly twenty-one percent of adolescents between the ages of twelve to nineteen years of age[1]. Obesity is known to be associated with several major health outcomes such as type 2 diabetes mellitus, hypertension, depression, anxiety, in addition to unhealthy weight loss practices such as dieting[2–4]. Obesity during childhood and adolescence is particularly concerning due to the increased risk that earlier acquisition of obesity presents with regards to many of the aforementioned health outcomes. Childhood obesity is also concerning given that adolescence is a critical developmental stage, where key health behaviors are developed, and where excess adiposity and its comorbid conditions can begin and then track into adulthood[5].

An important feature of adolescence that may have major biological and behavioral implications for obesity is stress exposure. Adolescence marks a critical period for increased risk for psychological stress, stemming from several physical, psychological, and social factors that change during the adolescent period[6,7]. Psychological stress can be experienced and appraised as threatening when the resources to deal with a demand (i.e. the stressor) are either unavailable or insufficient[8]. Adolescents with overweight and obesity in particular, experience greater biological stress responses (e.g. stress reactivity), increased cardiovascular risk (e.g. hypertension), and encounter greater stress exposure as a result of their weight (e.g. weight stigma and weight-related teasing) in comparison to their healthy weight counterparts[2,9,10]. One salient source of stress for adults and adolescents with overweight and obesity is weight discrimination. Weight discrimination has been shown to be extremely common in the United States and Canada, and stems in part from the beliefs that those with overweight and obesity are “lazy”, “lack self-discipline”, and “have poor will-power” often resulting in a lack of compliance with weight loss plans[11]. Weight discrimination manifests in different forms including but not limited to: bias, verbal and physical assaults, along with negative attitudes and stereotypes[12,13]. Furthermore, weight discrimination occurs across a multitude of settings including employment settings[14], health care settings[15], and schools[16–18]—a setting where children and adolescents spend a majority of their waking hours.

The increased weight discrimination experienced by individuals with overweight and obesity, naturally affects day to day activity, including health-promoting behaviors and the psychobehavioral constructs underlying such behaviors. This increased stress exposure resulting from weight discrimination has been found to be associated with health behaviors such as increased food intake[19]. Consequently, increased psychological stress exposure originating from weight discrimination along with increased stress responses in adolescents with overweight and obesity, may work together through multiple biological, psychological, and behavioral mechanisms to then increase weight gain. Emerging lines of evidence from longitudinal studies within the weight stigma literature point to the fact that weight related teasing and pressures during adolescence lead to weight gain in young adulthood[20–22].

Conceptual Framework: Cyclic Obesity/Weight-Based Stigma (COBWEBS) model

COBWEBS is a stigma-based obesity model[9] that proposes that individuals who are perceived to carry excess weight (i.e. overweight or obese), face greater obesity/weight-based stigma. It is an original, theory based framework that details how stress—particularly the stressor of weight discrimination—leads to a continuous cycle of weight gain amongst individuals experiencing weight discrimination and stigma. This pervasive stressor effectively leads to the promotion of weight gain via increased eating (i.e. stress-induced comfort eating) and increased release of the stress hormone cortisol, together acting to increase abdominal fat storage and weight gain over time. The presence of weight discrimination faced by those of higher weight status, leads to a positive feedback loop, with greater weight begetting more weight, further challenging, and disenabling those with overweight or obesity from successfully losing weight. The COBWEBS model is a unique model that advances the general model of stress and weight gain through three main contributions: it i) identifies weight stigma as a specific source of stress that has its own health damaging consequences; ii) highlights that normal coping responses are likely not available in the context of weight stigma; and iii) describes the cyclic nature of weight stigma[9].

Figure 1.1 Cyclic Obesity/Weight-Based Stigma (COBWEBS) model. A. Janet Tomiyama, Appetite, 2014.

It is important to note that psychological stress exposure in adolescents with overweight and obesity may not only lead to more weight gain, but may also accelerate the development of metabolic risk factors independently of weight gain. Psychological stress arising from weight-based stigmatization has been previously implicated with adverse metabolic risk profiles including but not limited to impaired insulin sensitivity and increased abdominal adiposity in adolescents. Specifically, investigators examining the association between weight-related pressures to be thin in adolescents with healthy weight and those with overweight/obesity observed that greater pressure to be thin was associated with greater fasting insulin and decreased insulin sensitivity[23]. Others studies within the adult stress literature have also noted that psychological stress is associated with increased metabolic risk[24,25]. The impact of a stressor such as weight discrimination on metabolic risk may go through a biological pathway involving cortisol. Evidence from studies such as the one conducted by Tomiyama and colleagues, where weight stigma experiences were associated with cortisol awakening response in adult women with overweight and obesity[26], along with others[27,28], demonstrate that weight discrimination and weight stigmatizing experiences may dysregulate the biological stress response [the hypothalamic pituitary adrenal (HPA) axis], which in turn may lead to increased risk of metabolic disease. Therefore, it is crucial to determine through which pathways psychological stress influences health-promoting behaviors during adolescence, in order to find ways to alleviate its long-term ramifications on metabolic health outcomes.

Statement of Problem

The COBWEBS model is an important model in considering how the pervasive phenomenon of weight discrimination perpetuates weight gain among individuals. Building upon the COBWEBS framework will not only help researchers in understanding the impact of weight discrimination, but will further enhance our understanding of the impact of psychological stress on weight gain, and the psychological, biological, and behavioral factors that underpin risk for metabolic disease. The COBWEBS model has provided a remarkable foundation for this investigation, however, as with all models, gaps remain, particularly regarding physical activity, physical activity enjoyment, disordered eating behavior (a deviation from a normal, socially acceptable, health maintenance-focused approach to food such as extreme caloric restriction, unhealthy dieting through skipping meals[29]), and metabolic risk factors that may precede or follow obesity. This dissertation fills in these gaps by expanding the COBWEBS framework through three main facets. First, this dissertation expands the COBWEBS model by investigating the psychobehavioral constructs that may underlie eating and physical activity behavior. Secondly, this dissertation expands the COBWEBS model by including physical activity, an important factor when considering energy balance within the framework. Furthermore, through the addition and consideration of risk factors, this dissertation also expands the COBWEBS framework by examining metabolic risk factors that may precede further weight gain in adolescents with overweight and obesity. Lastly, with the examination of the impact of weight stigma on future exercise and eating intentions, this dissertation not only further confirms the influence of weight stigma as a salient stressor that affects health behavior intentions, but also provides data for longitudinal studies to do the same.

In summary, the expansion of the COBWEBS model further advances our understanding of the impact of psychological stress (including that of weight stigma) on physical activity, as well as on the psychobehavioral factors underlying physical activity and eating behaviors, and metabolic risk. The findings from this dissertation improves our awareness of how stress acts on weight gain particularly during the critical period of adolescence.

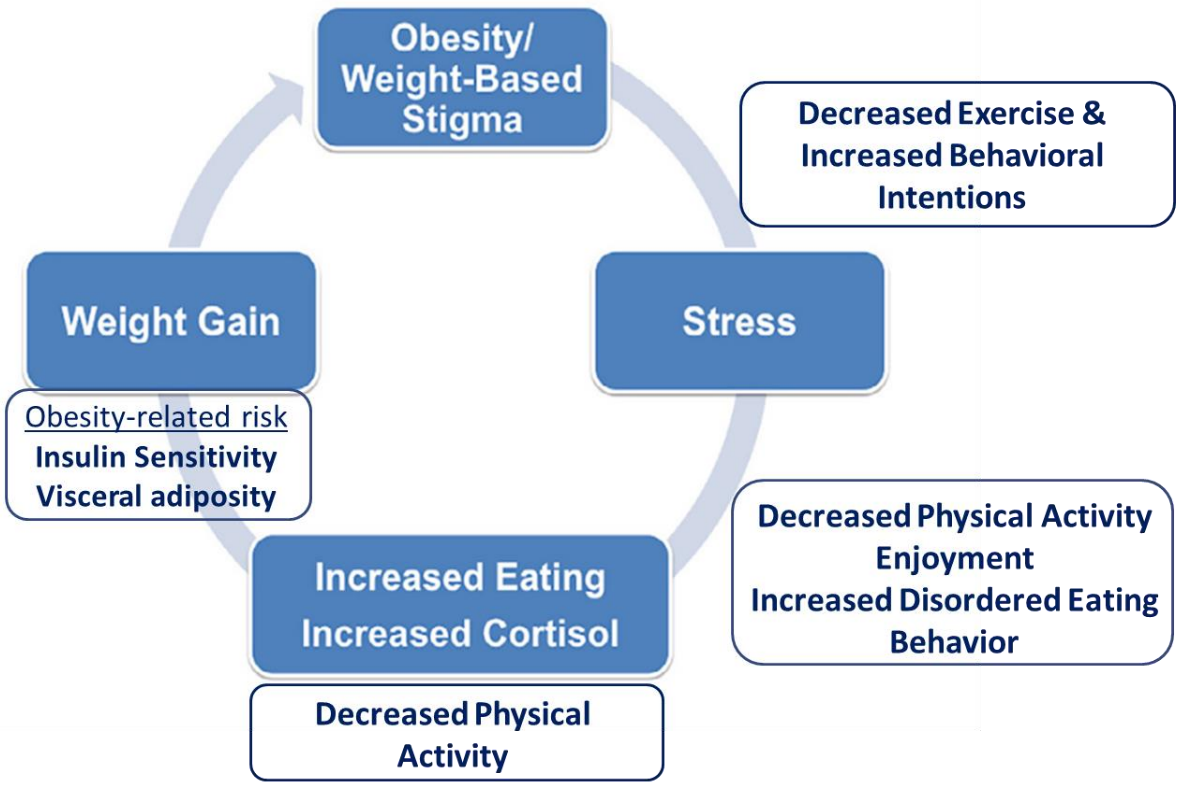

Figure 1.2 Expanded COBWEBS model, including psychobehavioral constructs, physical activity, and a metabolic risk factor (insulin resistance via insulin sensitivity).

Purpose and Aims

Study 1 Aims: The aim of the first study was twofold: (1) to determine the cross-sectional associations of psychological stress on physical activity enjoyment and physical activity participation [MVPA, and total physical activity (TPA)] in adolescents with overweight/obesity; and (2) to examine the potential moderating effect of physical activity enjoyment on the association between stress and physical activity participation.

Study 2 Aims: The aim of the second study was to examine the associations between acute daily stress dimensions (frequency, sum) and food intake in adolescents with overweight/obesity, and to explore the potential moderating effect of disordered eating behaviors on these associations.

Study 3 Aims: The aim of the third study was to examine racial differences in the association between psychological stress and metabolic outcomes—insulin sensitivity (S_i), disposition index (DI), and acute insulin response to intravenous glucose (AIR_g), among non-Hispanic White and non-Hispanic Black adolescents with overweight and obesity.

Study 4 Aims: The aim of the fourth study was two-fold: (1) to explore cross-sectional associations between prior weight stigma experiences, PA intentions and behaviors; and (2) to examine the acute effects of a weight stigma exposure on PA intentions and behaviors among older adolescents/young undergraduate college students.

Hypotheses

Hypothesis 1a: It was hypothesized that increased psychological stress would be associated with decreased physical activity enjoyment and physical activity participation.

Hypothesis 1b: Secondly, it was hypothesized that the associations between psychological stress, MVPA, and TPA participation would be moderated by enjoyment of physical activity, where greater participation in physical activity (MVPA and TPA) would be seen among high stress youth with higher enjoyment, compared with those with lower enjoyment.

Hypothesis 2a: The primary hypothesis was that higher acute daily stress would be associated with higher daily caloric intake.

Hypothesis 2b: It was also hypothesized that the associations between acute daily stress and daily caloric intake would be modified by disordered eating behavior.

Hypothesis 3: It was hypothesized that Black race and increased psychological stress would be associated with decreased S_i, DI, and increased AIR_g among sample participants, and that the association between psychological stress and metabolic outcomes would be modified by race.

Hypothesis 4a: It was hypothesized that higher levels of prior weight stigma experiences would be associated with lower physical activity intentions and behaviors.

Hypothesis 4b: It was also hypothesized that an acute exposure to weight stigma would be associated with short-term declines in physical activity intentions and behaviors.

Significance

Physical activity is a major component of energy balance, and a key factor in weight maintenance and metabolic health. By understanding the mechanistic pathways by which psychological stress affects physical activity behavior and the psychobehavioral constructs that affects one's drive to engage in physical activity, the influence of psychological stress—especially that of weight discrimination, may in turn be diminished or otherwise eliminated in individuals with overweight and obesity. This disruption to the feedforward cycle of weight stigma during the critical period of adolescence, may thereby allow for public health interventionists and other stakeholders to more effectively reduce weight gain and as a result aid in improving metabolic risk prior to adulthood. Importantly, an expansion of the COBWEBS framework examining psychobiobehavioral effects of psychological stress not only on eating behavior, but also on physical activity behavior along with central psychological constructs underlying these behaviors including physical activity enjoyment and disordered eating behavior, will grant a more comprehensive picture of how stress contributes to weight gain in adolescents with overweight/obesity. Additionally, these analyses will enable us to identify which adolescents are at increased risk of changes in their health behavior during periods of heightened psychological stress.

Limitations

Overall: This dissertation incorporated four studies, three of which are secondary analyses of existing data collected from the Stress, Obesity, and Diabetes in Adolescents (SODA) study and the Health and Culture Project (HCP) study conducted in the Childhood Disparities Research Laboratory. As with many studies utilizing secondary analysis of previously collected data, the limitation of using data that was not primarily collected to address the particular research questions or to test the specific hypotheses of this dissertation is an important limitation to consider.

Study design: The cross-sectional study design of studies 1-3 limited inferences on directionality among predictor and outcome variables, and did not allow for causality to be determined. However, the comprehensive analysis of validated and objective biological (salivary cortisol) and behavioral (physical activity measured via accelerometry) variables strengthened the study designs.

Generalizability: The studies included within this dissertation primarily involved a sample of Black/African-American and White adolescents with overweight and obesity in the United States. The study findings (particularly from studies 1-3) may not be generalizable or have limited generalizability to other racial groups as well as to adolescents of lower weight status. Additionally, participants in study 4 were older adolescents and college students from the University of Michigan, which also limited generalizability to others outside of the university environment. Furthermore, data collection for studies 1-3 was coordinated within a laboratory and clinical setting and therefore could not be readily generalized to other settings. Nonetheless, studies 1-3 were conducted within a well-controlled laboratory environment, and the use of two racial groups allowed for greater racial diversity, in turn providing additional insight into the effect of psychological stress on metabolic disease risk in adolescents with overweight and obesity across Black and White racial groups.

Online data collection: Study 4 utilized both in-person and online data collection. As it relates to online data collection, because of the lack of the physical presence of a research assistant, participants may have been more inclined to rush through questions and or may have been unable to ask clarifying questions that were confusing or unclear during the none in-person visits. Yet, the use of online data collection allowed for greater ease of data collection and greater reach to participants who may have otherwise be unable or unwilling to come to the laboratory. Additionally, the use of online data collection reduced the confounding influence of the research assistant(s)'s social identities (race, gender, weight status) from interfering with study findings, which could have been particularly important with regard to weight stigma.

Food frequency questionnaire as measure of caloric intake: Studies included within this dissertation used the food frequency questionnaire (FFQ) as a measure of total food intake. Studies 2-3 used the self-reported Youth/Adolescents Questionnaire with food intake within the past year, whereas study 4 used a 24-hr dietary recall. Given the self-reported nature of both questionnaires, they may not have been the most reliable measure of current food intake among study participants. Furthermore, a common occurrence among populations with obesity is an under-reporting of total caloric intake[30]. Nevertheless, both methods have been used among adolescents and the Youth/Adolescent Questionnaire has been previously validated and calibrated as a semi-quantitative FFQ designed for adolescents[31].

Weight stigma measure: The lack of a weight stigma measure in studies 1-3 did not allow for the examination of a direct association between the stressor of weight stigma with behavioral and metabolic health measures. Nevertheless, prior studies have demonstrated the association between weight stigma and psychological stress[9], and thus, a connection between these measures has been established. Furthermore, the lack of a weight stigma measure in studies 1-3 was precisely the reason why study 4 was conducted in a sample of older adolescents and college-aged students.

Age difference between studies: Adolescents between the ages of 13-19 were recruited for studies 1-3 (ages 13-19 included from HCP, and ages 14-19 from the SODA study). According to the American Academy of Pediatrics this group of adolescents fall primarily within what is termed the middle adolescent years, while those who were recruited for study 4 (ages 18-21), fall within late adolescence[32]. Though the adolescents may not be in the same adolescent bracket, both groups are still considered adolescents, and evidence suggest the adolescent brain has not typically reached the level of functioning seen in adulthood until the early twenties[32,33]. Data collected between studies 1-3 was not necessarily generalizable to study 4. Nevertheless, college age students were used for study 4 to allow for a tighter sample demographic (improving internal validity), but also primarily due to the fact that the videos used for the weight discriminatory exposure have been previously only used in adult samples[28]. Additionally, the uniqueness of this transitional period into early adulthood, where there is greater autonomy in decision making[34] which may impact daily schedules, food choices, and exercise behaviors, allowed for the researcher to examine the influence of the weight discriminatory exposure on the individual's behavior without the influence of parents which may be more salient in younger adolescents who are still likely living at home.

Dual Energy X-ray Absorptiometry for visceral measurements: Dual Energy X-ray Absorptiometry (DEXA) is a gold standard technique for measuring body composition in both research and clinical practice[35]. Although DEXA is a well-established technique for measuring body composition, it is not however, capable of differentiating between visceral adipose tissue and that of subcutaneous adipose tissue[36]. Because of this, total body fat percentage was used in order to capture whole body fat depots (including visceral, subcutaneous, and muscle fat depots).

Puberty measurement: The pubertal development of all participants in studies 1-3 were assessed via self-report using the Pubertal Development Scale[37]. Although this is a self-reported measure, this scale has been previously found to be a valid and reliable measure of pubertal development in adolescents longitudinally[37], and has been used widely across studies conducted in adolescents..

Definitions of Terms

Overweight: In accordance with the Centers for Disease Control and Prevention (CDC) guidelines, an individual with a body mass index (BMI) greater or equal to 25 and less than 30 kg/m^2 is considered an individual with overweight. In addition to BMI, BMI for age and sex percentile of greater or equal to 85th percentile and less than 95th percentile (measured using BMI, age, and biological sex) in agreement with the CDC growth charts for children and teenagers is considered overweight[38].

Obese/Obesity: In accordance with the Centers for Disease Control and Prevention (CDC) guidelines from the year 2000, an individual with a body mass index (BMI) equal to or greater than 30 kg/m^2 is considered an individual with obesity. In addition to BMI, BMI for age and sex percentile of 95th or greater percentile (measured using BMI, age, and biological sex) in agreement with the CDC growth charts for children and teenagers is considered obese[38].

Psychological stress: A construct classically defined as occurring when an individual's demands or threats exceed the available resources by which they can meet these demands or mitigate the threats[39]. Psychological stress was measured within this dissertation using either the Perceived Stress Scale or the Daily Stress Inventory. The Perceived Stress Scale is a commonly used scale that assessed each adolescent's perception of their ability to cope with stressors in their lives over the past month[40]. Whereas the Daily Stress Inventory is a measure of stress designed to assess daily sources and individualized impact of stressful events[41].

Adolescence: Adolescence is a developmental period that encompasses biological changes (puberty) and social-role transitions between childhood and adulthood[42]. Though the American Academy of Pediatrics defines adolescence starting at the age of 11 to age 21, there is no true consensus of what age ranges make up adolescence. Others including the U.S. Department of Health, the Food and Drug Administration define adolescence between the ages of 12-21, the World Health Organization defines it as being between the ages of 10-19 years [43]. However, it is generally agreed upon that puberty marks the start of adolescence, though the key social-role transitions that have historically signaled the end of adolescence along with continued brain development make age 21 a reasonable upper limit[32].

Weight discrimination: Discrimination is defined as the unequal treatment of individuals due to their membership to a particularly group, and in the case of weight discrimination, it is purposeful unequal treatment of individuals due to their weight status[12]. For the purposes of this dissertation, the terms weight stigma and weight stigmatization were used in the context of weight related devaluation and discrimination. Weight stigma experiences were measured using the brief 10-item form of the stigmatizing situations inventory that assesses whether, and how often, individuals have experienced weight-stigmatizing situations[44]. Additionally, an acute weight stigmatizing exposure was used via a weight-stigmatizing video.

Enjoyment: Physical activity enjoyment is a positive affective response to sports or exercise experience that reflects feelings and perceptions such as pleasure, liking, and experienced fun[45]. Physical activity enjoyment was measured using the Physical Activity Enjoyment Scale (PACES)[46] for this dissertation.

Disordered eating: Disordered eating behavior is defined as a deviation from a normal, socially acceptable and health maintenance-focused approach to food, that often manifests in a multitude of ways including extreme caloric restriction, unhealthy dieting, binge eating cycles, and compensatory behaviors such as laxative and diet pill usage for weight maintenance[29,47]. Disordered eating behaviors was measured via the self-reported Eating Attitudes Test (EAT-26) questionnaire[48].

Physical Activity: Physical activity is any bodily movement that is produced by skeletal muscles that results in energy expenditure above resting metabolic rate[49]. It includes exercise, sports, and physical activities done as part of daily living, occupation, leisure, and active transport.

Moderate-to-vigorous physical activity (MVPA): Moderate intensity activity expends between 3.5- 7 kcal/min, while vigorous intensity activity expends more than 7 kcal/min. Together moderate-to-vigorous physical activity (MVPA) is any activity that combines any combination of moderate or vigorous activity[50].

Caloric intake: Caloric intake is the amount of energy consumed via food and beverage, and is measured using the unit of energy termed calorie. Calories measure the energy in food, and the energy produced, stored, and utilized by living organisms[51]. Caloric intake was measured using the 2012 Adolescent Food Frequency Questionnaire for studies 1-3, and the automated self-administered 24-hr (ASA24) dietary recall for study 4.

Cortisol: Cortisol, a steroid hormone, is the primary hormone/agent of the neuroendocrine stress response, and an important regulator of inflammatory and metabolic activities[52]. Cortisol follows a diurnal pattern wherein circulating levels peak shortly after an individual wakes up, and decline gradually throughout the rest of the day[52]. Dysregulation of the diurnal cortisol pattern (e.g. blunting or heightening of peak cortisol) is believed to be part of the mechanistic pathway [hypothalamic-pituitary-adrenal (HPA) axis] by which chronic stress affects physical health[52]. Basal and diurnal cortisol patterns include but are not limited to distinct patterns such as the baseline awakening cortisol, cortisol awakening response (cortisol release between awakening at ~30min post waking), peak cortisol (~30min post awakening), and diurnal cortisol output. An area under the curve with respect to ground (AUC) calculation was used to estimate cortisol output for these measures where appropriate.

Visceral adiposity: Visceral adipose tissue is fat originating and present around the abdominal viscera, specifically in the mesentery and omentum peritoneum[53] is known to be highly sensitive to cortisol.

Insulin resistance: Insulin resistance (a requisite precursor for developing type 2 diabetes) is a metabolic condition characterized by impairment or otherwise attenuated biological response to “normal” concentrations of insulin produced by the pancreas by cells of the muscle, fat and liver[54,55]. Decreased insulin sensitivity equates to increased insulin resistance. Insulin resistance was measured using insulin sensitivity derived from physiological modeling utilizing plasma samples during intravenous glucose tolerance test (IVGTT).

Insulin sensitivity: Insulin sensitivity is defined as how sensitive cells of the muscle, fat, and liver are to insulin-mediated glucose disposal in the presence of insulin[55]. With decreased sensitivity, there is a compensatory increase in levels of insulin, with long-term implications including the development of type 2 diabetes mellitus. Decreased insulin sensitivity equates to increased insulin resistance. Insulin sensitivity was derived from physiological modeling utilizing plasma samples during an intravenous glucose tolerance test (IVGTT).

Organization of Dissertation

This dissertation consists of six chapters. Chapters 2 through 5 represent full-length manuscripts that are either published or accepted for publication in peer-reviewed academic journals, or are being prepared for submission in peer-reviewed journals. Chapter 2 examined the cross-sectional association between psychological stress, physical activity enjoyment and engagement, as well as the moderating effects of physical activity enjoyment on the association between stress and physical activity engagement among adolescents with overweight and obesity. Similarly, Chapter 3, examined the association between psychological stress and caloric intake as well as the moderating effect of disordered eating behavior. Chapter 4 explored racial differences in the association between psychological stress and metabolic outcomes among Black and White adolescents with overweight and obesity. Chapter 5 examined the acute effects of a weight stigma exposure on physical activity participation and intentions, and assessed the association between prior weight stigma experiences, physical activity intentions, and physical activity participation among young undergraduate college students. Finally, in Chapter 6, a summary and discussion of the main findings, significance, and guidance for future research is seen.

References

1. Ogden CL, Carroll MD, Lawman HG, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *Jama*. 2016;315(21):2292-2299.

2. Bridger T. Childhood obesity and cardiovascular disease. *Paediatrics & Child Health*. 2009;14(3):177-182. doi:10.1093/pch/14.3.177

3. Goldschmidt AB, Aspen VP, Sinton MM, Tanofsky-Kraff M, Wilfley DE. Disordered eating attitudes and behaviors in overweight youth. *Obesity*. 2008;16(2):257-264.

4. Rankin J, Matthews L, Cobley S, et al. Psychological consequences of childhood obesity: psychiatric comorbidity and prevention. *Adolesc Health Med Ther*. 2016;7:125-146. doi:10.2147/AHMT.S101631

5. Singh AS, Mulder C, Twisk JWR, Mechelen WV, Chinapaw MJM. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obesity Reviews*. 2008;9(5):474-488. doi:https://doi.org/10.1111/j.1467-789X.2008.00475.x

6. Casey BJ, Jones RM, Levita L, et al. The storm and stress of adolescence: Insights from human imaging and mouse genetics. *Developmental Psychobiology*. 2010;52(3):225-235. doi:https://doi.org/10.1002/dev.20447

7. Wiium N, Breivik K, Wold B. Growth Trajectories of Health Behaviors from Adolescence through Young Adulthood. *International Journal of Environmental Research and Public Health*. 2015;12(11):13711-13729. doi:10.3390/ijerph121113711

8. American Psychological Association. APA Working Group on Stress and Health Disparities.(2017). *Stress and health disparities: Contexts, mechanisms, and interventions among racial/ethnic minority and low-socioeconomic status populations Retrieved from http://www apa org/pi/health-disparities/resources/stress-report aspx*.

9. Tomiyama AJ. Weight stigma is stressful. A review of evidence for the Cyclic Obesity/Weight-Based Stigma model. *Appetite*. 2014;82:8-15. doi:10.1016/j.appet.2014.06.108

10. Verdejo-Garcia A, Moreno-Padilla M, Garcia-Rios MC, et al. Social Stress Increases Cortisol and Hampers Attention in Adolescents with Excess Weight. *PLoS One*. 2015;10(4). doi:10.1371/journal.pone.0123565

11. Puhl RM, Heuer CA. Obesity Stigma: Important Considerations for Public Health. *Am J Public Health*. 2010;100(6):1019-1028. doi:10.2105/AJPH.2009.159491

12. Andreyeva T, Puhl RM, Brownell KD. Changes in Perceived Weight Discrimination Among Americans, 1995–1996 Through 2004–2006. *Obesity*. 2008;16(5):1129-1134. doi:https://doi.org/10.1038/oby.2008.35

13. Puhl RM, Luedicke J, Heuer C. Weight-Based Victimization Toward Overweight Adolescents: Observations and Reactions of Peers. *Journal of School Health*. 2011;81(11):696-703. doi:https://doi.org/10.1111/j.1746-1561.2011.00646.x

14. Roehling MV. Weight-Based Discrimination in Employment: Psychological and Legal Aspects. *Personnel Psychology*. 1999;52(4):969-1016. doi:https://doi.org/10.1111/j.1744-6570.1999.tb00186.x

15. Teachman BA, Brownell KD. Implicit anti-fat bias among health professionals: is anyone immune? *Int J Obes Relat Metab Disord*. 2001;25(10):1525-1531. doi:10.1038/sj.ijo.0801745

16. Greenleaf C, Weiller K. Perceptions of Youth Obesity among Physical Educators. *Soc Psychol Educ*. 2005;8(4):407-423. doi:10.1007/s11218-005-0662-9

17. Neumark-Sztainer D, Story M, Harris T. Beliefs and Attitudes about Obesity among Teachers and School Health Care Providers Working with Adolescents. *Journal of Nutrition Education*. 1999;31(1):3-9. doi:10.1016/S0022-3182(99)70378-X

18. O'Brien KS, Hunter JA, Banks M. Implicit anti-fat bias in physical educators: physical attributes, ideology and socialization. *International journal of obesity*. 2007;31(2):308-314.

19. Puhl RM, Luedicke J. Weight-Based Victimization Among Adolescents in the School Setting: Emotional Reactions and Coping Behaviors. *J Youth Adolescence*. 2012;41(1):27-40. doi:10.1007/s10964-011-9713-z

20. Puhl RM, Wall MM, Chen C, Bryn Austin S, Eisenberg ME, Neumark-Sztainer D. Experiences of weight teasing in adolescence and weight-related outcomes in adulthood: A 15-year longitudinal study. *Preventive Medicine*. 2017;100:173-179. doi:10.1016/j.ypmed.2017.04.023

21. Hunger JM, Tomiyama AJ. Weight Labeling and Obesity: A Longitudinal Study of Girls Aged 10 to 19 Years. *JAMA Pediatrics*. 2014;168(6):579-580. doi:10.1001/jamapediatrics.2014.122

22. Suelter CS, Schvey N, Kelly NR, et al. Relationship of pressure to be thin with gains in body weight and fat mass in adolescents. *Pediatric Obesity*. 2018;13(1):14-22. doi:https://doi.org/10.1111/ijpo.12179

23. Schvey NA, Shomaker LB, Kelly NR, et al. Pressure To Be Thin and Insulin Sensitivity Among Adolescents. *Journal of Adolescent Health*. 2016;58(1):104-110. doi:10.1016/j.jadohealth.2015.09.010

24. Heraclides A, Chandola T, Witte DR, Brunner EJ. Psychosocial Stress at Work Doubles the Risk of Type 2 Diabetes in Middle-Aged Women: Evidence from the Whitehall II Study. *Diabetes Care*. 2009;32(12):2230-2235. doi:10.2337/dc09-0132

25. Yan Y-X, Xiao H-B, Wang S-S, et al. Investigation of the relationship between chronic stress and insulin resistance in a Chinese population. *Journal of epidemiology*. 2016;26(7):355-360.

26. Tomiyama AJ, Epel ES, McClatchey TM, et al. Associations of Weight Stigma With Cortisol and Oxidative Stress Independent of Adiposity. *Health Psychol*. 2014;33(8):862-867. doi:10.1037/hea0000107

27. Himmelstein MS, Belsky ACI, Tomiyama AJ. The weight of stigma: Cortisol reactivity to manipulated weight stigma. *Obesity*. 2015;23(2):368-374. doi:https://doi.org/10.1002/oby.20959

28. Schvey NA, Puhl RM, Brownell KD. The Stress of Stigma: Exploring the Effect of Weight Stigma on Cortisol Reactivity. *Psychosomatic Medicine*. 2014;76(2):156-162. doi:10.1097/PSY.0000000000000031

29. Pereira RF, Alvarenga M. Disordered Eating: Identifying, Treating, Preventing, and Differentiating It From Eating Disorders. *Diabetes Spectrum*. 2007;20(3):141-148. doi:10.2337/diaspect.20.3.141

30. Johnson RK. Dietary Intake—How Do We Measure What People Are Really Eating? *Obesity Research*. 2002;10(S11):63S-68S. doi:https://doi.org/10.1038/oby.2002.192

31. Araujo MC, Yokoo EM, Pereira RA. Validation and Calibration of a Semiquantitative Food Frequency Questionnaire Designed for Adolescents. *Journal of the American Dietetic Association*. 2010;110(8):1170-1177. doi:10.1016/j.jada.2010.05.008

32. Hardin AP, Hackell JM, Medicine C on P and A. Age Limit of Pediatrics. *Pediatrics*. 2017;140(3). doi:10.1542/peds.2017-2151

33. Sawyer SM, Azzopardi PS, Wickremarathne D, Patton GC. The age of adolescence. *The Lancet Child & Adolescent Health*. 2018;2(3):223-228. doi:10.1016/S2352-4642(18)30022-1

34. Nelson MC, Story M, Larson NI, Neumark-Sztainer D, Lytle LA. Emerging Adulthood and College-aged Youth: An Overlooked Age for Weight-related Behavior Change. *Obesity*. 2008;16(10):2205-2211. doi:https://doi.org/10.1038/oby.2008.365

35. Bazzocchi A, Diano D, Ponti F, et al. A 360-degree overview of body composition in healthy people: Relationships among anthropometry, ultrasonography, and dual-energy x-ray absorptiometry. *Nutrition*. 2014;30(6):696-701. doi:10.1016/j.nut.2013.11.013

36. Meredith-Jones K, Haszard J, Stanger N, Taylor R. Precision of DXA-derived visceral fat measurements in a large sample of adults of varying body size. *Obesity*. 2018;26(3):505-512.

37. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of youth and adolescence*. 1988;17(2):117-133.

38. Kuczmarski RJ. *2000 CDC Growth Charts for the United States: Methods and Development*. Department of Health and Human Services, Centers for Disease Control and …; 2002.

39. Lazarus RS, Folkman S. *Stress, Appraisal, and Coping*. Springer publishing company; 1984.

40. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *Journal of health and social behavior*. Published online 1983:385-396.

41. Brantley PJ, Waggoner CD, Jones GN, Rappaport NB. A daily stress inventory: Development, reliability, and validity. *J Behav Med*. 1987;10(1):61-73. doi:10.1007/BF00845128

42. Sawyer SM, Afifi RA, Bearinger LH, et al. Adolescence: a foundation for future health. *The Lancet*. 2012;379(9826):1630-1640. doi:10.1016/S0140-6736(12)60072-5

43. WHO | The second decade: Improving adolescent health and development. WHO. Accessed April 5, 2021. https://www.who.int/maternal_child_adolescent/documents/frh_adh_98_18/en/

44. Vartanian LR. Development and validation of a brief version of the Stigmatizing Situations Inventory. *Obesity Science & Practice*. 2015;1(2):119-125. doi:https://doi.org/10.1002/osp4.11

45. Kimiecik JC, Harris AT. What is enjoyment? A conceptual/definitional analysis with implications for sport and exercise psychology. *Journal of Sport and Exercise Psychology*. 1996;18(3):247-263.

46. Motl RW, Dishman RK, Saunders R, Dowda M, Felton G, Pate RR. Measuring enjoyment of physical activity in adolescent girls. *American journal of preventive medicine*. 2001;21(2):110-117.

47. Neumark-Sztainer D. School-Based Programs for Preventing Eating Disturbances. *Journal of School Health*. 1996;66(2):64-71. doi:https://doi.org/10.1111/j.1746-1561.1996.tb07912.x

48. Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. The Eating Attitudes Test: psychometric features and clinical correlates. *Psychological Medicine*. 1982;12(4):871-878. doi:10.1017/S0033291700049163

49. Howley, ET. Type of activity: resistance, aerobic and leisure versus occupational physical activity. *Med Sci Sports Exerc*. 2001;33(6 Suppl):S364-9; discussion S419. doi:10.1097/00005768-200106001-00005

50. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of Physical Activities: classification of energy costs of human physical activities. *Medicine & Science in Sports & Exercise*. 1993;25(1):71-80.

51. Roehrig M, Duncan J, Sularz A. Caloric Intake. In: Gellman MD, Turner JR, eds. *Encyclopedia of Behavioral Medicine*. Springer; 2013:282-283. doi:10.1007/978-1-4419-1005-9_1107

52. Karlamangla AS, Friedman EM, Seeman TE, Stawksi RS, Almeida DM. Daytime trajectories of cortisol: Demographic and socioeconomic differences—Findings from the National Study of Daily Experiences. *Psychoneuroendocrinology*. 2013;38(11):2585-2597. doi:10.1016/j.psyneuen.2013.06.010

53. Ibrahim MM. Subcutaneous and visceral adipose tissue: structural and functional differences. *Obesity Reviews*. 2010;11(1):11-18. doi:https://doi.org/10.1111/j.1467-789X.2009.00623.x

54. Kahn CR. Insulin resistance, insulin insensitivity, and insulin unresponsiveness: a necessary distinction. *Metabolism*. 1978;27(12):1893-1902.

55. Reaven GM. Role of insulin resistance in human disease. *Diabetes*. 1988;37(12):1595-1607.

Chapter 2

Psychological Stress and Lowered Physical Activity Enjoyment in Adolescents with Overweight/Obesity

Preamble

The **original study aims** at the time of the dissertation proposal for study 1 was as follows: "To determine the cross-sectional associations of psychological stress on health-promoting behaviors (physical activity engagement and caloric intake), and the moderating effects of psychobehavioral constructs (physical activity enjoyment and disordered eating behavior) on these associations in adolescents age 13-19 years old with overweight and obesity." However, over the course of the past two years, these aims were refined and split into separate manuscripts focused on physical activity and eating behavior, respectively.

Psychological stress and lowered physical activity enjoyment in adolescents with overweight/obesity is the first study of the dissertation. It is a study that examined the role of psychological stress and physical activity enjoyment and participation among adolescents with overweight/obesity. The following manuscript has been published in the *American Journal of Health Promotion*. The published version of the manuscript is included in the dissertation.

*Reprinted from the *American Journal of Health Promotion*: T. Ajibewa, L. Beemer, K. Sonneville, A. Miller, C. Toledo-Corral, L. Robinson, R. Hasson. Psychological stress and lowered physical activity enjoyment in adolescents with overweight/obesity (2021). Final

publication is available from Sage Publishing: https://doi.org/10.1177/0890117121997042.

Abstract

Purpose: The purpose of this study was 2-fold: 1) to determine the cross-sectional associations between psychological stress, physical activity enjoyment, and physical activity participation [moderate-to-vigorous physical activity (MVPA), total physical activity (TPA)]; and 2) to determine the moderating effect of physical activity enjoyment on the associations between stress, MVPA, and TPA in adolescents with overweight/obesity.

Design: Cross-sectional, secondary data analysis of the Health and Culture Project and the Stress, Obesity, and Diabetes in Adolescents study.

Sample: One hundred and ten adolescents (73% female; 65.4% non-white; age 15.8 + 1.9 years) with overweight/obesity (BMI percentile ≥ 85th percentile) were included in this analysis.

Measures: Psychological stress was assessed using the Perceived Stress Scale (PSS-14); enjoyment was measured via the Physical Activity Enjoyment Scale; and MVPA and TPA were objectively measured using accelerometry over a minimum of 4 days.

Results: Higher perceived stress was associated with lower physical activity enjoyment (β= -0.41±0.15; p=0.008). Stress was not associated with MVPA or TPA (ps > 0.05), nor was enjoyment a significant moderator in the associations between stress and MVPA or stress and TPA ($p_{interaction} > 0.05$).

Conclusions: These findings suggest that psychological stress is associated with lower physical activity enjoyment among adolescents with overweight/obesity. Longitudinal studies are needed to understand the long-term effects of stress on psychological factors that may serve as antecedents to physical activity participation among adolescents with overweight/obesity.

Purpose

The transition from childhood to adolescence often comes with increased social-environmental stressors including increased academic demands (e.g., standardized tests), extra-curricular obligations and expectations (e.g., sports and after-school clubs), as well as tension from conflicts with peers, friends, and romantic relationships.[1,2] Adolescence is a particularly difficult time for youth with overweight/obesity as they are often exposed to higher levels of stress related to weight teasing, stigmatization, and criticism from peers and family members as a function of their weight status.[3,4] Adolescents with overweight/obesity are twice as likely to be bullied compared with their lower weight peers and are more likely to avoid participation in physical activity and physical education due to psychological stress experienced as a result of their weight.[4-8] Hence, the daily challenges of navigating adolescence combined with the unique stressors associated with obesity may further intensify psychological stress and negatively influence health-related behaviors including physical activity participation in this at-risk weight group.

Previously, researchers have observed a link between psychological stress and health-enhancing physical activity participation (i.e. moderate or vigorous intensity activity) in children and adolescents, but the findings have been mixed. In a five-year longitudinal study, Abi Nader and colleagues demonstrated that increased life stressors such as personal circumstances (e.g., academic challenges, rejection from a sports team) were associated with decreased moderate-to-vigorous physical activity (MVPA) across adolescent development.[9] McGlumphy and colleagues observed that increased psychological stress was a significant predictor of MVPA, with greater psychological stress associated with lower MVPA among African American male adolescents.[10] In a sample of high school adolescents in 10-12th grade, Li and colleagues observed that academically stressed Chinese adolescents reported lower moderate and vigorous physical activity participation, respectively.[11] Others, however, have found that psychological stress is not associated with MVPA in adolescents. For example, Gerber and Pühse in their study examining the impact of psychological stress, physical activity, and self-esteem in adolescents, observed that perceived school-based stress was unrelated to leisure time MVPA.[12] Similarly, null findings have been observed more recently in a study of academic stress and MVPA participation during recess and after school in adolescents.[13] Given that adolescence is a transitional developmental period with different social, environmental, and biological changes, it is critical to understand how psychological stress may influence physical activity in this age group, particularly among adolescents with overweight/obesity, in order to identify factors that hinder, alter, or promote physical activity participation within this at-risk group.

Despite the current recommended guidelines of 60 minutes of MVPA per day in addition to the numerous physical health benefits (e.g. increased cardiorespiratory fitness, bone strengthening), and mental health benefits (e.g. reduced depression, anxiety, and improved self-esteem) of physical activity, few adolescents with overweight/obesity engage in MVPA.[13-16] Considering the aforementioned link between psychological stress and low physical activity participation, one plausible explanation for this relationship is physical activity enjoyment, a factor that is commonly linked with sustained physical activity participation in youth.[17,18] Investigators examining stress related to weight stigma found that experiencing increased stigma was associated with lowered enjoyment of physical activity in children and adolescents.[19] Similar findings have been observed within the adult literature.[20,21] In their seminal article examining the effects of psychological stress on exercise adherence in adult women, Stetson and colleagues noticed that during weeks of high stress (weeks with high frequency of stressful events), participants not only reported reduced time spent exercising, but also decreased enjoyment of physical activity during times when they were active compared to times when they were less stressed.[20] Stults-Kolehmainen and colleagues examined the association between stress and affective responses to exercise in a sample of undergraduate college students and observed that increased perceived stress was associated with decreased pleasure during strenuous exercise. Together, these studies point to the important influence of psychological stress in decreasing physical activity enjoyment among youth and adults. However, it remains unclear whether psychological stress decreases physical activity enjoyment in adolescents with overweight/obesity, or if it plays an essential role in altering the association between stress and physical activity participation in this group. Given the evidence from prior investigators indicating lower levels of physical activity enjoyment among adolescents with overweight/obesity,[22] stress may further decrease physical activity enjoyment, in turn lowering participation in health-enhancing physical activity. If enjoyment is indeed associated with psychological stress and does moderate the relationship between psychological stress and physical activity participation, then, finding ways to increase physical activity enjoyment may not only increase physical activity participation, but enable youth to better utilize physical activity as a means to cope with their stress.

The purpose of this present study was 2-fold: (1) to determine the cross-sectional to associations of psychological stress on physical activity enjoyment and physical activity participation [MVPA, and total physical activity (TPA)] in adolescents with overweight/obesity; and (2) to examine the potential moderating effect of physical activity enjoyment on the association between stress and physical activity participation. It was hypothesized that increased psychological stress would be associated with decreased physical activity enjoyment and physical activity participation. Secondly, it was hypothesized that the associations between psychological stress, MVPA, and TPA participation would be moderated by enjoyment of physical activity, where greater participation in physical activity (MVPA and TPA) would be seen among high stress youth with higher enjoyment, compared to those with lower enjoyment.

Methods

Study Design and Study Sample

Data from 2 observational studies conducted at the University of Michigan Childhood Disparities Research Laboratory (CDRL) were included in the analysis: The Health and Culture Project (HCP), and the Stress, Obesity, and Diabetes in Adolescents (SODA) study. The primary aim of HCP was to examine the social, cultural, and psychological predictors of obesity in children and adolescents. The primary aim of the SODA study was to examine cross-sectional associations between different indices of stress (exposure, perception, physiological response) on type 2 diabetes risk in Hispanic, and non-Hispanic Black and White adolescents with overweight/ obesity. Data collection procedures for both studies occurred at the CDRL or off-site at local schools, after-school programs, community centers, or at the University of Michigan Clinical Research Unit (MCRU). A detailed description of the protocol and study sample for both studies have been previously published.[23,24] Participants were included in the present analysis if: i) they were between the ages of 13-19 years old; ii) they were individuals with overweight/obesity (BMI percentile ≥ 85th percentile); iii) they had 4 days of valid accelerometer data; iv) they had completed psychological stress and physical activity enjoyment questionnaires. In total, 110 adolescents (ages 13-19) with overweight/obesity were included in this analysis. Among participants that were included compared with those excluded from the final analysis, there were significant differences (mean ± SE) in age (included: 15.8 ± 1.9 years vs excluded: 16.6 ± 1.7 years; $p < 0.01$), MVPA (included: 12.3 ± 12.9 min/day vs excluded: 4.5 ± 13.7 min/day; $p < 0.01$), and TPA (included: 104.9 ± 40.5 minutes/day vs excluded: 33.1 ± 57.3 minutes/day; $p < 0.01$). There were, however, no significant differences in psychological stress (via PSS-14), pubertal development category scores, BMI, BMI percentile, gender and racial composition, or physical activity enjoyment (via the physical activity enjoyment scale) between those included and those excluded from the present analysis (ps > 0.05). Before the initiation of both studies (HCP and SODA studies), all experimental procedures were approved by the University of Michigan Institutional Review Board (HUM00083264 and HUM00080820). Before the commencement of all data collection procedures, written informed assent was obtained from all participants under the age of 18 years, and written informed consent obtained from each of their caregivers. For participants over the age of 18 years, only written informed consent was obtained.

Measures

MVPA and TPA participation was measured using the GT3X ActiLife accelerometers (Actigraph LLC., Pensacola, FL). All participants were asked to wear the 3 axial accelerometers for 7 days (including at least 1 weekend day) snugly on their right hip using an adjustable belt. At home, participants were also asked to complete a non-wear/sleep diary in tandem with the accelerometer. Participants were advised to remove the accelerometers during bathing, water-based activities, contact sports, and while they slept. All accelerometers were initialized to collect raw data at a frequency of 30 Hz and adjusted to include each participant’s measured body weight and height, self-reported age, gender, and race. Raw accelerometer data were downloaded and integrated into 60s epochs via ActiLife software version 6.11.8 using Puyau cut points.[25] The Puyau cut-points were used to derive moderate-to-vigorous activity.[25] A wear time of ≥ 600 minutes/day was used as the criteria for a valid day, and ≥4 valid wear-days was used valid wear-days was used as the criteria for an accurate baseline measure, in line with established accelerometry recommendations.[26] Sleep time was manually entered, and marked as non-wear time. Consecutive periods of zero counts for ≥ 60 minutes were also defined as non-wear time.[27] Non-wear times were excluded from the analyses with only valid wear-time data used to determine intensities of daily activity intensities in minutes.

Physical activity enjoyment was measured using the revised physical activity enjoyment scale (PACES). PACES was originally developed to assess physical activity enjoyment in college-aged students and consisted of 18 bipolar statements on a 7-point range.[28] A modified and validated version of the PACES, consisting of 16 different statements regarding feelings when engaging in physical activity on a 5-point Likert-type scale anchored by "disagree a lot" (1) to "agree a lot" (5) was used for the present study.[29] For each individual question, participants were asked to respond honestly, indicating how they feel when they are active. An example item from the PACES questionnaire was: "*When I am physically active...It gives me a strong feeling of success*." Physical activity enjoyment scores were calculated by adding together the responses for all of the questionnaire items to obtain a sum score, with higher scores indicating greater enjoyment. The PACES questionnaire has been previously validated and used in adolescent samples.[29,30] The PACES questionnaire had excellent reliability within our sample (Cronbach α = 0.93).

Psychological stress was measured using the perceived stress scale (PSS-14),[31] a global measure of stress designed to capture how unpredictable, uncontrollable, and overloaded an individual may appraise their life circumstances over the past month. An example question from the PSS-14 was: "*In the last month, how often have you dealt successfully with day-to-day* *problems and annoyances?*" Answers for all questions on the PSS-14 ranged from 0 (never) to 4 (very often). Answers to positively worded questions were reverse scored (e.g. 0=4, 1=3, 2=2, 3=1, 4=0). All 14 items were added together to obtain the total perceived stress score, with higher scores indicating higher perceived stress. The perceived stress scale is a validated and reliable instrument31 that has been previously used in an adolescent sample.[32] The PSS-14 demonstrated good reliability within our sample (Cronbach $\alpha = 0.80$).

Covariates such as anthropometric measures of height and weight were measured in the CDRL by trained study staff members. Height (cm) was measured to the nearest 0.1 cm using ShorrBoard ® (Weigh and Measure, LLC. Olney, MD) with standardized procedures.[33] Body weight (kg) was measured to the nearest 0.1 kg, using an electronic scale (Doran Scales, Inc, Batavia, IL). BMI percentiles for age and gender were calculated using the Centers for Disease Control and Prevention guidelines (http://www.cdc.gov/growthcharts). All participants were asked to self-report whether their development was earlier or later than their peers, using the pubertal development scale (PDS).[34] Based on the self-reported completion of developmental stages on the PDS, pubertal category scores were added together and were classified into the following categories: pre-pubertal, early pubertal, mid-pubertal, late pubertal, or post-pubertal. Race and gender were self-reported. Given the possible confounding influence of gender, race, pubertal development, and BMI percentile on the association between psychological stress and physical activity, these variables were added as covariates and controlled for in all of the statistical models.[17,35-37]

Table 2-1. Descriptive Statistics for Psychological Stress and Physical Activity Analysis.

Variables	Mean (SD) or Frequency (%)
Psychological stress (PSS-14)	24.4 (7.7)
PA enjoyment (PACES)	63.5 (12.1)
MVPA (min per/day)	12.3 (12.9)
TPA (min per/day)	104.9 (40.5)
Age (years)	15.8 (1.9)
Gender (% female)	73%
Ethnicity	--
Non-Hispanic White (n=38)	34.6%
Non-Hispanic Black or African-American (n=68)	61.8%
Hispanic (n=4)	3.6%
BMI percentile	93.8 (7.7)
BMI (kg/m^2)	31.5 (8.2)
Pubertal Category Score	9.7 (2.0)

PA: Physical Activity; MVPA: Moderate-to-vigorous Physical Activity; TPA: Total Physical Activity; BMI: Body Mass Index.

Analysis

All data analyses were conducted using Stata 16.0 (StataCorp LP, College Station, TX). Prior to all statistical analyses, mean MVPA and TPA participation (minutes/day) was evaluated for normality, and was natural-log transformed to help normalize its distribution. Descriptive analysis for the dependent variables, independent variable, and all covariates were also conducted. For the main effect models, multivariable linear regression was used to examine the association between the independent variable (psychological stress) and the dependent variables (physical activity enjoyment, MVPA, TPA participation), adjusted for all covariates (race, gender, BMI percentile, and pubertal development) in each model. In additional models, the moderating effects of physical activity enjoyment on the relationship between psychological stress and MVPA, and psychological stress and TPA were tested by adding an interaction term (e.g. psychological stress*PACES). The continuous variable of physical activity enjoyment was used as opposed to a dichotomized variable in order to avoid common issues with such groupings (i.e. increased probability of false positives, arbitrariness of dichotomizing at the median).[38,39]

Results

Participant Characteristics

Participant characteristics are described in Table 1. In total, study participants were: 73% female (27% male), 65.5% adolescents of color. In the adolescents of color group, 68 participants self-identified as non-Hispanic Black or African-American, whereas 4 participants self-identified as Hispanic. Overall, 34.5% of the sample, self-identified as non-Hispanic White. Mean age of study participants was 15.8 ± 1.9 years, with a mean BMI percentile of 93.8 ± 7.7 percentile (BMI: 31.5 ± 8.2 kg/m2). Mean score for physical activity enjoyment was 63.5, similar to scores previously seen among an adolescent sample using PACES,[40] and indicative of a generally high rating of physical activity enjoyment among the sample participants. An average score of 24.4 was observed for psychological stress, suggesting that stress levels were moderate across the sample (based on a tertile classification of low, moderate, and high stress from possible scores of 0-56). Stress scores were similar to that of another sample of adolescents using the PSS-14.[10] On average, participants engaged in 12.3 minutes of MVPA per day, which is slightly less than the national median of 15 minutes per/day that has been previously reported.[41] The range for MVPA was 0 to 58 minutes of MVPA per day, with a standard deviation of 12.9 min/day. Mean TPA per day was 104.9 minutes, with a range of 25.7 to 233.2 minutes of activity per day, and a standard deviation of 40.5 minutes.

Psychological Stress, Physical Activity Enjoyment, and MVPA Participation

Table 2 displays the linear regression models used to examine the associations between psychological stress, physical activity enjoyment, and MVPA participation. In model 1, it was observed that psychological stress was associated with physical activity enjoyment (β= -0.41 ± 0.15; p=0.008), with a one unit increase in psychological stress associated with a 0.41 decrease in physical activity enjoyment score in adolescents with overweight/obesity. In models 2 (examining the association between psychological stress and MVPA) and 3 (examining the association between enjoyment and MVPA), it was observed that psychological stress was not associated with MVPA participation, nor did physical activity enjoyment predict MVPA (*p*s > 0.05). Lastly, in model 4, examining the moderating effect of physical activity enjoyment in the association between psychological stress and MVPA participation, it was observed that enjoyment of physical activity did not significantly moderate the association between stress and MVPA ($p_{\text{interaction}} > 0.05$).

Table 2-2. Linear Regression Analysis for Psychological Stress, Physical Activity Enjoyment, and Log-Based MVPA Engagement.

	β	SE	p-value
Model 1: Physical activity enjoyment			
Psychological stress	-0.41	0.15	**0.008***
BMI percentile	-0.06	0.15	0.71
Gender	1.78	2.81	0.53
Race	2.10	2.23	0.35
Pubertal category score	-1.12	0.62	0.07
Model 2: MVPA participation			
Psychological stress	0.01	0.02	0.41
BMI percentile	-0.005	0.02	0.76
Gender	-1.05	0.29	**<0.001***
Race	-0.14	0.23	0.54
Pubertal category score	0.005	0.06	0.94
Model 3: MVPA participation			
PA enjoyment	0.02	0.01	0.11
BMI percentile	-0.003	0.02	0.86
Gender	-1.03	0.28	**<0.001***
Race	-0.13	0.22	0.57
Pubertal category score	0.02	0.06	0.80
Model 4: MVPA participation			
Psychological stress*PA enjoyment	<0.001	0.001	0.77
BMI percentile	-0.004	0.02	0.80
Gender	-1.08	0.29	**<0.001***
Race	-0.18	0.23	0.43
Pubertal category score	0.02	0.07	0.73

PA: physical activity; MVPA: Moderate-to-vigorous Physical Activity; * Denotes $p < 0.05$. β: Unstandardized Regression Coefficient.

Psychological Stress, Physical Activity Enjoyment, TPA Participation

Table 3 displays the linear regression models used to examine the association between psychological stress, physical activity enjoyment, and TPA participation. In model 1 examining the association between psychological stress and TPA it was observed that psychological stress was not associated with TPA ($p > 0.05$). Similarly, physical activity enjoyment was not associated with TPA, nor did physical activity enjoyment moderate the association between psychological stress and TPA (*p*s > 0.05) (see models 2 and 3).

Table 2-3. Linear Regression Analysis for Psychological Stress, Physical Activity Enjoyment, and Log-Based TPA Engagement

	β	SE	p-value
Model 1: TPA participation			
Psychological stress	0.005	0.005	0.28
BMI percentile	0.01	0.005	**0.04***
Gender	-0.33	0.09	**<0.001***
Race	0.13	0.07	0.08
Pubertal category score	-0.01	0.02	0.58
Model 2: TPA participation			
PA enjoyment	0.004	0.003	0.25
BMI percentile	0.01	0.005	**0.03***
Gender	-0.33	0.09	**<0.001***
Race	0.14	0.07	0.056
Pubertal category score	-0.01	0.02	0.64
Model 3: TPA participation			
Psychological stress*PA enjoyment	<0.001	<0.001	0.95
BMI percentile	0.01	0.005	**0.04***
Gender	-0.34	0.09	**<0.001***
Race	0.12	0.07	0.10
Pubertal category score	-0.006	0.02	0.77

PA: Physical activity; TPA: Total physical activity; * Denotes $p < 0.05$. β: Unstandardized Regression Coefficient.

Discussion

Consistent with our hypothesis, greater psychological stress was associated with lower enjoyment of physical activity among adolescents with overweight/obesity. In contrast to our hypothesis, psychological stress was not associated with MVPA, TPA, nor was physical activity enjoyment a significant moderator in the models examining psychological stress and MVPA, as well as psychological stress and TPA. Taken together, these results demonstrate an important association between psychological stress and decreased enjoyment of physical activity in adolescents with overweight/obesity. Physical activity enjoyment serves as an antecedent to physical activity participation, thus longitudinal studies are needed to understand the long-term effects of stress on physical activity enjoyment and other psychological factors related to physical activity in adolescents with overweight/obesity.

Physical activity enjoyment is a factor commonly linked with sustained physical activity participation in youth and is a reflection of one's intrinsic motivation toward physical activity[17,18]. Within the context of physical education, studies have also found that increased enjoyment of physical activity is associated with increased physical activity participation (e.g.[40,42,43]). Moreover, prior research supports the present findings that greater psychological stress is associated with decreased enjoyment of physical activity.[19-21] Faith et al. found that increased weight criticism was associated with reductions in sports enjoyment in fifth- through eighth-grade children and adolescents.[19] Unlike the present study however, the children and adolescents in the study by Faith and colleagues were of lower weight-status, pointing to the pervasiveness of stress and weight-related stressors during youth and the value of examining this association in adolescents with overweight/obesity. In another study composed of undergraduate college students (mean age of 20 years) Stults-Kolehmainen and colleagues observed that increased perceived stress was associated with decreased enjoyment during strenuous exercise,[21] indicating how stress may decrease enjoyment of health-enhancing higher intensity activity. Lastly, in a group of adult women, Stetson and colleagues noted that participants reported significantly less enjoyment of exercise during weeks of high stress in comparison to weeks of lower perceived stress.[20] Although, this present study was done in a group of adolescents, the findings are consistent with that of Stetson and colleagues, and reveal the notable effect of stress in decreasing enjoyment of physical activity in an earlier period of life among those with overweight/obesity. It is plausible that increased stress may further worsen self-perceptions about physical activity and actual participation in physical activity among youth with overweight/obesity in the long-term. In agreement with this hypothesis, other investigators have pointed to negative perceptions of physical activity among adolescents with overweight/ obesity.[44,45] These negative social experiences (e.g. weight-based bullying, stigmatization) and perceptions (e.g. insecurity about their appearance) during activity may work to reinforce negative stereotypes and self-perceptions during a crucial time of psychosocial development, resulting in decreases in enjoyment of activity. Additional research is needed to better understand the deleterious effects of psychological stress on physical activity enjoyment.

Surprisingly, psychological stress was not associated with MVPA or TPA participation in the present analysis. This finding was counter to our hypothesis as prior studies have previously found a significant association between stress and physical activity participation among adolescents (e.g.[9-11]). Specifically, in a longitudinal study conducted by Abi Nader and colleagues, they found that increased life stressors were associated with decreased MVPA participation in late childhood and early adolescence. Similarly, McGlumphy et al. as well as Li and colleagues in their studies examining the relationship between stress and physical activity, also found an association between higher levels of stress and lowered MVPA in youth.[10,11] Yet, other investigators have found that stress was not associated with physical activity. Gerber and Pühse observed that perceived school-based stress was not related to moderate-to-vigorous leisure time physical activity[12] and Frömel and colleagues observed no differences in MVPA participation in adolescents experiencing and not experiencing academic stress.[46] We extend these previous findings by explicitly examining the association between psychological stress and both MVPA and TPA in adolescents with overweight/obesity, observing a null result in the relationship between stress and physical activity in adolescents with higher weight status. With few studies using device-based objective measures of physical activity in adolescents with overweight/obesity,[47] longitudinal studies incorporating device-based measures are needed to understand the long-term influence of psychological stress on objectively-measured physical activity behavior in adolescents with overweight/obesity.

Another surprising finding in the present study was that physical activity enjoyment did not moderate the association between psychological stress and physical activity participation, nor did enjoyment predict MVPA or TPA. These findings were unexpected, given that physical activity enjoyment is a known construct associated with physical activity participation.[17,18] Nevertheless, similar findings have been previously observed in an early adolescent sample of 12-year-old girls. In their study examining the role of physical activity enjoyment in the relationship between social and physical environmental pathways and MVPA, Budd and colleagues found that enjoyment of physical activity did not mediate the relationship between participants' social environment (measured as support from friends and family in engaging in physical activity) and physical environment (neighborhood safety and aesthetics) with their MVPA participation, nor was enjoyment associated with MVPA participation.[48] Similar to our present study, the authors noticed low levels of physical activity (i.e. MVPA) in their sample, and pointed to these low levels as a possible reason for the lack of an association between enjoyment and MVPA participation. Even so, it must be noted that participants in the present study generally had a high enjoyment of physical activity (average ratings of 63.7 on a scale from 16-80 for physical activity enjoyment). Hence, there appeared to have been a disconnect between the enjoyment of physical activity and actual participation in physical activity. Given the limited research on the link between physical activity enjoyment and physical activity participation in adolescents with overweight/ obesity, it is also possible the lack of an association between physical activity enjoyment and physical activity participation may be due to our sample characteristics (i.e. adolescents with higher weight status), and the generally low levels of physical activity often seen in this population.

It is plausible that the desire to be physically active among adolescents with overweight/obesity was hindered by physical, and social-environmental barriers. Barriers such as bodily complaints (e.g., difficulty breathing, muscle pain, fatigue), perceptions about not being good at physical activity or sports, lack of social support from friends and family, as well as body image concerns or insecurities, may have hindered actual participation in physical activity. These barriers are particularly relevant among adolescents with overweight/obesity, and have been noted as barriers for engaging in physical activity in this group.[45,49] These concerns are also more visible during this developmental period in general, where concerns about body image and body dissatisfaction are heightened[50,51] and peer acceptance is paramount.[52] When the threat of peer bullying is high due to one's weight and is coupled with known barriers to engaging in physical activity, it is unlikely that an adolescent would be inclined to exercise even if they enjoy participating in exercise. It is also possible that the low level of physical activity participation may be an indirect consequence of adolescents with overweight/obesity avoiding stigmatizing domains.[50] Thus, although the construct of physical activity enjoyment is important for sustained physical activity, other environmental factors may play a larger role in the case of adolescents with overweight/obesity. Lastly, the choice of being physically active is also influenced by access to active or sedentary alternatives.[53] For a majority of people the decision to engage in sedentary behavior is driven in part by the increased accessibility of such behaviors,[53] particularly as the modern environments favor a more sedentary lifestyle.[54] Moreover, it requires more work to access and engage in physical activity, such as changing clothes, showering post exercise, and modest discomfort during exercise.[53] Although sedentary behavior was not examined in this study, it is likely that the increased accessibility of sedentary activities such television viewing and video game playing,[54] as well as the higher reported barriers by adolescents with overweight/obesity may hinder participation in physical activity. Additional research is warranted to further comprehend the interrelationships between psychological stress, internalized stigma, physical activity enjoyment, and physical activity participation in this population of adolescents.

Several strengths and limitations of this study should be noted. Strengths of the study include the use of a validated, objective measurement of physical activity (accelerometry), in a naturalistic setting (outside of the laboratory). Additionally, the assessment of relevant dependent variables in an at-risk target group are other notable strengths of this study. No other studies to our knowledge have examined the association of stress, enjoyment, and objectively measured physical activity (MVPA, TPA) in a racially diverse sample of adolescents with high obesity rates. Nevertheless, the cross-sectional study design limits inferences on the directionality of the associations between psychological stress, physical activity enjoyment, and physical activity participation. Second, the use of self-reported psychological stress and the recruitment of a convenience sample of adolescents with overweight/obesity limits generalizability to adolescents of lowered weight status. Furthermore, lack of information regarding the type of physical activity adolescents engaged in (e.g. structured sports programs) beyond intensity of activities is an additional limitation. It is plausible that adolescents involved in structured activities may have opportunities to engage in physical activity regardless of their enjoyment, thus more research is needed in this area. Lastly, measures of physical and social-environmental barriers that may have impeded physical activity engagement (e.g. neighborhood safety) or body image concerns were not taken. Despite these limitations, we were able to observe a significant association between psychological stress and physical activity enjoyment in adolescents with overweight/obesity.

In conclusion, the present study sheds light on the implications of stress on a vital intrinsic physical activity antecedent—enjoyment, among adolescents with overweight/obesity. Given that physical activity enjoyment is a key factor in sustained physical activity participation, this finding is noteworthy. With prior evidence indicating lowered enjoyment of physical activity among adolescents with overweight/obesity, it is possible that increased stress exposure experienced by adolescents with overweight/obesity may further worsen or exert more immediate effects on psychological factors related to or preceding physical activity. Ultimately, decreases in such psychological factors over longer periods may lead to lowered participation in health-enhancing physical activity due in part to a lack of intrinsic enjoyment of physical activity. Nevertheless, longitudinal studies are warranted to confirm the long-term effects of stress on physical activity enjoyment and other related psychological factors associated with physical activity during this critical period of growth and development. By understanding the long-term effects of stress on determinants of physical activity, researchers and interventionists may be able to find more appropriate ways for adolescents to cope with psychological stress thereby maintaining higher levels of physical activity enjoyment and participation in health enhancing physical activity.

Acknowledgments

We would like to thank the Health and Culture Study team (HCP), the Stress, Obesity, and Diabetes in Adolescents (SODA) study team, Michigan Clinical Research Unit and

Michigan Consulting for Statistics, Computing and Analytics Research. We are also grateful for our study participants and their families for their involvement. The results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the American Diabetes Association (Grant # 7-13JF-24), the University of Michigan School of Public Health Momentum Center, and the University of Michigan Office of Research. The Health and Culture Project is registered at www.clinicaltrials.gov (No. NCT02938663).

References

1. Rith-Najarian LR, McLaughlin KA, Sheridan MA, Nock MK. The biopsychosocial model of stress in adolescence: self awareness of performance versus stress reactivity. Stress. 2014; 17(2):193-203. doi:10.3109/10253890.2014.891102

2. Steinberg L, Morris AS. Adolescent development. Ann Rev Psychol. 2001;52(1):83-110. doi:10.1146/annurev.psych.52.1.83

3. Himmelstein M, Puhl R. Weight-based victimization from friends and family: implications for how adolescents cope with weight stigma. Pediatric Obes. 2019;14(1):e12453.

4. Tomiyama AJ.Weight stigma is stressful. A review of evidence for the cyclic obesity/weight-based stigma model. Appetite. 2014; 82:8-15. doi:10.1016/j.appet.2014.06.108

5. Hayden-Wade HA, Stein RI, Ghaderi A, Saelens BE, Zabinski MF, Wilfley DE. Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. Obes Res. 2005;13(8):1381-1392.

6. Losekam S, Goetzky B, Kraeling S, Rief W, Hilbert A. Physical activity in normal-weight and overweight youth: associations with weight teasing and self-efficacy. Obes Facts. 2010;3(4): 239-244. doi:10.1159/000319433

7. Puhl RM, Luedicke J. Weight-based victimization among adolescents in the school setting: emotional reactions and coping behaviors. J Youth Adolesc. 2012;41(1):27-40. doi:10.1007/ s10964-011-9713-z

8. Slater A, Tiggemann M. Gender differences in adolescent sport participation, teasing, self-objectification and body image concerns. J Adolesc. 2011;34(3):455-463.

9. Abi Nader P, Ward S, Eltonsy S, B´elanger M. The impact of life stresses on physical activity participation during adolescence: a 5year longitudinal study. Prev Med. 2018;116:6-12. doi:10.1016/j. ypmed.2018.08.030

10. McGlumphy KC, Shaver ER, Ajibewa TA, Hasson RE. Perceived stress predicts lower physical activity in African-American boys, but not girls. Am J Health Behav. 2018;42(2):93-105.

11. Li C, Yuan Z, Clements-Noelle K, Fu Y, Deadmond M, Yang W. Physical activity and overweight/obesity among academic stressed adolescents. Biostatistics Epidemiol Int J. 2018;1(2): 40-46. doi:10.30881/beij.00011

12. Gerber M, Pühse U. “Don’t crack under pressure!”—Do leisure time physical activity and self-esteem moderate the relationship between school-based stress and psychosomatic complaints? J Psychosomat Res. 2008;65(4):363-369.

13. Elmesmari R, Martin A, Reilly JJ, Paton JY. Comparison of accelerometer measured levels of physical activity and sedentary time between obese and non-obese children and adolescents: a systematic review. BMC Pediatrics. 2018;18(1):106.

14. Piercy KL, Troiano RP, Ballard RM, et al. The physical activity guidelines for Americans. JAMA. 2018;320(19):2020-2028. doi: 10.1001/jama.2018.14854

15. Dale LP, Vanderloo L, Moore S, Faulkner G. Physical activity and depression, anxiety, and self-esteem in children and youth: an umbrella systematic review. Mental Health Phys Activity. 2019; 16:66-79. doi:10.1016/j.mhpa.2018.12.001

16. Biddle SJH, Asare M. Physical activity and mental health in children and adolescents: a review of reviews. Br J Sports Med. 2011; 45(11):886-895. doi:10.1136/bjsports-2011-090185

17. Labbrozzi D, Robazza C, Bertollo M, Bucci I, Bortoli L. Pubertal development, physical self-perception, and motivation toward physical activity in girls. J Adolesc. 2013;36(4):759-765.

18. Taylor IM, Ntoumanis N, Standage M, Spray CM. Motivational predictors of physical education students' effort, exercise intentions, and leisure-time physical activity: a multilevel linear growth analysis. J Sport Exerc Psychol. 2010;32(1):99-120.

19. Faith MS, Leone MA, Ayers TS, Heo M, Pietrobelli A. Weight criticism during physical activity, coping skills, and reported physical activity in children. Pediatrics. 2002;110(2):e23-e23.

20. Stetson BA, Rahn JM, Dubbert PM, Wilner BI, Mercury MG. Prospective evaluation of the effects of stress on exercise adherence in community-residing women. Health Psychol. 1997;16(6):515.

21. Stults-Kolehmainen MA, Lu T, Ciccolo JT, Bartholomew JB, Brotnow L, Sinha R.Higher chronic psychological stress is associated with blunted affective responses to strenuous resistance exercise: RPE, pleasure, pain. Psychol Sport Exerc. 2016;22: 27-36. doi:10.1016/j.psychsport.2015.05.004

22. Fairclough S, Stratton G. Physical activity, fitness, and affective responses of normal-weight and overweight adolescents during physical education. Pediatric Exerc Sci. 2006;18(1):53-63.

23. Ajibewa TA, Zhou M, Barry MR, et al. Adolescent stress: a predictor of dieting behaviors in youth with overweight/obesity. Appetite. 2020;147:104560.

24. Nelson DS, Gerras JM, McGlumphy KC, et al. Racial discrimination and low household education predict higher body mass index in African American youth. Child Obes. 2018;14(2):114-121.

25. Puyau MR, Adolph AL, Vohra FA, Butte NF. Validation and calibration of physical activity monitors in children. Obes Res. 2002;10(3):150-157.

26. Trost SG, Pate RR, Freedson PS, Sallis JF, Taylor WC. Using objective physical activity measures with youth: how many days of monitoring are needed? Med Sc Sports Exerc. 2000;32(2):426.

27. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. Med Sci Sports Exerc. 2008;40(1):181-188.

28. Kendzierski D, DeCarlo KJ. Physical activity enjoyment scale: two validation studies. J Sport Exerc Psychol. 1991;13(1):50-64.

29. Motl RW, Dishman RK, Saunders R, Dowda M, Felton G, Pate RR. Measuring enjoyment of physical activity in adolescent girls. Am J Prev Med. 2001;21(2):110-117.

30. Carraro A, Young MC, Robazza C. A contribution to the validation of the physical activity enjoyment scale in an Italian sample. Social Behav Person: Int J. 2008;36(7):911-918.

31. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. J Health Social Behav. Published online. 1983; 24(4):385-396.

32. Finkelstein DM, Kubzansky LD, Capitman J, Goodman E. Socioeconomic differences in adolescent stress: the role of psychological resources. J Adolesc Health. 2007;40(2):127-134. doi:10.1016/j.jadohealth.2006.10.006

33. Shorr IJ. How to Weigh and Measure Children. Hunger Watch; 1984.

34. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: reliability, validity, and initial norms. J Youth Adolesc. 1988;17(2):117-133.

35. Hasson RE. Addressing disparities in physical activity participation among African American and Latino Youth. Kinesiol Rev. 2018;7(2):163-172. doi:10.1123/kr.2018-0015

36. Hsu ARC, Nguyen-Rodriguez S, Spruijt-Metz D. Age, physical activity motivation and perceived stress in minority girls. Californian J Health Promot. 2019;17(2):1-12. doi:10.32398/cjhp. v17i2.2285

37. Laugero KD, Falcon LM, Tucker KL. Relationship between perceived stress and dietary and activity patterns in older adults participating in the Boston Puerto Rican health study. Appetite. 2011;56(1):194-204. doi:10.1016/j.appet.2010.11.001

38. Naggara O, Raymond J, Guilbert F, Roy D, Weill A, Altman DG. Analysis by categorizing or dichotomizing continuous variables is inadvisable: an example from the natural history of unruptured aneurysms. Am J Neuroradiol. 2011;32(3):437-440. doi:10.3174/ ajnr.A2425

39. Royston P, Altman DG, Sauerbrei W. Dichotomizing continuous predictors in multiple regression: a bad idea. Stat Med. 2006; 25(1):127-141. doi:10.1002/sim.2331

40. Woods CB, Tannehill D, Walsh J. An examination of the relationship between enjoyment, physical education, physical activity and health in Irish adolescents. Irish Educ Stud. 2012;31(3):263-280.

41. LeBlanc AG, Janssen I. Difference between self-reported and accelerometer measured moderate-to-vigorous physical activity in youth. Pediatric Exerc Sci. 2010;22(4):523-534.

42. Barr-Anderson DJ, Young DR, Sallis JF, et al. Structured physical activity and psychosocial correlates in middle-school girls. Prev Med. 2007;44(5):404-409.

43. Sallis JF, Prochaska JJ, Taylor WC. A review of correlates of physical activity of children and adolescents. Med Sci Sports Exerc. 2000;32(5):963-975.

44. Stankov I, Olds T, Cargo M. Overweight and obese adolescents: what turns them off physical activity? Int J Behav Nutri Phys Activity. 2012;9(1):1-15.

45. Zabinski MF, Saelens BE, Stein RI, Hayden-Wade HA, Wilfley DE. Overweight children's barriers to and support for physical activity. Obes Res. 2003;11(2):238-246.

46. Frömel, K., Šafář, M., Jakubec, L., Groffik, D., & Žatka, R. Academic stress and physical activity in adolescents. BioMed Res Int. 2019; 2020: Article ID 4696592. doi:10.1155/2020/4696592

47. Smith KE, Haedt-Matt A, Mason TB, et al. Associations between naturalistically assessed physical activity patterns, affect, and eating in youth with overweight and obesity. J Behav Med. Published online April 17, 2020. doi:10.1007/s10865-020-00152-3

48. Budd EL, McQueen A, Eyler AA, Haire-Joshu D, Auslander WF, Brownson RC. The role of physical activity enjoyment in the pathways from the social and physical environments to physical activity of early adolescent girls. Prev Med. 2018; 111:6-13.

49. Deforche BI, De Bourdeaudhuij IM, Tanghe AP. Attitude toward physical activity in normal-weight, overweight and obese adolescents. J Adolesc Health. 2006;38(5):560-568. doi:10.1016/j.jadohealth.2005.01.015

50. Goldschmidt AB, Aspen VP, Sinton MM, Tanofsky-Kraff M, Wilfley DE. Disordered eating attitudes and behaviors in overweight youth. Obesity. 2008;16(2):257-264.

51. Rohde P, Stice E, Marti CN. Development and predictive effects of eating disorder risk factors during adolescence: implications for prevention efforts. Int J Eating Disord. 2015;48(2):187-198.

52. Cillessen AH, Rose AJ. Understanding popularity in the peer system. Current Directions in Psycholog Sci. 2005;14(2): 102-105.

53. Epstein LH, Roemmich JN. Reducing sedentary behavior: role in modifying physical activity. Exerc Sport Sci Rev. 2001;29(3): 103-108.

54. Chaput J, Klingenberg L, Astrup A, Sjödin AM. Modern sedentary activities promote overconsumption of food in our current obesogenic environment. Obes Rev. 2011;12(5):e12-e20.

Chapter 3

Acute Daily Stress, Daily Food Consumption and the Moderating Effect of Disordered Eating Among Adolescents with Overweight/Obesity

Preamble

The **original study aims** at the time of the dissertation proposal for study 2 was as follows: “To determine the cross-sectional associations of psychological stress on health-promoting behaviors (physical activity engagement and caloric intake), and the moderating effects of psychobehavioral constructs (physical activity enjoyment and disordered eating behavior) on these associations in adolescents age 14-19 years old with overweight and obesity.” However, over the course of the past two years, these aims were refined and split into separate manuscripts focused on physical activity and eating behavior, respectively.

Acute daily stress, daily food consumption, and the moderating effect of disordered eating among adolescents with overweight/obesity is the second study of the dissertation. It is a study that examined the cross-sectional associations between dimensions of daily stress on daily caloric intake and the potential moderating effect of disordered eating behaviors on these associations among adolescents with overweight/obesity. The following manuscript has been accepted in the journal *Childhood Obesity*. The accepted version of the manuscript is included in the dissertation.

*Reprinted from the journal *Childhood Obesity*: T. Ajibewa, L.Robinson, C. Toledo-Corral, A. Miller, K. Sonneville, R. Hasson. Acute daily stress, daily food consumption and the moderating effect of disordered eating among adolescents with overweight/obesity (2021). Final publication will be available from Mary Ann Liebert, Inc upon publication.

Abstract

OBJECTIVE: The purpose of this study was to examine the associations between acute daily stress dimensions (frequency, sum) and food intake in adolescents with overweight/obesity, and to explore the potential moderating effect of disordered eating behaviors on these associations. **METHODS:** 182 adolescents with overweight/obesity (65% females; 68.7% non-White; 16.2±1.8 years of age) were included in this analysis. Acute daily stress was measured using the Daily Stress Inventory and daily caloric intake was measured using a food frequency questionnaire. Disordered eating behavior was assessed using the Eating Attitudes Test (EAT-26). **RESULTS:** Acute daily stress frequency (B=0.013±0.003; $p<0.001$) and acute daily stress sum (B=0.003±0.001; $p<0.001$) were associated with greater daily caloric intake. Disordered eating behavior moderated the association between acute daily stress frequency and caloric intake ($p_{interaction}=0.039$) with greater daily caloric intake among those with higher levels of disordered eating. Disordered eating behavior did not significantly moderate the association between acute daily stress sum and daily caloric intake ($p_{interaction}=0.053$). **CONCLUSIONS:** These findings suggest that greater exposure to acute daily stressors may increase daily food intake in adolescents with overweight/obesity, with greater susceptibility among those engaging in high levels of disordered eating. Longitudinal research is warranted to elucidate the long-term effect of acute daily stressors and disordered eating on food intake among adolescents with overweight/obesity.

Introduction

Acute daily stressors are brief, time-limited exposures that are common and often include events such as rushing, deadlines, and arguments[1]. Under normal circumstances, acute stress and subsequent stress responses typically do not have long-term health consequences[1]. However, if an individual has repeated exposure to an acute stressor or perceives a continual threat of a stressor reoccurring, then this acute stressor may manifest into a chronic stressor, increasing susceptibility to long-term health risk.

Adolescence is a particularly vulnerable period of heightened stress, due in part to biological changes, interpersonal conflicts with peers, increased academic load at school, and greater performance-related stressors[2–5]. Adolescents with overweight/obesity often experience additional psychosocial stressors related to their weight. Unlike their peers with lower body weights, adolescents with overweight/obesity are more likely to receive criticism from close family members because of their weight, have higher incidences of teasing and rejection, and are twice as likely to be bullied[6–9]. The heightened exposure to stress observed in this high weight group increases their susceptibility to stress eating, a maladaptive behavioral response to both acute and chronic stressors[10].

Adolescents with overweight/obesity display several characteristics that may further increase their risk of stress eating. In comparison to their lower-weight peers, adolescents with overweight/obesity have greater sensitivity and exaggerated cortisol responses to acute stress[11]. Like adults with obesity, youth with overweight/obesity have been shown to exhibit lower satiety and greater sensitivity and responsiveness to appetizing food cues (e.g. smell) compared with lower-weight youth[12,13]. Evidence also suggests that adolescents with obesity are prone to exhibiting greater dietary restraint[14,15], a characteristic associated with counter-regulatory eating when exposed to acute stressors[16,17]. Lastly, adolescents with overweight/obesity have also been shown to engage in emotional eating in response to stress[16]. In sum, these characteristics suggest that adolescents with overweight/obesity may have greater vulnerability to stress-induced eating.

While adolescents with overweight/obesity may be more susceptible to stress eating, empirical data and results supporting this association within higher weight [higher BMI (body mass index)] sample populations are mixed. In adults with overweight, Lemmens et al. observed increased caloric intake after an acute psychological stressor[18]. Herhaus and colleagues more recently observed increased food consumption among men and women with obesity following an acute stressor in a laboratory environment[19]. In contrast, Nagy and colleagues observed decreased food intake following an acute laboratory stressor in adolescents with overweight/obesity and heightened cortisol reactivity[20]. Appelhans and colleagues also noticed reduced food intake among women with obesity and high stress responses following an acute stressor[21]. Given this inconsistency in food intake among sample populations with higher weight/BMI and limited studies examining this association among higher weight/BMI adolescents, more studies are needed to identify factors that contribute or otherwise alter eating behavior during periods of heightened acute stress within this group.

One plausible link that may influence the relationship between stress and food intake is disordered eating behavior. Disordered eating behaviors are defined as problematic eating behaviors (e.g. restrictive, bingeing, self-control, and compensatory behaviors) occurring at a lower frequency or severity than clinically diagnosed eating disorders[22]. Engagement in disordered eating often stems from body dissatisfaction and a desire to control one's weight/shape. Furthermore, body dissatisfaction, shape/weight concerns, dietary restriction, and the use of extreme or otherwise unhealthy weight control behaviors have been found to be more common among adolescents with higher weight status[23]. For example, epidemiological evidence suggests that approximately 40% of adolescent girls and 20% of adolescent boys with overweight/obesity engage in disordered eating behaviors over a year[24]. Empirical evidence also suggests that disordered eating predicts further weight gain among children and adolescents[25–27]. It has also been previously demonstrated that adolescents with overweight/obesity are at greater risk of engaging in disordered eating behaviors due to increased stress exposure[28]. More recently, our group observed cross-sectional associations between increased psychological stress and greater dieting behavior in adolescents with overweight/obesity[29]. Examining the potential moderating role of disordered eating behaviors in the association between stress and eating behavior in adolescents with overweight/obesity may provide additional insight into how acute stress predicts eating behavior in this at-risk group.

The purpose of this study was to examine the associations between acute daily stress and daily caloric food intake in adolescents with overweight/obesity, and to determine the potential moderating effect of disordered eating behavior on these associations. Given the racial/ethnic diversity of the sample, an exploratory aim examining how race/ethnicity may influence acute stress and eating behaviors was also conducted. The primary hypothesis was that higher acute daily stress (as defined by frequency and sum) would be associated with higher daily caloric intake. Additionally, it was hypothesized that the associations between acute daily stress and daily caloric intake would be modified by disordered eating behavior.

Methods

Sample population and procedure

Baseline data from two studies conducted at the University of Michigan Childhood Disparities Research Laboratory (CDRL) were included in this analysis: the Health and Culture

Project (HCP) and the Stress, Obesity, and Diabetes in Adolescents (SODA) study. The primary aim of the HCP study was to examine the social, cultural, and psychological predictors of obesity in adolescents. The primary aim of the SODA study was to examine cross-sectional associations among different indices of stress (exposure, perception, biological response) on type 2 diabetes risk in Hispanic, and non-Hispanic Black and White adolescents with overweight/obesity. Descriptions of both studies have been previously published[29,30]. Data collection procedures for both studies occurred at the CDRL, off-site at local schools, after-school programs, community centers, or at the University of Michigan Clinical Research Unit (MCRU). Participants were included in the present analysis if: i) they were between the ages of 13-19 years old; ii) they were individuals with overweight/obesity (BMI percentile ≥ 85^{th} percentile); iii) they had complete dietary intake data; and iv) they had completed acute daily stress data. In total, a subset of 182 adolescents with overweight/obesity were included in this analysis. There were no significant differences in distribution of race/ethnicity, gender, mean age, BMI percentile, BMI, pubertal development score, or acute daily stress among those who were included and those excluded from the analysis (p>0.05). Before starting both studies (HCP and SODA studies), all experimental procedures were approved by the University of Michigan Institutional Review Board (HUM00083264 and HUM00080820). Informed assent was obtained from all participants under the age of 18 years, and written informed consent obtained from each of their caregivers before the start of data collection procedures. For participants 18 years of age and over, solely written informed consent was obtained.

Dependent Variable

Total daily caloric intake

Total daily caloric intake is the average amount of energy consumed via food and beverage daily, and was obtained using the 2012 version of the Youth/Adolescent Food Frequency Questionnaire (YAQ) developed by Harvard University[31,32]. The YAQ is a self-reported questionnaire that asks questions related to food and drink consumption throughout the preceding year. All completed YAQs were shipped to the T.H. Chan School of Public Health at Harvard University, where they were analyzed for mean total daily calories (kcals) along with other measures (e.g. total fat) using the Nutrition Data System for Research (NDSR) Windows-based dietary analysis program. The questionnaire is designed for adolescents, and has been previously validated and calibrated as a semi-quantitative food frequency questionnaire[32].

Independent Variable

Acute daily stress

Acute daily stress was conceptualized as a brief, short-term stressor involving a specific event[33]. Acute daily stress was assessed using the Daily Stress Inventory[34], a measure of stress designed to assess daily sources and individualized impact of stressful events. The Daily Stress Inventory is a 58-item self-reported questionnaire that allows an individual to indicate the stressful events that they may have experienced over the past day (24-hours). An example question from the Daily Stress Inventory included: "*In the past 24 hours…I was criticized or verbally attacked".* Participants were asked to respond honestly, indicating whether or not the event occurred, and if so, how much stress did it cause on a Likert-type scale anchored by (1) *"occurred but was not stressful"* to (7) *"caused me to panic"*. Two scores were derived from the Daily Stress Inventory: an acute daily stress frequency score (FREQ) and an acute daily stress sum (SUM). FREQ was calculated as the total number of stressful events that occurred in the past 24 hours with possible scores ranging from 0 to 58. SUM was calculated as the summation of the impact ratings of the endorsed events (i.e., how much stress did each event cause), with a maximum possible score of 406[34]. The Daily Stress Inventory demonstrated excellent internal consistency within the study sample for acute stress FREQ (Cronbach α=0.94) and SUM (Cronbach α=0.97). The Daily Stress Inventory has been previously used in adolescent samples[35,36].

Covariates

Given the possible confounding influence of gender, race/ethnicity, pubertal development, and BMI on the association between psychological stress and caloric intake, these variables were added as covariates and controlled for in all of the statistical models[10,37–39]. Anthropometric measures of height and weight were measured in the CDRL by trained study staff members. Height (cm) was measured to the nearest 0.1 cm using ShorrBoard ® (Weigh and Measure, LLC. Olney, MD) with standardized procedures. Body weight (kg) was measured to the nearest 0.1 kg, using an electronic scale (Doran Scales, Inc, Batavia, IL). BMI-for-age and sex percentile was calculated using the Centers for Disease Control and Prevention guidelines (http://www.cdc.gov/growthcharts). Pubertal development was measured using the Pubertal Development Scale (PDS)[40]. Based on the self-reported completion of developmental stages on the PDS, pubertal category scores were calculated and classified into the following categories: pre-pubertal, early pubertal, mid-pubertal, late pubertal, or post-pubertal. Given the hormonal changes that occur with puberty, the variable was used as a covariate rather than biological age. Race and gender were self-reported.

Moderator

Disordered eating was defined and conceptualized as problematic eating behaviors (e.g. restrictive, bingeing, compensatory behaviors) occurring at a lower frequency or severity than clinically diagnosed eating disorders[22]. Disordered eating behavior was measured using the total EAT-26 score from the 26-item Eating Attitudes Test (EAT-26)[41]. The EAT-26 questionnaire is a screening instrument used to identify participants with various levels and types of eating pathology, who may be at risk for eating disorders and may therefore, need to be referred to a qualified professional[42]. It is an abbreviated version of the EAT-40 questionnaire created by Garner and colleagues[41]. The EAT-26 contains 3 subscales; the dieting subscale, bulimia and food preoccupation subscale, and an oral control subscale altogether assessing restrictive, bingeing, self-control, and compensatory behaviors. For each question, participants were asked to indicate how often each statement applied to them on a Likert-type scale anchored by 1 (never) to 6 (always). Example items from each of the EAT-26 subscales included: "*I am occupied with a desire to be thinner*" (dieting), "*I have gone on eating binges where I feel that I may not be able to stop*" (food preoccupation), and "*I display self-control around food*" (oral control)." Total EAT-score was obtained by adding all items from the questionnaire (including a reverse scored item), to get a general score of disordered eating behavior. Higher self-reported scores on EAT-26 denoted greater concern regarding body weight, body shape, and eating[41]. The questionnaire demonstrated acceptable internal consistency for the study sample (Cronbach α= 0.79). The EAT-26 questionnaire has been previously used to assess disordered eating risk in adolescent samples and among individuals with overweight/obesity[43,44].

Statistical Analysis

All data analysis was conducted using Stata 16.0 (StataCorp LLC, College Station, TX). Prior to all analyses, variables were evaluated for normality, and total daily energy intake was natural-log transformed to help normalize its distribution. Means and frequencies were determined for age, gender, race, BMI, BMI percentile, pubertal development, FREQ, SUM, and total EAT-26 score. For main effects models, multivariable linear regression was used to examine the association between the independent acute daily stress dimensions (FREQ, SUM) and the dependent variable (total daily caloric intake), adjusted for all *a priori* covariates (race, gender, BMI, and pubertal development) in each model. In the interaction models exploring the moderating effects of disordered eating behavior on the relationship between the acute daily stress dimensions and daily caloric intake, an interaction term was added to each of acute daily stress dimension (e.g. acute daily stress FREQ*EAT-score) in the regression models to test moderation. Similarly, for the exploratory aim examining how race/ethnicity may influence acute stress and caloric intake, race/ethnicity was added as an interaction term. A continuous disordered eating variable was used in place of a dichotomized or categorized variable to avoid the common issues with such variables (i.e. arbitrariness of dichotomizing at the median)[45,46]. A p-value of $p<0.05$ was set to determine significance for all models.

Results

Participant characteristics

Participant characteristics in mean and standard deviation are seen in Table 1. In total, study participants were: 65% female (35% male), 68.7% non-White. Among the non-White adolescents, 63.2% of participants self-identified as non-Hispanic Black or African-American, 4.4% as Hispanic, and 1.1% as Other (e.g. Asian, Pacific-Islander). Participants had a mean age of 16.2±1.8 years and a mean BMI percentile of 94.3±4.2% (31.1±6.1 kg/m^2). Approximately 44% (n=80) of sample participants were classified as adolescents with overweight and 56% (n=102) were classified as adolescents with obesity. On average, participants consumed 2,007 kilocalories per day, consistent with the national average of 2,100 kilocalories for boys and 1,755 kilocalories for girls[47]. There were however, 16 participants with daily caloric intake values above 3,500 kcals/day, and 4 participants with a daily caloric intake value below 500 kcals/day. FREQ score was 18.0±11.9, with participants experiencing on average 18 acute stress events over a 24-hour period. Mean SUM score of the impact of acute daily stressors was 47.3±47.7 over a 24-hour period. Both the mean FREQ and SUM scores of acute daily stressors are consistent with acute daily stress FREQ and SUM scores observed in other adolescent samples[36,48]. The average sample score for the EAT-26 was 7.8±7.1 for disordered eating behavior, a score that is also consistent with a prior study conducted in an adolescent sample [49]. Furthermore, there were 170 participants (93.4% of total sample) with EAT-26 scores under 20 (the suggested cutoff value for a recommendation to see a qualified professional for a possible eating disorder), and 12 participants (4.6% of total sample) with EAT-26 scores at or above the 20 point cutoff.

Table 3-1. Participant characteristics for the acute daily stress and daily caloric intake analysis.

Variables	**N**	**Mean (SD) or Frequency (%)**
Age (years)	182	16.2 (1.8)
Gender (% female)	182	65%
Race/ethnicity (% non-white)	182	68.7%
Non-Hispanic Black	115	63.2%
Non-Hispanic White	57	31.3%
Hispanic	8	4.4%
Other races/ethnicities	2	1.1%
BMI (kg/m^2)	182	31.1 (6.1)
BMI percentile	182	94.3 (4.3)
Pubertal category score	182	9.8 (2.0)
Acute daily stress frequency (FREQ)	182	18.0 (11.9)
Total acute daily stress (SUM)	182	47.3 (47.7)
EAT-26 score (disordered eating)	182	7.8 (7.1)
Total daily caloric intake (kcals)	182	2007.1 (1187.5)

Statistics are reported in either mean and standard deviation (SD) or frequency (%). BMI; body mass index.

Associations between acute daily stress FREQ, SUM, and total daily caloric intake

The associations between acute daily stress FREQ and SUM, with that of total daily caloric intake can be seen in Table 2. The FREQ score of acute daily stress was positively associated with total daily caloric intake, with a higher number of acute daily stress events, associated with greater total daily caloric intake (B=0.013±0.003; $p<0.001$). The SUM score of acute daily stress was also associated with total daily caloric intake among the study sample, with higher total distress from acute stress events associated with greater total daily caloric intake (B=0.003±0.001; $p<0.001$).

Predictor	Caloric intake (B±SE)	*p-value*
Total distress (SUM)	0.003±0.001	<0.001*
BMI percentile	-0.017±0.009	0.06
Gender	-0.255±0.086	0.003*
Race	0.021±0.063	0.74
Pubertal development score	-0.006±0.021	0.78
Acute stress frequency (FREQ)	0.013±0.003	<0.001*
BMI percentile	-0.017±0.01	0.053
Gender	-0.243±0.086	0.005*
Race	0.026±0.063	0.68
Pubertal development score	-0.001±0.021	0.95

Table 3-2. Association between acute daily stressors and daily caloric intake. Natural log-transformed caloric intake values.

Acute daily stress dimensions and the moderating effects of disordered eating

Interaction models were conducted to examine the moderating effects of disordered eating behavior in the association between acute daily stress dimensions and daily caloric intake. It was observed that disordered eating behavior (EAT-26 score) modified the association between acute daily stress FREQ and daily caloric intake ($p_{\text{interaction}}$= 0.039). The model suggested that for individuals with higher EAT-26 scores, a higher number of acute daily stress events (FREQ) was associated with greater daily caloric intake compared to adolescents with lower EAT-26 scores (see Table 3). Disordered eating behavior however, did not significantly modify the association between acute daily stress SUM and daily caloric intake, but trended towards significance ($p_{\text{interaction}}$=0.053; Table not shown).

Predictor	Caloric intake (B±SE)	*p-value*
Acute daily stress FREQ* Total EAT-score	0.001±0.000	0.039*
BMI percentile	-0.017±0.01	0.055
Gender	-0.23±0.085	0.008*
Race	0.02±0.06	0.78
Pubertal development score	0.004±0.02	0.84

Table 3-3. Moderating effect of disordered eating on the association between acute daily stress FREQ and daily caloric intake. Natural log-transformed caloric intake values.

Exploratory analyses of the influence of race/ethnicity on acute stress and eating behaviors

Given the small representation of participants who were Hispanic or Other (those self-identifying with another racial/ethnic group apart from Hispanic, non-Hispanic Black, or White), no racial/ethnic comparisons were conducted including these two groups in the exploratory analyses. However, among non-Hispanic White and Black participants, there were significant racial differences between scores for acute daily stress. Higher acute daily stress SUM (non-Hispanic Black: 53.7±5.1 vs non-Hispanic White: 34.2±3.7; p=0.01) and daily stress FREQ scores (non-Hispanic Black: 19.4±1.2 vs non-Hispanic White: 15.2±1.1; p=0.03) were noted among non-Hispanic Black participants compared to their White peers. A racial difference in disordered eating was also observed, with higher disordered eating levels (EAT-26 scores) among non-Hispanic Black participants compared to their non-Hispanic White peers (non-Hispanic Black: 8.58±0.75 vs non-Hispanic White: 6.28±0.65; p=0.048). Non-Hispanic Black participants also reported greater total daily caloric intake compared to their White counterparts, but this difference was not statistically significant (non-Hispanic Whites: 1785.1±96.3 kcals/day vs non-Hispanic Blacks: 2126.7±125.7 kcals/day; p=0.075). Race/ethnicity did not modify the association between acute daily stress FREQ and total daily caloric intake, nor the association between acute daily stress SUM and total daily caloric intake (ps>0.05).

Discussion

Consistent with our primary hypothesis, significant associations were observed between acute daily psychological stress dimensions and caloric intake; where higher daily stress FREQ and SUM were associated with greater daily caloric intake within our diverse sample of adolescents with overweight/obesity. In addition, adolescents who reported engaging in more disordered eating behaviors (as indicated by higher EAT-26 scores) were at an even greater risk of engaging in stress eating than those engaging in less disordered eating behaviors. Lastly, race/ethnicity differences were observed across acute stress and disordered eating levels, however, race/ethnicity did not moderate the association between acute daily stress dimensions and daily caloric intake. Given the higher stress exposure of adolescents with overweight/obesity and increased susceptibility to stress eating, it is essential to find ways to facilitate and equip higher weight adolescents with healthy stress coping strategies.

While many researchers have focused on chronic stress[50–52], much less research has examined the effects of acute daily psychological stress on eating behavior in adolescents. One such study however, was conducted by Michaud and colleagues, who previously observed that increased acute stress stemming from an academic exam was associated with increased energy intake among adolescents[53]. Similarly, the present study extends these findings by observing increased daily caloric intake among adolescents with higher weight status in response to acute daily stress. Although acute stress responses are typically associated with suppression of appetite[54], it is plausible that under circumstances where there is high cumulative burden of acute stress (as measured here in number of events and amount of distress), that maladaptive behavioral responses may occur. Adolescents with overweight/obesity may, in turn, seek to cope with their stress via increased food intake of palatable comfort foods. Eating palatable foods triggers increased secretion of dopamine, a hormone that aids in reducing feelings of stress and lowers the HPA-axis response[55]. With evidence suggesting that adolescents with overweight/obesity display high dietary restraint[14,17]—a characteristic associated with overeating when faced with an acute stressor—it is possible that adolescents may overeat when facing a high cumulative burden of acute daily stress. In addition to a higher cumulative burden of acute daily stress, evidence from the stress literature also points to the salience of stressors such as weight stigma as a social/stereotype threat known to increase food intake among those with higher weight[8]. Considering that unhealthy behaviors persist throughout adolescence and may track into adulthood[56], it is necessary to provide social-emotional resources to help adolescents with overweight/obesity cope with the pressing acute stressors they may experience on a daily basis.

In the present study, it was also observed that adolescents engaging in disordered eating were especially vulnerable to higher food intake when encountering acute daily stress. These findings are noteworthy, given the increased prevalence and susceptibility of developing eating disorders, dysfunctional attitudes to food, and heightened body dissatisfaction among adolescents with overweight/obesity[23,24]. It is possible that participants who exhibited high disordered eating behavior may already have a pathological relationship with food and thus, when experiencing acute bouts of stress (e.g. acute daily stressors) may experience reduced self-control, resulting in increased food intake. This phenomenon has been found to be particularly noticeable among those with higher weight, where stress from weight stigma for example has been found to deplete self-regulatory resources[57]. Emerging evidence also suggests that children with overweight/obesity with coexisting disordered eating behavior (i.e., loss of control eating) may display even lower self-regulation than those with overweight/obesity without disordered eating[58]. Given the higher prevalence of disordered eating behaviors among youth with overweight/obesity, in addition to the strong link between stress and self-regulation[59,60], the combination of decreased self-regulation and increased disordered eating behavior may affect food intake in this group of adolescents when experiencing acute stress. Nevertheless, future research should explore the long-term influence of acute stress on caloric intake with specific disordered eating behaviors (e.g. binge eating) as moderators among adolescents with overweight/obesity.

Due to the racial/ethnic diversity of the study sample, exploratory analyses examining how race/ethnicity may influence stress and eating behaviors were conducted. Although, non-Hispanic Black adolescents had higher levels of acute daily stressors, in addition to higher levels of disordered eating behavior, we observed that race/ethnicity did not moderate the associations between acute daily stressors and daily caloric intake. It should be noted however that the higher levels of acute daily stress observed among non-Hispanic Black participants compared to non-Hispanic White adolescents is consistent with higher social and environmental stress exposure observed in the existing stress literature (e.g.[61]). Though race/ethnicity can be used as a proxy measure of understanding race-related stress exposure (e.g. racial discrimination), it is plausible that the lack of a significant moderating effect of race/ethnicity could be due to race/ethnicity not capturing specific race related social-environmental stressors. Nevertheless, future studies examining potential racial/ethnic differences in the association between acute daily stress and eating behaviors should use specific race related stressors, which may provide additional mechanistic insight into the pathways of stress and eating behavior across racial groups.

Important strengths and limitations of this study should be noted. Strengths of the study include the assessment of relevant dependent variables in a racially diverse sample of adolescents at a higher weight status. Additionally, the comprehensive assessment of acute daily stress (utilizing frequency and total distress) are additional strengths of this study. Despite these strengths, the present findings should be interpreted in light of a few limitations. First, the cross-sectional study design limits inferences on the directionality of the associations between the acute daily stress dimensions and caloric intake, and causality cannot be inferred. Secondly, funding limitations precluded the use of a gold-standard measurement of current energy intake (e.g. doubly labeled water or direct calorimetry), and as a result, the study utilized the commonly used self-reported food frequency questionnaire. Although the food frequency questionnaire is not a gold-standard measurement of current caloric intake, it has been found to be a valid measure of caloric intake across various adolescent samples[62,63]. Moreover, the EAT-26 is a self-reported screening measure of disordered eating behavior and did not explicitly capture specific types of disordered eating behaviors that may be common to adolescents with overweight/obesity such as binge-eating and loss of control overeating. Nonetheless, the EAT-26 is among the most widely used standardized measures of symptoms and concerns of eating pathology[42], and was used presently given the wide array of pathological eating behaviors that can be experienced by adolescents with overweight/obesity. Additional research using an interview method and the contextualization of specific types of disordered eating behavior among adolescents with higher weight is necessary to gain a deeper mechanistic understanding of the interrelationship between disordered eating, stress, and food intake in this group. Lastly, it should be noted that the use of self-reported daily stress, and the recruitment of a convenience sample of adolescents with overweight/obesity limits generalizability to other adolescents without overweight/obesity. Despite these limitations, we observed significant associations between acute daily stress and higher daily caloric intake along with moderating effects of disordered eating among adolescents with overweight/obesity.

Conclusion & implications

Overall, these findings suggest that acute daily stressors are associated with increased daily caloric intake among adolescents with overweight/obesity, and that those who engage in disordered eating behaviors may be particularly at risk of increasing food intake when experiencing acute bouts of stress. If experiencing acute stress in the long-term, this group of adolescents may further gain weight and face higher cardiometabolic disease risk. This possible long-term effect of stress on food intake has been previously demonstrated, as chronic stressors have been shown to be associated with increased food intake, particularly sweetened and highly processed foods (e.g.[64]). Given that modest changes in energy intake such as an additional 100 kcals/day can lead to continuous weight gain[65], higher consistent caloric intake over-time as a result of acute stress, may be detrimental to the long-term health and wellbeing of adolescents with overweight/obesity. While the vast majority of the sample participants had an EAT-26 score below the 20-point cut-off, engaging in higher levels of the behaviors listed in the EAT-26 questionnaire points to higher risk of eating disorders and an advanced progression along the disordered eating continuum[66]. Adolescents with overweight/obesity with higher levels of disordered eating behavior who when experiencing high daily stress, may be especially vulnerable to increased food intake. When engaging in behaviors such as restriction and binging at higher levels over-time (chronically), it is highly plausible that this may lead to increased weight gain in addition to actual manifestation of an eating disorder. While our findings may not have direct clinical or treatment significance, it is important for clinicians and researchers alike to find ways to prevent this at-risk group of adolescents from being caught in a vicious and dangerous cycle of weight gain and pathological eating behaviors.

With the increased stress and mental health challenges among youth in the twenty-first century[67] and easier access to unhealthy foods[68], the challenge of finding adaptive, multi-pronged strategies and interventions focused on promoting healthy coping in this group is of the utmost importance. Strategies such as adopting anti-bullying policies within school settings to protect students from weight-based bullying, as well as providing families with the education and resources to recognize the harmful impact of weight stigma should be prioritized[69]. Fostering social support among families and friends of those experiencing weight stigma in addition to encouraging adolescents to use and practice strategies such as positive self-talk and healthy lifestyle behaviors are additional approaches that should be considered among several others. In doing so, researchers, clinicians, and families alike, may ultimately help adolescents break the cycle of stress-related eating behaviors among those at greatest risk.

ACKNOWLEDGEMENTS

We would like to thank the Health and Culture Study team (HCP), the Stress, Obesity, and Diabetes in Adolescents (SODA) study team, Michigan Clinical Research Unit and Michigan Consulting for Statistics, Computing and Analytics Research. We are also grateful for our study participants and their families for their involvement. The results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. **Conflicts of Interest:** No competing financial interests exist.

References

1. Epel ES, Crosswell AD, Mayer SE, et al. More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology*. 2018;49:146-169. doi:10.1016/j.yfrne.2018.03.001

2. Markova S, Nikitskaya E. Coping strategies of adolescents with deviant behaviour. *International Journal of Adolescence and Youth*. 2017;22(1):36-46. doi:10.1080/02673843.2013.868363

3. Rith-Najarian LR, McLaughlin KA, Sheridan MA, Nock MK. The biopsychosocial model of stress in adolescence: self-awareness of performance versus stress reactivity. *Stress*. 2014;17(2):193-203. doi:10.3109/10253890.2014.891102

4. Roemmich JN, Lambiase MJ, Balantekin KN, Feda DM, Dorn J. Stress, Behavior, and Biology: Risk Factors for Cardiovascular Diseases in Youth. *Exerc Sport Sci Rev*. 2014;42(4):145-152. doi:10.1249/JES.0000000000000027

5. Steinberg L, Morris AS. Adolescent Development. *Annual Review of Psychology*. 2001;52(1):83-110. doi:10.1146/annurev.psych.52.1.83

6. Pont SJ, Puhl R, Cook SR, Slusser W, Obesity SO, Society TO. Stigma Experienced by Children and Adolescents With Obesity. *Pediatrics*. 2017;140(6). doi:10.1542/peds.2017-3034

7. Puhl RM, Luedicke J. Weight-Based Victimization Among Adolescents in the School Setting: Emotional Reactions and Coping Behaviors. *J Youth Adolescence*. 2012;41(1):27-40. doi:10.1007/s10964-011-9713-z

8. Tomiyama AJ. Weight stigma is stressful. A review of evidence for the Cyclic Obesity/Weight-Based Stigma model. *Appetite*. 2014;82:8-15. doi:10.1016/j.appet.2014.06.108

9. Van Den Berg P, Neumark-Sztainer D, Eisenberg ME, Haines J. Racial/ethnic differences in weight-related teasing in adolescents. *Obesity*. 2008;16(S2):S3-S10.

10. Jääskeläinen A, Nevanperä N, Remes J, Rahkonen F, Järvelin M-R, Laitinen J. Stress-related eating, obesity and associated behavioural traits in adolescents: a prospective population-based cohort study. *BMC Public Health*. 2014;14(1):321.

11. Verdejo-Garcia A, Moreno-Padilla M, Garcia-Rios MC, et al. Social Stress Increases Cortisol and Hampers Attention in Adolescents with Excess Weight. *PLoS One*. 2015;10(4). doi:10.1371/journal.pone.0123565

12. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. *The American journal of clinical nutrition*. 2008;88(1):22-29.

13. Jansen A, Theunissen N, Slechten K, et al. Overweight children overeat after exposure to food cues. *Eating behaviors*. 2003;4(2):197-209.

14. Hofmann J, Ardelt-Gattinger E, Paulmichl K, Weghuber D, Blechert J. Dietary restraint and impulsivity modulate neural responses to food in adolescents with obesity and healthy adolescents. *Obesity*. 2015;23(11):2183-2189.

15. Snoek HM, van Strien T, Janssens JM, Engels RC. Restrained eating and BMI: A longitudinal study among adolescents. *Health Psychology*. 2008;27(6):753.

16. Kubiak T, Vögele C, Siering M, Schiel R, Weber H. Daily hassles and emotional eating in obese adolescents under restricted dietary conditions—The role of ruminative thinking. *Appetite*. 2008;51(1):206-209.

17. Roemmich JN, Wright SM, Epstein LH. Dietary Restraint and Stress-Induced Snacking in Youth. *Obesity Research*. 2002;10(11):1120-1126. doi:10.1038/oby.2002.152

18. Lemmens SG, Rutters F, Born JM, Westerterp-Plantenga MS. Stress augments food 'wanting'and energy intake in visceral overweight subjects in the absence of hunger. *Physiology & behavior*. 2011;103(2):157-163.

19. Herhaus B, Ullmann E, Chrousos G, Petrowski K. High/low cortisol reactivity and food intake in people with obesity and healthy weight. *Translational psychiatry*. 2020;10(1):1-8.

20. Nagy MR, Gill A, Adams T, et al. Stress-Induced Suppression of Food Intake in Overweight and Obese Adolescents. *Psychosomatic Medicine*. 2019;81(9):814-820. doi:10.1097/PSY.0000000000000732

21. Appelhans BM, Pagoto SL, Peters EN, Spring BJ. HPA axis response to stress predicts short-term snack intake in obese women. *Appetite*. 2010;54(1):217-220. doi:10.1016/j.appet.2009.11.005

22. Pereira RF, Alvarenga M. Disordered Eating: Identifying, Treating, Preventing, and Differentiating It From Eating Disorders. *Diabetes Spectrum*. 2007;20(3):141-148. doi:10.2337/diaspect.20.3.141

23. Goldschmidt AB, Aspen VP, Sinton MM, Tanofsky-Kraff M, Wilfley DE. Disordered eating attitudes and behaviors in overweight youth. *Obesity*. 2008;16(2):257-264.

24. Neumark-Sztainer DR, Wall MM, Haines JI, Story MT, Sherwood NE, van den Berg PA. Shared risk and protective factors for overweight and disordered eating in adolescents. *American journal of preventive medicine*. 2007;33(5):359-369.

25. Field AE, Austin S, Taylor C, et al. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics*. 2003;112(4):900-906
.

26. Stice E, Presnell K, Shaw H, Rohde P. Psychological and behavioral risk factors for obesity onset in adolescent girls: a prospective study. *Journal of consulting and clinical psychology*. 2005;73(2):195.

27. Tanofsky-Kraff M, Cohen ML, Yanovski SZ, et al. A prospective study of psychological predictors of body fat gain among children at high risk for adult obesity. *Pediatrics*. 2006;117(4):1203-1209.

28. Libbey HP, Story MT, Neumark-Sztainer DR, Boutelle KN. Teasing, disordered eating behaviors, and psychological morbidities among overweight adolescents. *Obesity*. 2008;16(S2):S24-S29.

29. Ajibewa TA, Zhou M, Barry MR, et al. Adolescent stress: A predictor of dieting behaviors in youth with overweight/obesity. *Appetite*. 2020;147:104560.

30. Nelson DS, Gerras JM, McGlumphy KC, et al. Racial discrimination and low household education predict higher body mass index in African American youth. *Childhood Obesity*. 2018;14(2):114-121.

31. Rockett HRH, Wolf AM, Colditz GA. Development and Reproducibility of a Food Frequency Questionnaire to Assess Diets of Older Children and Adolescents. *Journal of the American Dietetic Association*. 1995;95(3):336-340. doi:10.1016/S0002-8223(95)00086-0

32. Rockett HRH, Breitenbach M, Frazier AL, et al. Validation of a Youth/Adolescent Food Frequency Questionnaire. *Preventive Medicine*. 1997;26(6):808-816. doi:10.1006/pmed.1997.0200

33. Þórarinsdóttir H, Faurholt-Jepsen M, Ullum H, Frost M, Bardram JE, Kessing LV. The Validity of Daily Self-Assessed Perceived Stress Measured Using Smartphones in Healthy Individuals: Cohort Study. *JMIR Mhealth Uhealth*. 2019;7(8). doi:10.2196/13418

34. Brantley PJ, Waggoner CD, Jones GN, Rappaport NB. A daily stress inventory: Development, reliability, and validity. *J Behav Med*. 1987;10(1):61-73. doi:10.1007/BF00845128

35. Moskow DM, Addington J, Bearden CE, et al. The relations of age and pubertal development with cortisol and daily stress in youth at clinical risk for psychosis. *Schizophrenia Research*. 2016;172(1):29-34. doi:10.1016/j.schres.2016.02.002

36. Tessner KD, Mittal V, Walker EF. Longitudinal Study of Stressful Life Events and Daily Stressors Among Adolescents at High Risk for Psychotic Disorders. *Schizophr Bull*. 2011;37(2):432-441. doi:10.1093/schbul/sbp087

37. Harris C, Flexeder C, Thiering E, et al. Changes in dietary intake during puberty and their determinants: results from the GINIplus birth cohort study. *BMC Public Health*. 2015;15. doi:10.1186/s12889-015-2189-0

38. Laugero KD, Falcon LM, Tucker KL. Relationship between perceived stress and dietary and activity patterns in older adults participating in the Boston Puerto Rican Health Study. *Appetite*. 2011;56(1):194-204. doi:10.1016/j.appet.2010.11.001

39. Xie B, Gilliland FD, Li Y-F, Rockett HR. Effects of Ethnicity, Family Income, and Education on Dietary Intake among Adolescents. *Preventive Medicine*. 2003;36(1):30-40. doi:10.1006/pmed.2002.1131

40. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of youth and adolescence*. 1988;17(2):117-133.

41. Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. The Eating Attitudes Test: psychometric features and clinical correlates. *Psychological Medicine*. 1982;12(4):871-878. doi:10.1017/S0033291700049163

42. McEwen C, Flouri E. Fathers' parenting, adverse life events, and adolescents' emotional and eating disorder symptoms: the role of emotion regulation. *Eur Child Adolesc Psychiatry*. 2009;18(4):206-216. doi:10.1007/s00787-008-0719-3

43. Attie I, Brooks-Gunn J. Development of eating problems in adolescent girls: A longitudinal study. *Developmental Psychology*. 1989;25(1):70-79. doi:10.1037/0012-1649.25.1.70

44. Edwards CG, Walk AM, Thompson SV, Mullen SP, Holscher HD, Khan NA. Disordered Eating Attitudes and Behavioral and Neuroelectric Indices of Cognitive Flexibility in Individuals with Overweight and Obesity. *Nutrients*. 2018;10(12):1902. doi:10.3390/nu10121902

45. Naggara O, Raymond J, Guilbert F, Roy D, Weill A, Altman DG. Analysis by Categorizing or Dichotomizing Continuous Variables Is Inadvisable: An Example from the Natural History of Unruptured Aneurysms. *American Journal of Neuroradiology*. 2011;32(3):437-440. doi:10.3174/ajnr.A2425

46. Royston P, Altman DG, Sauerbrei W. Dichotomizing continuous predictors in multiple regression: a bad idea. *Statistics in Medicine*. 2006;25(1):127-141. doi:10.1002/sim.2331

47. Ervin RB, Ogden CL. *Trends in Intake of Energy and Macronutrients in Children and Adolescents from 1999-2000 through 2009-2010*. US Department of Health and Human Services, Centers for Disease Control and …; 2013.

48. McGlumphy KC, Shaver ER, Ajibewa TA, Hasson RE. Perceived stress predicts lower physical activity in African-American boys, but not girls. *American journal of health behavior*. 2018;42(2):93-105.

49. Zerwas S, Holle AV, Watson H, Gottfredson N, Bulik CM. Childhood anxiety trajectories and adolescent disordered eating: Findings from the NICHD study of early child care and youth

development. *International Journal of Eating Disorders*. 2014;47(7):784-792. doi:https://doi.org/10.1002/eat.22318

50. Cartwright M, Wardle J, Steggles N, Simon AE, Croker H, Jarvis MJ. Stress and dietary practices in adolescents. *Health psychology*. 2003;22(4):362.

51. Jeong E-Y, Kim K-N. Influence of stress on snack consumption in middle school girls. *Nutr Res Pract*. 2007;1(4):349-355. doi:10.4162/nrp.2007.1.4.349

52. Kim Y, Yang HY, Kim A-J, Lim Y. Academic stress levels were positively associated with sweet food consumption among Korean high-school students. *Nutrition*. 2013;29(1):213-218. doi:10.1016/j.nut.2012.08.005

53. Michaud C, Kahn JP, Musse N, Burlet C, Nicolas JP, Mejean L. Relationships between a critical life event and eating behaviour in high-school students. *Stress Medicine*. 1990;6(1):57-64. doi:10.1002/smi.2460060112

54. Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. *Nutrition*. 2007;23(11-12):887-894.

55. Epel ES, Tomiyama AJ, Dallman MF. Stress and reward. *Food and Addiction: A Comprehensive Handbook*. 2012;266.

56. Wiium N, Breivik K, Wold B. Growth Trajectories of Health Behaviors from Adolescence through Young Adulthood. *International Journal of Environmental Research and Public Health*. 2015;12(11):13711-13729. doi:10.3390/ijerph121113711

57. Major B, Eliezer D, Rieck H. The psychological weight of weight stigma. *Social Psychological and Personality Science*. 2012;3(6):651-658.

58. Goldschmidt AB, O’Brien S, Lavender JM, Pearson CM, Le Grange D, Hunter SJ. Executive functioning in a racially diverse sample of children who are overweight and at risk for eating disorders. *Appetite*. 2018;124:43-49. doi:10.1016/j.appet.2017.03.010

59. Duckworth AL, Kim B, Tsukayama E. Life Stress Impairs Self-Control in Early Adolescence. *Front Psychol*. 2013;3. doi:10.3389/fpsyg.2012.00608

60. Miller AL, Gearhardt AN, Fredericks EM, et al. Targeting self-regulation to promote health behaviors in children. *Behaviour Research and Therapy*. 2018;101:71-81. doi:10.1016/j.brat.2017.09.008

61. Williams DR. Stress and the mental health of populations of color: Advancing our understanding of race-related stressors. *Journal of health and social behavior*. 2018;59(4):466-485.

62. Araujo MC, Yokoo EM, Pereira RA. Validation and Calibration of a Semiquantitative Food Frequency Questionnaire Designed for Adolescents. *Journal of the American Dietetic Association*. 2010;110(8):1170-1177. doi:10.1016/j.jada.2010.05.008

63. Slater B, Philippi ST, Fisberg RM, Latorre MRDO. Validation of a semi-quantitative adolescent food frequency questionnaire applied at a public school in São Paulo, Brazil. *European Journal of Clinical Nutrition*. 2003;57(5):629-635. doi:10.1038/sj.ejcn.1601588

64. Adam TC, Epel ES. Stress, eating and the reward system. *Physiology & Behavior*. 2007;91(4):449-458. doi:10.1016/j.physbeh.2007.04.011

65. Hill JO, Peters JC, Wyatt HR. Using the Energy Gap to Address Obesity: A Commentary. *Journal of the American Dietetic Association*. 2009;109(11):1848-1853. doi:10.1016/j.jada.2009.08.007

66. Sundgot-Borgen J, Torstveit MK. Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine & Science in Sports*. 2010;20(s2):112-121. doi:https://doi.org/10.1111/j.1600-0838.2010.01190.x

67. Bor W, Dean AJ, Najman J, Hayatbakhsh R. Are child and adolescent mental health problems increasing in the 21st century? A systematic review. *Aust N Z J Psychiatry*. 2014;48(7):606-616. doi:10.1177/0004867414533834

68. de Vet E, de Wit JB, Luszczynska A, et al. Access to excess: how do adolescents deal with unhealthy foods in their environment? *The European Journal of Public Health*. 2013;23(5):752-756.

69. Puhl RM, Himmelstein MS, Gorin AA, Suh YJ. Missing the target: including perspectives of women with overweight and obesity to inform stigma-reduction strategies. *Obesity Science & Practice*. 2017;3(1):25-35. doi:https://doi.org/10.1002/osp4.101

Chapter 4

Psychological Stress and Insulin Sensitivity in Non-Hispanic Black and White Adolescents with Overweight/Obesity

Preamble

The **original study aims** at the time of the dissertation proposal for study 3 was as follows: "To examine biobehavioral pathways (physical activity, caloric intake, cortisol patterning) that link psychological stress to a metabolic risk factor (insulin resistance) in adolescents ages 14-19 with overweight and obesity." Study aims have however been revised as the manuscript was put together over the past 2 years since the original dissertation proposal.

Psychological stress and insulin sensitivity in non-Hispanic Black and White adolescents with overweight/obesity is the third study of the dissertation. It is a study that examined the cross-sectional racial differences in the association between psychological stress, insulin sensitivity (S_i), acute insulin response (AIR_g), and disposition index (DI) among non-Hispanic Black and White adolescents with overweight/obesity.

The following manuscript will be submitted for peer review in the Journal of Clinical Endocrinology and Metabolism.

Abstract

INTRODUCTION: Racial differences in type 2 diabetes risk persist among non-Hispanic Black and White adolescents with overweight/obesity; however, the role of psychological stress in this disparity is less clear. **PURPOSE:** To examine racial differences in the association between psychological stress, insulin sensitivity (S_i), acute insulin response to glucose (AIR_g), and disposition index (DI) among Black and White adolescents with overweight/obesity.

METHODS: Ninety-six adolescents (60% female; 51% non-Hispanic Black; 16.6±1.8 years of age) with overweight/obesity (BMI percentile ≥ 85^{th} percentile) were included in this analysis. Psychological stress was assessed using the 14-item Perceived Stress Scale. Glucose and insulin data from an intravenous glucose tolerance test was modeled to obtain S_i, AIR_g, and DI. Multivariable linear regression models were used to examine the association between race, psychological stress and metabolic outcomes. **RESULTS:** Race was a significant predictor of log-AIR_g and log-DI (*ps*<0.001) independent of all covariates in the main effects models. Lower S_i was also observed among Black adolescents who reported higher stress levels, whereas higher S_i was observed among White adolescents reporting higher stress ($p_{interaction}$= 0.022) in the race interaction models. However, race did not moderate the association between psychological stress and other metabolic outcomes (*ps*>0.05), nor was stress associated with S_i, AIR_g, or DI (*ps*>0.05) across all other models. **CONCLUSIONS**: These findings suggest that psychological stress may play an important and distinct role in shaping racial differences in S_i among adolescents with overweight/obesity. Additional research is needed to understand the long-term effects of psychological stress on metabolic health among Black and White adolescents with overweight/obesity.

Introduction

Lower insulin sensitivity (S_i) and hyperinsulinemia have been consistently observed among non-Hispanic Black children and adolescents compared with their non-Hispanic White peers [1–3]. Lindquist and colleagues noted that Black children had lower insulin sensitivity (S_i) and greater acute insulin response to glucose (AIR_g) compared to White children. This disparity remained significant after controlling for body composition, social class, and diet[1]. Likewise, Ku et al. observed lowered S_i and greater AIR_g among Black children compared with White children even after adjusting for body composition and physical activity participation[2]. Hannon and colleagues observed higher first phase insulin response and disposition index (DI) among Black adolescents compared to their White counterparts even with comparable S_i[3]. Together, these studies highlight the heightened risk of type 2 diabetes (T2D) and early onset of disease among non-Hispanic Black adolescents, and underscores the pressing need to understand the underlying mechanisms that contribute to this disparity.

Given the evidence that biological and behavioral factors do not fully explain differences in T2D risk, it is important to consider how the social environment and, in particular, psychological stress may contribute to racial differences. Psychological stress is classically defined as occurring when an individual experiences or perceives demands or threats that exceeds their resources or adaptive capacity[4,5]. In response to a psychological stressor, corticotrophin-releasing hormone (CRH) is released from the hypothalamus, eliciting the release of adrenocorticotropic hormone (ACTH) from the pituitary gland into circulation. The release of ACTH into circulation thereafter leads to the signaling of cortisol secretion from the adrenal cortex[6]. This relatively short-term response to stress provides energy and vigilance to deal with the stressor. Chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis in response to psychological stress can be assessed by higher morning cortisol levels or disrupted cortisol patterning[7]. Furthermore, the dysregulation of the stress response system has been associated with pancreatic beta-cell dysfunction and insulin resistance[8]. To this end, Adam and colleagues conducted a study among Hispanic children and adolescents with overweight, wherein they observed that higher baseline levels of morning cortisol was associated with a greater decrease in S_i longitudinally[8]. In the same study, the authors also observed a cross-sectional association between higher levels of cortisol and lower AIR_g and DI—further highlighting the potential metabolic dysregulation resulting from disruptions to the HPA axis, presumably stemming from chronic psychological stress[9].

Racial differences in the levels and sources of chronic psychological stress have also been previously observed between non-Hispanic Blacks and Whites. Data from the adult literature has documented higher cumulative levels of stress among non-Hispanic Blacks compared to their non-Hispanic White peers, stemming in part from higher levels of financial stress, stressful life events, and chronic stress due to racial discrimination ([10–12]). Similarly, among pediatric populations, greater exposure to community violence, racial and institutional discrimination, and traumatic life events have been documented in non-Hispanic Black children and adolescents compared with their non-Hispanic White counterparts (e.g.[13–15]). Not only are there racial differences in levels and sources of stress among non-Hispanic White and Black adolescents, emerging evidence also suggests that increased stress is associated with increased T2D risk. Specifically, evidence from both adults and adolescents of various races have found that higher levels of psychological distress in the forms of perceived stress and depression are linked with increased insulin resistance and abnormal glucose metabolism[16–19]. Considering the heightened stress exposure documented among non-Hispanic Blacks, it is plausible that higher levels of psychological stress may in turn dysregulated biological stress responses, resulting in the observed racial differences in T2D risk among non-Hispanic Black and White adolescents.

Given the elevated risk for T2D as well as heightened levels of psychosocial stress among Black adolescents, this study sought to examine racial differences in the association between psychological stress and measures of T2D risk (S_i, DI, and AIR_g—assessed via an intravenous glucose tolerance test or IVGTT) among non-Hispanic White and Black adolescents with overweight/obesity. It was hypothesized that Black race and increased psychological stress would be associated with decreased S_i, DI, and increased AIR_g among sample participants, and that the association between psychological stress and metabolic outcomes would be modified by race.

Methods

Recruitment and study participants

Adolescents with overweight/obesity between the ages of 14-19 years old from Southeast Michigan—primarily from the local cities of Ann Arbor and Ypsilanti, MI, were recruited to participate in the Stress, Obesity, and Diabetes in Adolescents (SODA) study. The primary aim of the laboratory- and clinically-based SODA study was to examine cross-sectional associations between different indices of stress (exposure, perception, physiological response) on T2D risk in Hispanic, non-Hispanic Black, and White adolescents with overweight/obesity. Participants were excluded from the SODA study if: (i) they were currently involved, or had been involved, in a weight loss or exercise program; (ii) they were taking medications known to influence body composition or metabolism; (iii) they had a previous history of metabolic diseases such as diabetes; (iv) they had a clinical diagnosis of depression or any other mental health disorder that may influence mood, emotions or stress perception; (v) they had any food allergies, intolerances, and other dietary restrictions; or (vi) they were pregnant. Prior to all data collection, written informed assent was obtained from all participants under the age of 18 years, and written informed consent obtained from each of their caregivers. For participants 18 years of age or older, only written informed consent was requested and obtained. Prior to the initiation of the study, all experimental procedures were approved by the University of Michigan Institutional Review Board (HUM00083264).

Data collection for the SODA study began in 2014 and was completed in 2017. Data collection and analysis took place in the Childhood Disparities Research Laboratory (CDRL) at the University of Michigan, and at the Michigan Clinical Research Unit of the University of Michigan Hospital. Upon inclusion into the study, participants came to the CDRL and the University of Michigan Clinical Research Unit on two separate occasions (visit 1 and 2), to complete the SODA study (see Fig. 1). Visit 1 consisted of participants completing demographic, psychological, and health behavior questionnaires as well as anthropometric measurements at the CDRL. Visit 2 consisted of participants completing physiological measures at the Michigan Clinical Research Unit (MCRU) via an insulin-modified IVGTT with the purpose of examining measures of T2D risk. Blood plasma samples were collected as part of the IVGTT, following a twelve-hour fast, and all participants were asked to refrain from strenuous physical activity. A total of 171 participants completed questionnaires at visit 1, and of the 171 participants, 99 participants completed physiological measurements at visit 2. Reasons for decreased enrollment at visit 2 included: voluntarily opting out of this portion of the study, lost to follow up, unsuccessful collection of blood samples during the IVGTT due to non-adherence to fasting restrictions, blood collection challenges prior to or during blood collection (see Fig. 2). Due to low enrollment of Hispanic participants (n=10), these participants were excluded from the final analysis and only non-Hispanic Black and White adolescents were included. A total of 96 participants with complete IVGTT data were included in this analysis.

Independent variable

Psychological stress was measured using the perceived stress scale (PSS-14)[20]. The PSS is a 14-item, self-reported questionnaire that assessed each participant's ability to cope with stressors in their lives over the past month. An example question from the PSS-14 included: *"In the last month, how often have you felt nervous and 'stressed'"* with answers anchored by 'never' (0) to 'very often' (4). Answers to positively worded questions were reverse-scored (e.g. 0=4, 1=3, 2=2, 3=1, 4=0), and the responses to all 14 questions were added together to obtain the total psychological stress score. Possible scores ranged from 0 to 56, with higher scores indicating higher psychological stress. The perceived stress scale is a validated and reliable instrument that has been previously used in adolescent samples[21]. The PSS-14 was found to have good internal consistency (Cronbach alpha=0.76). Participant self-reported race was also included as a primary independent variable.

Dependent variables

S_i, AIR_g, and DI were derived from plasma samples during the IVGTT. S_i is a measure that assesses how sensitive cells of the muscle, fat, and liver are to insulin-mediated glucose disposal in the presence of insulin[22]. S_i values obtained from the IVGTT that are $\leq 2 \times 10^{-4} min^{-1}/(\mu lU/ml)$ are reflective of insulin resistance[23,24]. AIR_g is a measure of first phase insulin secretion (i.e. initial response to rise in glucose) and is a marker of early beta-cell compensation[25]. DI is the product of both insulin sensitivity and insulin secretion, and is a composite measure beta-cell function[26].

Baseline plasma samples were collected twice at 15-, and 5-minutes before glucose infusion at time 0, and again twelve additional times during the IVGTT: +2, +4, +8, +19, +20, +22, +30, +40, +50, +70, +100, and +180 minutes in 3 mL EDTA tubes and placed on ice. Plasma samples were then centrifuged at 2000 g, divided into 2 aliquots and stored at -80˚C until analysis. Samples were then analyzed at the Michigan Diabetes Research Center's Clinical Core Chemistry Laboratory using two separate glucose and insulin assays.

The glucose assay is a hexokinase method that is run on a Randox RX Series Daytona chemistry analyzer [Glucose Assay (GLUC-HK) Product Insert, RX Series GL 3816, Randox Laboratories Limited, U.K.]. The analyzer was calibrated using 0.9 NaCl as a zero calibrator and Randox Calibration Serum level 3. The assay is linear up to a glucose concentration of 639 mg/dl and the minimum detectable concentration of glucose with an acceptable level of precision was determined to be 11.9 mg/dl. Intra assay precision was determined at three levels (n=20): 3.47% (31.5 mg/dL), 3.37% (103 mg/dL), and 2.57% (297 mg/dL). Inter-assay precision was determined at three levels (n=20): 3.80% (31.1 mg/dL), 1.33% (103 mg/dL), and 1.71% (286 mg/dL). The insulin assay, a double-antibody radioimmunoassay using a 125I-Human insulin tracer (Linco Research), a guinea pig anti-porcine insulin first antibody (MDRTC, 68.5% cross-reaction to human proinsulin), and a goat anti-guinea pig gamma globulin (Antibodies Inc.)-PEG second antibody and standardized against the Human Insulin International Reference Preparation (NIBSC) was used. Limit of sensitivity for the assay is 2.1 µU/ml, while the inter-assay and intra-assay variabilities are 3.8% and 2.7% at 25 µU/ml, respectively[27].

The minimal model of glucose regulation (MINMOD) is a mathematical model formulated by Bergman and colleagues that was used in conjunction with the IVGTT to calculate S_i, DI, and AIR_g[25]. The MINMOD model has been classically used to describe the physiological mechanism underlying plasma insulin and glucose regulation within the body[28]. Together with the IVGTT, the MINMOD allows for the analysis of the combined effects of insulin secretion and S_i on risk for T2D, through a mathematical representation of both the first phase—pancreatic response to glucose infusion, and the second phase—exogenous response following the infusion of exogenous insulin.

Covariates

Anthropometric measures of height and weight were measured in the laboratory by trained study staff during visit 1. Additionally, body fat percentage was measured using DEXA whole-body scans (GE-Healthcare Lunar Prodigy Advance DXA, Madison, WI). All scans were performed and analyzed by a certified technician. All participants were also asked to self-report whether their development was earlier or later than their peers, using the pubertal development scale (PDS)[29]. Based on the self-reported completion of developmental stages on the PDS, pubertal category scores were summed and categorized into the following categories: pre-pubertal= 3, early pubertal= 4 or 5, mid-pubertal= 6-8, late pubertal= 9-11, or post-pubertal= 12 for each participant's respective sex. Household education was assessed using a seven-category variable: less than 8th grade= 1, (2) finished 8th grade= 2, some high school= 3, high school graduate/GED= 4, some college or vocational school (at least 1 year)= 5, college graduate= 6, or graduate or professional training= 7. Caregivers of participants completed the questionnaire to determine household education. For households with multiple caretakers, the highest parental education score was used. Between visit 1 and 2, participants were asked to collect five saliva samples per day over a 3 day period using 2 mL SalivaBio Cryovials (Passive Drool; Salimetrics LLC, State College, PA). Participants were asked to collect saliva samples at the following times: (1) immediately after waking, (2) 30 min after waking, (3) between 3−4:00 pm, (4) 6−7:00 pm, and (5) 9−10:00 pm. Samples were analyzed by Salimetrics' SalivaLab (Carlsbad, CA), where cortisol levels were determined using the Salimetrics high-sensitive cortisol assay kit (Salimetrics, Carlsbad, CA), without modifications to the manufacturers' protocol. Baseline cortisol at awakening was used as a covariate within the present study, and was calculated as the cortisol concentration at awakening averaged over the three-day period. Race, ethnicity, and biological sex of each participant was self-reported, with non-Hispanic White and male as reference group.

Statistical Analysis

All statistical analyses were performed using Stata 16.0 (StataCorp, TX). Prior to all analyses, outcome variables (S_i, DI, AIR_g) were evaluated for normality. Due to the skewness of all three variables, all were natural-log transformed to help normalize their distribution and improve linear regression assumptions. Means for the total sample and mean differences by race were determined for age, BMI, BMI percentile, body fat percentage, pubertal development, household education, and perceived stress using an independent t-test. The statistical procedure of multiple imputation was used to account for missing data using chained regression equations with 100 data sets imputed[30]. Most study variables had complete data or ≤10% missing data. The variable with the highest fraction of missing data was household education, with 50% of the data missing at random.

For the main effect models, multivariable linear regression was used to examine the association between the independent variable (psychological stress; race) and the dependent variables (log-S_i, log-AIR_g, and log-DI). For the interaction models, the moderating effects of race in the association between the psychological stress and metabolic outcomes (log-S_i, log-AIR_g, and log-DI) were tested by adding a race interaction term to psychological stress (e.g.

psychological stress*race). Given that S_i is a major determinant of pancreatic beta-cell function, S_i was included as a covariate in the regression models predicting AIR_g and DI[31]. A p-value of $p<0.05$ was set *a priori* to determine significance for all models.

Results

Participant characteristics

Participant characteristics are displayed in mean and standard deviation and percentage in Table 1. Mean age of participants was 16.6±1.8 years, with 60% of the sample self-identifying as female (40% male), and 51% as non-Hispanic Black (49% non-Hispanic White). Mean household education score was 6.1±0.9, indicating that most households had at least one caregiver who had graduated college. Mean BMI percentile was 93.67% (BMI: 30.6±5.8 kg/m^2), while total body fat was 31.6±13.9 kg. In total, 50% of participants were classified as having overweight (BMI percentile 85^{th} - 94^{th}), and 50% as having obesity (BMI percentile $\geq 95^{th}$). Mean S_i for the sample was $2.74 \pm 2.12 \times 10^{-4} min^{-1}/(\mu lU/ml)$, suggesting that the majority of adolescents in this sample were insulin resistant. Racial differences were observed among participants. Specifically, non-Hispanic Black participants had, on average, significantly higher AIR_g (non-Hispanic Blacks: 1447.5±1064.1 vs. non-Hispanic Whites: 921.4±656.6 μU/ml; *p*=0.004) and DI (non-Hispanic Blacks: 3301.1±4324.9 vs. non-Hispanic Whites: 1853.3±963.7 $x10^{-4}/min^{-1}$; *p*=0.025) compared to their non-Hispanic White peers, indicating possible pancreatic compensation in response to their insulin resistance. Mean PSS-14 for the total sample and by race was approximately 22, with a range of 7 to 40, suggesting that stress levels were moderate across the sample (based on a tertile classification of low, moderate, and high stress from possible scores of 0-56).

Psychological stress and the moderating effect of race/ethnicity on metabolic outcomes

Table 2 displays the associations between psychological stress, race, S_i, AIR_g, and DI. Psychological stress was not associated with log-S_i (β=0.004±0.01, p=0.75), log-AIR_g (β= -0.007±0.01, p=0.44), or log-DI (β= -0.007±0.01, p=0.44) when controlling for covariates and S_i, where appropriate. Race was also associated with log-AIR_g (β=0.49±0.12, p<0.001) and log-DI (β=0.49±0.12, p<0.001) independent of all other covariates.

Figure 3 illustrates the interaction model examining the moderating effect of race/ethnicity on the association between psychological stress and Si. It was observed that race/ethnicity modified the association between stress and Si ($p_{interaction}$= 0.022), where non-Hispanic Black adolescents had significantly lower Si compared with their non-Hispanic White counterparts. Race did not modify the association between stress and AIR_g, nor the association between stress and DI (ps>0.05).

DISCUSSION

This study sought to examine racial differences in the association between psychological stress and measures of T2D risk (S_i, AIR_g, DI) in non-Hispanic Black and White adolescents with overweight/obesity. When both non-Hispanic Black and White participant data were analyzed together, no association between psychological stress and measures of T2D risk were seen; however, race did independently predict T2D risk factors. Racial differences were observed with AIR_g and DI, with non-Hispanic Black participants displaying a 44.4% higher AIR_g and a 56.2% higher mean DI compared to their non-Hispanic White peers. When race was tested as a moderator, it played a unique and distinct role in the association between psychological stress and S_i. Specifically, higher levels of psychological stress were associated with lower S_i among non-Hispanic Black adolescents, whereas higher levels of stress were associated with higher S_i

among non-Hispanic White adolescents. Together, these findings suggest that psychological stress may have a differential impact on metabolic dysfunction and T2D risk among Black and White adolescents with overweight/obesity.

Our finding that higher psychological stress was associated with lower S_i among non-Hispanic Black adolescents is consistent with prior studies demonstrating associations between psychological distress (a negative emotional state that results from perception of stress) and decreased S_i in pediatric and adolescent samples[18,19]. Shomaker et al. observed, among adolescents ages 12-18 years across all weight statuses, that increased levels of psychological distress (specifically depression) was associated with decreased S_i (as measured using the quantitative insulin sensitivity check index—QUICKI); this association remained significant even when controlling for factors such as age, fat mass, and pubertal status[18]. In a longitudinal study by the same author in a sample of children and adolescents ages 6-13, higher baseline psychological distress was associated with greater insulin resistance (via HOMA-IR) six years later[19]. The association between higher psychological stress and lower S_i among non-Hispanic Black participants presently may have been influenced by the participants' stress appraisal. Specifically, the degree to which an individual appraises a stressor or situation as a threat (i.e. stressor that is too demanding) or as a challenge, is dependent on their experiences of stress (both past and present), as well as their individual characteristics (e.g., personality) and the scope of the stressor (e.g. how dangerous the stressor or situation is)[32]. Evidence suggests that there are racial differences in relation to the amount and type of stressors experienced by individuals, even among adolescent populations. For example, there is substantial evidence that suggests that non-Hispanic Black adolescents are exposed to stressors stemming from violence, traumatic stressful life events, as well as institutional discrimination, at higher rates compared to their non-Hispanic

White counterparts (e.g.[13–15,33]). It is plausible that the higher levels of 'threat' type stress exposure among non-Hispanic Black adolescents compared to their non-Hispanic White peers may have contributed to the differential relationship observed between psychological stress and S_i. Given that there were no significant differences in perceived stress scores in the current analysis, it is likely that the Perceived Stress Scale may not have captured differences in the specific types or appraisal of psychological stress. Moreover, the biological stress marker cortisol was also not significant within our statistical models, pointing again to the role of other possible biological and psychological mechanisms. Thus, additional research is needed to disentangle the interrelationships between amount and types of stress exposure, stress appraisal, and metabolic risk among non-Hispanic Black and White adolescents with overweight/obesity.

The finding that higher psychological stress was associated with greater insulin sensitivity among non-Hispanic White adolescents was unexpected. Nevertheless, there are a few plausible explanations for this finding. The impact of social support—both structural and functional support—and the indirect effect of positive social relationships may have influenced the association between psychological stress and S_i among non-Hispanic White adolescents. It is plausible that increased social support acts as a mechanism that dampens stress responses through pathways that include but are not limited to instrumental (i.e. access and utilization of services to cope with a stressor), emotional (i.e. providing feelings or self-worth, usefulness to help cope with a stressor), and informational assistance (i.e. giving advice, talking through a stressor)[34,35]. These forms of social support together or individually may in turn work to not only buffer the impact of stress physiologically, but also enhance resilience over time among non-Hispanic White adolescents. Among adults there is an emerging body of evidence indicating that higher levels of structural (i.e. social networks) and functional (i.e. quality of social relationships) support confer metabolic benefits such as decreased risk of T2D (e.g.[36,37]). Much less is known about the role of social support on metabolic benefits among children and adolescents. Nevertheless, a recent longitudinal study by Gangel and colleagues examining children's positive social relationships and their effects on adolescents' T2D risk showed positive benefits. Specifically, the authors found that higher social preference (level of being accepted vs rejected by peers) at age 7 was associated with reduced T2D risk (HOMA-IR) at age 16[38]. The authors noted that higher social preference may lead to decreased T2D risk via behavioral and biological pathways. Higher social acceptance by friends may allow for children to be able to spend more time with their friends engaging in physical activities such as sports, in turn, reducing T2D risk[39]. Similarly, increased social support may lead to physiological increases in favorable hormones such as oxytocin and decreases in other hormones such as cortisol; altogether reducing risk of T2D physiologically[38]. With recent evidence pointing to possible racial differences in types of social support—particularly regarding social networks[40,41], tangible support, and appraisal support[41]—it is plausible that compared to non-Hispanic Black adolescents, White adolescents may have had greater access to social support resources when facing stress earlier in their development. This is particularly relevant as extreme and toxic stress (e.g. chronic neglect, intrafamily violence) in comparison to manageable stress (i.e. where one has the necessary resources to cope) in early childhood can damage the developing brain and increase risk for dysregulation of the stress response system[42]. Over time, greater access to support resources may have provided buffering and promoted capacity to manage higher levels of stress later in development, contributing to the inverse relationship seen presently. More studies are needed to replicate our findings and to confirm the effect of social support on stress and metabolic function among adolescents and across different racial groups.

Unlike S_i, however, race did not significantly moderate the association between psychological stress and AIR_g or between psychological stress and DI. Nevertheless, there were significant differences in baseline levels of AIR_g and DI among non-Hispanic Black and White adolescents with overweight/obesity. The fact that race only moderated the relationship between stress and S_i and not AIR or DI in the current study, suggests a potential mechanism of tissue insensitivity to insulin as opposed to beta-cell dysfunction. More specifically, S_i is a measure of muscle, fat, and liver cell sensitivity to insulin, whereas the measures of AIR_g and DI are measures of insulin secretion and pancreatic beta-cell function, respectively. Given the lack of a significant difference in S_i among non-Hispanic Black and White adolescents coupled with the significant racial difference in the association between stress and S_i, it is possible that high psychological stress demands without the necessary resources will lead in part to a mechanistic disruption to tissue sensitivity to insulin. Our results together with the existing literature point once more to the differential action in metabolic function across race.

There are many strengths of this study that must be noted. Strengths include the use of a comprehensive clinical measurement of metabolic function (S_i, DI, AIR_g) and the inclusion of body fat percentage measured using Dual Energy X-ray Absorptiometry (DEXA)—a gold standard technique for measuring body composition in both research and clinical practice[43]. Furthermore, the diverse study sample and the use of a validated psychological stress questionnaire are other major strengths. However, a few limitations should be mentioned. Of note, the study is cross-sectional and therefore causality cannot be inferred. A large portion of missing data were noted for the household education variable in this analysis (50% of household education data were missing), however, multiple imputation was used to account for this missing data. Also, the inclusion of non-Hispanic Black and White adolescents with overweight/obesity limits the generalizability of these findings to other ethnic groups as well as to adolescents with lower weights. Additional limitations included self-reported pubertal development as opposed to a physical examination of pubertal development and a lack of social support or stress appraisal data. Despite these limitations, we were able to observe a significant racial difference between psychological stress and S_i among adolescents with overweight/obesity.

Conclusion

Adolescents with overweight and obesity are at increased risk for metabolic diseases such as T2D that maybe partly associated to psychosocial stressors. Findings from the present study add to the extant literature by highlighting the particularly harmful association between psychological stress on S_i for non-Hispanic Black/African American adolescents. Given the higher levels of stress exposure often experienced by non-Hispanic Black adolescents, these notable findings may inform researchers and behavioral interventionists to create innovative means to lower stress and increase stress buffers and resilience among non-Hispanic Black adolescents experiencing high levels of psychological stress. Given the positive association between stress and S_i among non-Hispanic White adolescents, interventions that leverage supportive positive relationships among family and friends may help buffer the influence of stress on both Black and White adolescents. Furthermore, enacting policies that address stressors within the built and social environment may in turn help decrease disparities often observed in T2D risk among children and adults in ethnic minority populations.

Tables and Figures

Table 4-1. Participant characteristics. Statistics are in mean and standard deviation (SD).

Characteristic		Total sample		Race/ethnicity			
			n	NHW	n	NHB	p-value
Age (years)	96	16.6 (1.8)	49	16.4±1.9	47	16.9±1.6	0.18
Sex (% female)	96	60%	49	59%	47	62%	0.80
BMI (kg/m^2)	96	30.6 (5.8)	49	30.1±5.7	47	31.1±5.8	0.40
BMI percentile (%)	96	93.7 (4.1)	49	93.4±4.1	47	93.9±4.1	0.60
Body fat (kg)	86	31.6 (13.9)	43	33.0±14.1	43	30.2±13.6	0.36
Pubertal category score	92	10.1 (1.9)	47	10.2±2.1	45	10.1±1.7	0.88
Perceived Stress Score	96	22.0 (6.7)	49	21.8±7.1	47	22.1±6.3	0.79
Si [($x10^{-4}/min^{-1}$)/(μU/ml)])	96	2.74 (2.1)	49	2.61±1.4	47	2.88±2.7	0.55
AIRg (μU/ml)	96	1179.0 (914.4)	49	921.4±656.6	47	1447.5±1064.1	**0.004***
DI ($x10^{-4}/min^{-1}$)	96	2562.1 (914.4)	49	1853.3±963.7	47	3301.1±4324.9	**0.025***

BMI; body mass index. NHW= non-Hispanic White; NHB= non-Hispanic Black.

Table 4-2. Table 2. Linear regression analysis for psychological stress, metabolic outcomes.

		Parameter estimate	SE	p-value
Model 1: Log-Si				
Psychological stress		0.004	0.01	0.75
Biological sex				
	Male	ref	ref	ref
	Female	0.348	0.22	0.11
Race				
	Non-Hispanic White	ref	ref	ref
	Non-Hispanic Black	-0.10	0.16	0.53
Body fat percentage		-4.20	1.05	<0.001*
Pubertal category score		0.06	0.05	0.24
Household education		0.002	0.10	0.99
Baseline cortisol at awakening		0.21	0.51	0.68
Model 2: Log-AIRg				
Psychological stress		-0.007	0.01	0.44
Biological Sex				
	Male	ref	ref	ref
	Female	-0.433	0.155	0.007
Race				
	Non-Hispanic White	ref	ref	ref
	Non-Hispanic Black	0.492	0.116	<0.001*
Body fat percentage		2.648	0.817	0.002
Pubertal category score		0.01	0.034	0.78
Household education		-0.012	0.074	0.87
Baseline cortisol at awakening		-0.004	0.376	0.99
Log-Si		-0.392	0.076	<0.001*
Model 3: Log-DI				
Psychological stress		-0.007	0.01	0.44
Biological Sex				
	Male	ref	ref	ref
	Female	-0.433	0.155	0.007
Race				
	Non-Hispanic White	ref	ref	ref
	Non-Hispanic Black	0.492	0.116	<0.001*
Body fat percentage		2.648	0.817	0.002
Pubertal category score		0.01	0.034	0.78
Household education		-0.012	0.074	0.87
Baseline cortisol at awakening		-0.004	0.376	0.99
Log-Si		0.608	0.076	<0.001*

Si= insulin sensitivity; AIR= acute insulin response; DI= disposition index*denotes p>0.05.

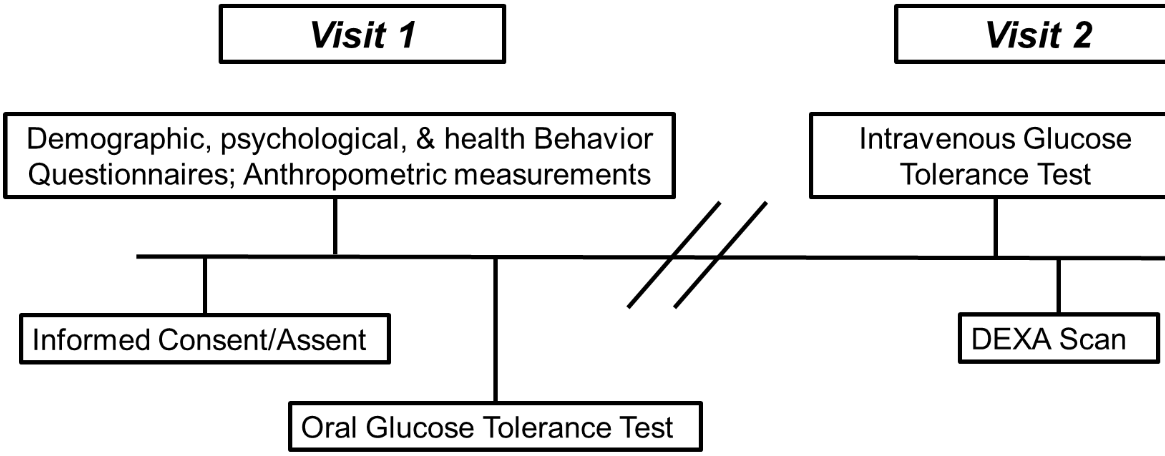

Figure 4.1. Study timeline and measures during visits 1 and 2.

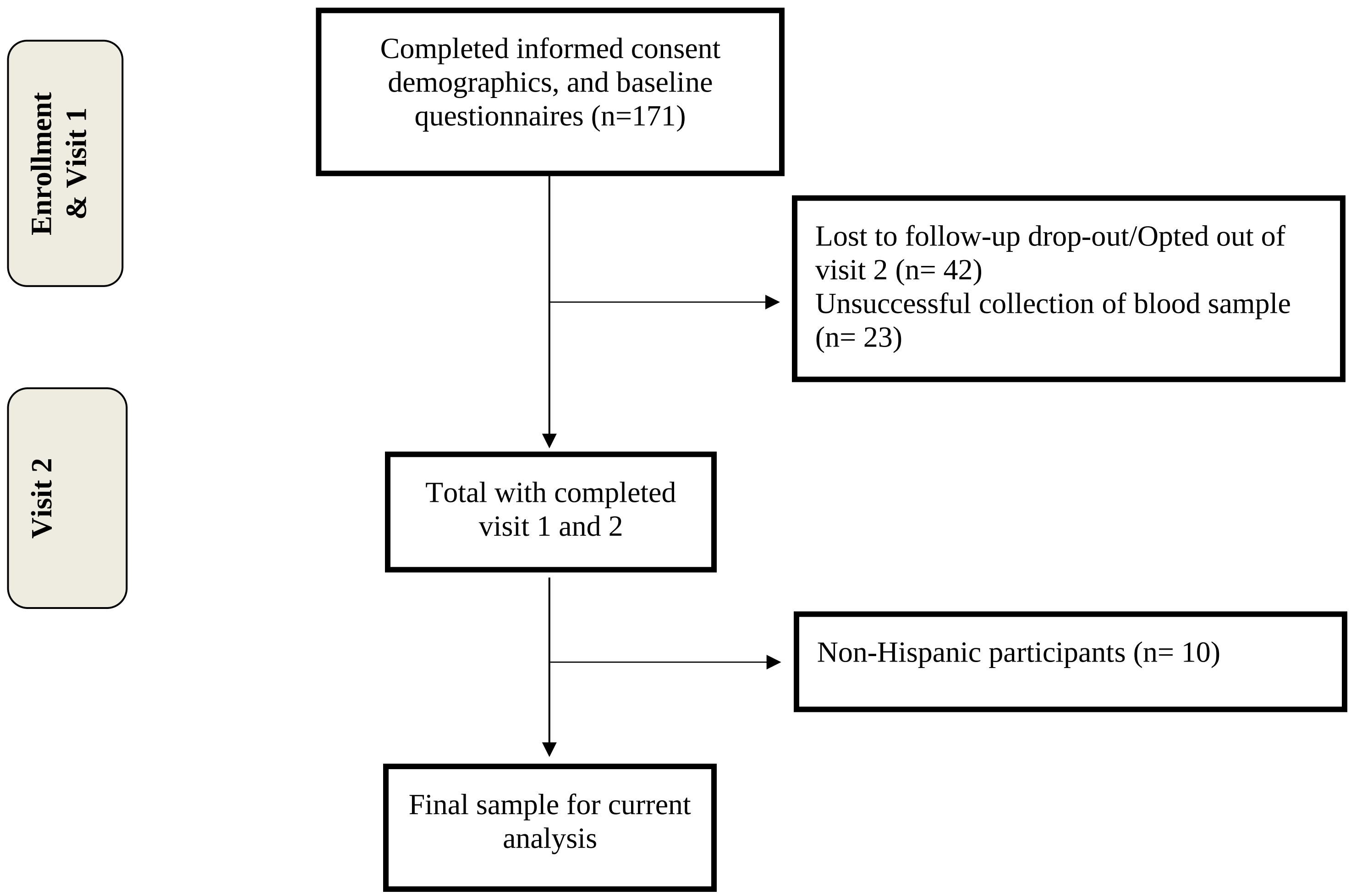

Figure 4.2. Participant enrollment and inclusion diagram

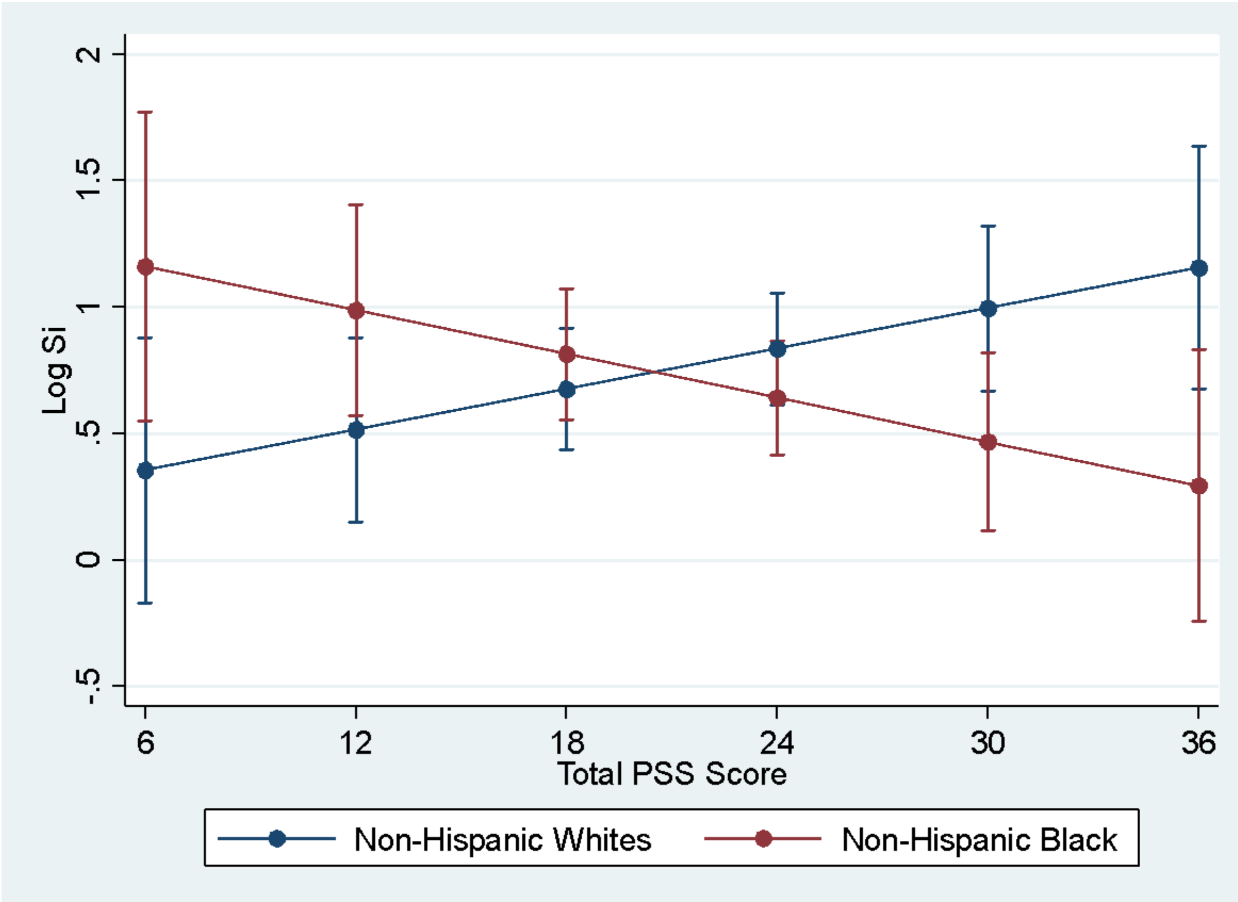

Figure 4.3. Racial difference in the association between psychological stress and Si.

REFERENCES

1. Lindquist CH, Gower BA, Goran MI. Role of dietary factors in ethnic differences in early risk of cardiovascular disease and type 2 diabetes. *The American journal of clinical nutrition*. 2000;71(3):725-732.

2. Ku C, Gower BA, Hunter GR, Goran MI. Racial differences in insulin secretion and sensitivity in prepubertal children: role of physical fitness and physical activity. *Obesity Research*. 2000;8(7):506-515.

3. Hannon TS, Bacha F, Lin Y, Arslanian SA. Hyperinsulinemia in African-American adolescents compared with their American white peers despite similar insulin sensitivity: a reflection of upregulated β-cell function? *Diabetes care*. 2008;31(7):1445-1447.

4. Lazarus RS, Folkman S. *Stress, Appraisal, and Coping*. Springer publishing company; 1984.

5. Cohen S, Janicki-Deverts D, Miller GE. Psychological Stress and Disease. *JAMA*. 2007;298(14):1685-1687. doi:10.1001/jama.298.14.1685

6. Tsigos C, Chrousos GP. Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of psychosomatic research*. 2002;53(4):865-871.

7. Duan H, Yuan Y, Zhang L, et al. Chronic stress exposure decreases the cortisol awakening response in healthy young men. *Stress*. 2013;16(6):630-637. doi:10.3109/10253890.2013.840579

8. Adam TC, Hasson RE, Ventura EE, et al. Cortisol is negatively associated with insulin sensitivity in overweight Latino youth. *The Journal of Clinical Endocrinology & Metabolism*. 2010;95(10):4729-4735.

9. McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiological reviews*. 2007;87(3):873-904.

10. Sternthal MJ, Slopen N, Williams DR. RACIAL DISPARITIES IN HEALTH: How Much Does Stress Really Matter? 1. *Du Bois review: social science research on race*. 2011;8(1):95.

11. Williams DR. Stress and the mental health of populations of color: Advancing our understanding of race-related stressors. *Journal of health and social behavior*. 2018;59(4):466-485.

12. Dowd JB, Palermo T, Chyu L, Adam E, McDade TW. Race/ethnic and socioeconomic differences in stress and immune function in The National Longitudinal Study of Adolescent Health. *Social Science & Medicine*. 2014;115:49-55.

13. Buka SL, Stichick TL, Birdthistle I, Earls FJ. Youth exposure to violence: Prevalence, risks, and consequences. *American journal of orthopsychiatry*. 2001;71(3):298-310.

14. Hatch SL, Dohrenwend BP. Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: A review of the research. *American journal of community psychology*. 2007;40(3-4):313-332.

15. Fisher CB, Wallace SA, Fenton RE. Discrimination distress during adolescence. *Journal of youth and adolescence*. 2000;29(6):679-695.

16. Novak M, Björck L, Giang K, Heden-Ståhl C, Wilhelmsen L, Rosengren A. Perceived stress and incidence of Type 2 diabetes: a 35-year follow-up study of middle-aged Swedish men. *Diabetic medicine*. 2013;30(1):e8-e16.

17. Williams ED, Magliano DJ, Tapp RJ, Oldenburg BF, Shaw JE. Psychosocial Stress Predicts Abnormal Glucose Metabolism: The Australian Diabetes, Obesity and Lifestyle (AusDiab) Study. *Annals of Behavioral Medicine*. 2013;46(1):62-72. doi:10.1007/s12160-013-9473-y

18. Shomaker LB, Tanofsky-Kraff M, Young-Hyman D, et al. Psychological symptoms and insulin sensitivity in adolescents. *Pediatric diabetes*. 2010;11(6):417-423.

19. Shomaker LB, Tanofsky-Kraff M, Stern EA, et al. Longitudinal study of depressive symptoms and progression of insulin resistance in youth at risk for adult obesity. *Diabetes care*. 2011;34(11):2458-2463.

20. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *Journal of health and social behavior*. Published online 1983:385-396.

21. Finkelstein DM, Kubzansky LD, Capitman J, Goodman E. Socioeconomic Differences in Adolescent Stress: The Role of Psychological Resources. *Journal of Adolescent Health*. 2007;40(2):127-134. doi:10.1016/j.jadohealth.2006.10.006

22. Reaven GM. Role of insulin resistance in human disease. *Diabetes*. 1988;37(12):1595-1607.

23. Brun J-F, Ghanassia E, Fédou C, Bordenave S, De Mauverger ER, Mercier J. Assessment of insulin sensitivity (S I) and glucose effectiveness (S G) from a standardized hyperglucidic breakfast test in type 2 diabetics exhibiting various levels of insulin resistance. *Acta diabetologica*. 2013;50(2):143-153.

24. Ten S, Maclaren N. Insulin resistance syndrome in children. *The Journal of Clinical Endocrinology & Metabolism*. 2004;89(6):2526-2539.

25. Bergman RN, Prager R, Volund A, Olefsky J. Equivalence of the insulin sensitivity index in man derived by the minimal model method and the euglycemic glucose clamp. *The Journal of clinical investigation*. 1987;79(3):790-800.

26. Rickels MR. Metabolic and endocrine evaluation of islet transplant function. In: *Transplantation, Bioengineering, and Regeneration of the Endocrine Pancreas*. Elsevier; 2020:565-578.

27. HAYASHI M, FLOYD JR JC, PEK S, FAJANS SS. Insulin, proinsulin, glucagon and gastrin in pancreatic tumors and in plasma of patients with organic hyperinsulinism. *The Journal of Clinical Endocrinology & Metabolism*. 1977;44(4):681-694.

28. Nittala A, Ghosh S, Stefanovski D, Bergman R, Wang X. Dimensional analysis of MINMOD leads to definition of the disposition index of glucose regulation and improved simulation algorithm. *Biomedical engineering online*. 2006;5(1):1-15.

29. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of youth and adolescence*. 1988;17(2):117-133.

30. Royston P, White IR. Multiple imputation by chained equations (MICE): implementation in Stata. *J Stat Softw*. 2011;45(4):1-20.

31. Kahn SE, Prigeon RL, McCulloch DK, et al. Quantification of the relationship between insulin sensitivity and β-cell function in human subjects: evidence for a hyperbolic function. *Diabetes*. 1993;42(11):1663-1672.

32. O'Donovan A, Tomiyama AJ, Lin J, et al. Stress appraisals and cellular aging: A key role for anticipatory threat in the relationship between psychological stress and telomere length. *Brain, behavior, and immunity*. 2012;26(4):573-579.

33. Louie P, Wheaton B. The Black-White paradox revisited: understanding the role of counterbalancing mechanisms during adolescence. *Journal of health and social behavior*. 2019;60(2):169-187.

34. Pilcher JJ, Bryant SA. Implications of social support as a self-control resource. *Frontiers in behavioral neuroscience*. 2016;10:228.

35. Hefner J, Eisenberg D. Social support and mental health among college students. *American Journal of Orthopsychiatry*. 2009;79(4):491-499.

36. Hilding A, Shen C, Östenson C-G. Social network and development of prediabetes and type 2 diabetes in middle-aged Swedish women and men. *Diabetes Research and Clinical Practice*. 2015;107(1):166-177. doi:10.1016/j.diabres.2014.09.057

37. Brinkhues S, Dukers-Muijrers NH, Hoebe CJ, et al. Socially isolated individuals are more prone to have newly diagnosed and prevalent type 2 diabetes mellitus-the Maastricht study–. *BMC Public Health*. 2017;17(1):1-12.

38. Gangel MJ, Dollar J, Brown A, et al. Childhood social preference and adolescent insulin resistance: Accounting for the indirect effects of obesity. *Psychoneuroendocrinology*. 2020;113:104557.

39. Fitzgerald A, Fitzgerald N, Aherne C. Do peers matter? A review of peer and/or friends' influence on physical activity among American adolescents. *Journal of adolescence*. 2012;35(4):941-958.

40. Hedegard D. Why do blacks have smaller social networks than whites? The mechanism of racial identity strength. *Ethnic and Racial Studies*. 2018;41(14):2464-2484. doi:10.1080/01419870.2017.1367019

41. Flores M, Ruiz JM, Goans C, et al. Racial-ethnic differences in social networks and perceived support: Measurement considerations and implications for disparities research. *Cultural Diversity and Ethnic Minority Psychology*. 2020;26(2):189-199. doi:10.1037/cdp0000283

42. Shonkoff JP, Richter L, van der Gaag J, Bhutta ZA. An integrated scientific framework for child survival and early childhood development. *Pediatrics*. 2012;129(2):e460-e472.

43. Bazzocchi A, Diano D, Ponti F, et al. A 360-degree overview of body composition in healthy people: Relationships among anthropometry, ultrasonography, and dual-energy x-ray absorptiometry. *Nutrition*. 2014;30(6):696-701. doi:10.1016/j.nut.2013.11.013

Chapter 5

Weight Stigma and Physical Activity Avoidance Among College-Aged Students

Preamble

The **original study aims** at the time of the dissertation proposal for study 4 was as follows: "To examine the cross-sectional associations of a weight discriminatory exposure on health-promoting behavioral intentions (exercise and eating intentions) and the moderating effects of stress on these associations in older adolescents ages 18-21 years of age." The original aims were revised due in part to the COVID-19 pandemic that led to data collection stoppages in March of 2020. As a result, only 49 participants were able to complete both in-person visits for the study, which is far less than the original proposal of 200 participants. Furthermore, given the low sample size and its possible implications on statistical power, moderation analyses using the Perceived Stress Scale were conducted, but were not reported in the study manuscript. Nevertheless, noteworthy results and implications were obtained with the data from 49 participants as described in chapter 5 of this dissertation.

Weight stigma and physical activity avoidance among college-aged students is the fourth study in the dissertation. It is a study that explored the cross-sectional associations between prior weight stigma experiences, physical activity intentions (positive intent and avoidance), and physical activity behavior [moderate-to-vigorous physical activity (MVPA), total physical activity (TPA)]. The study also examined the acute effects of a weight stigma exposure on future physical activity intentions and behaviors among young undergraduate college students.

The abstract for this study has been submitted and accepted for an e-poster at the American College of Sports Medicine 2021 Annual Meeting. This manuscript will be submitted for publication in a peer-review journal.

Abstract

Weight stigma is a common form of discrimination that has been associated with unhealthy eating, however, less is known about the impact of weight stigma on physical activity (PA) intentions and objectively measured PA behaviors. **PURPOSE:** The purpose of this study was two-fold: (1) to explore cross-sectional associations between prior weight stigma experiences, PA intentions and behaviors; and (2) to examine the acute effects of a weight stigma exposure on future PA intentions and behaviors among young undergraduate college students. **METHODS:** Prior experiences of weight stigma were evaluated using the Brief Stigmatizing Situations Inventory. Positive PA intentions were measured via the Exercise Intentions Scale, and PA avoidance was measured via the Exercise Avoidance-Motivation Scale. PA behaviors were measured using accelerometry over a minimum of 4 days. To assess the acute effects of weight stigma exposure, participants were randomized into one of two experimental conditions: (1) a weight stigmatizing video condition or (2) a neutral video control condition. Following each condition, participants answered questions regarding their PA intentions for the upcoming week and were given an accelerometer to assess MVPA and TPA for one week. **RESULTS:** Forty-nine students between the ages of 18-21 years (84% female; 55% non-Hispanic white; 19.6±1.1 years of age; BMI: 23.9±4.0 kg/m2; 35% with overweight/obesity) completed both the questionnaires and experimental protocol. Prior weight stigma experiences were associated with PA avoidance (β= 12.1±2.7; $p<0.001$), with greater weight stigma experiences predicting higher avoidance of PA. Prior weight stigma experiences were not associated with positive PA intentions, MVPA, or TPA (*p*s>0.05). Across experimental conditions, there were no differences in positive PA intentions (weight-stigma: 13.2±1.0 vs control: 12.9±0.89), PA avoidance (weight-stigma: 16.2±1.6 vs. control: 15.2±1.9), MVPA (weight-stigma: 36.6±5.1 min/day vs. control: 31.4±4.7 min/day), or TPA (weight-stigma: 98.6±8.2 min/day vs. control: 99.5±7.1 min/day) (all *p'*s>0.05). **CONCLUSIONS:** Greater weight stigma experiences were associated with higher avoidance of PA among young college-age students. Future studies should examine the long-term effects of weight stigma on PA intentions and objectively measured PA behaviors in young college students.

Introduction

Individuals with overweight and obesity often face discrimination and undue stigma from others because of their weight[1]. Those with overweight and obesity are frequent targets of day-to-day discrimination across several different settings including but not limited to: educational, employment, health care, media, and within legal settings[1]. The media in particular, has been shown to play a crucial role in spreading weight bias—defined as negative-weight related attitudes, beliefs, and assumptions about individuals with overweight and obesity[2]. Children movies for example, often overtly characterize children with overweight or obesity as being lazy, aggressive, unpopular, and unintelligent compared with their normal weight counterparts, in turn, sending stigmatizing messages to those with overweight and obesity[3,4]. The pervasiveness of weight bias and the unequal treatment (i.e. discrimination) of individuals with higher weight in the U.S. is rising. Current evidence suggests that incidences of weight discrimination rose substantially from the late 1990s to the mid-2010s and is currently believed to be similar to rates of racial discrimination in the United States[5], highlighting the extensiveness of weight stigma in the U.S.

Weight stigma—defined as the social rejection or devaluation of those with higher weight status[6], is not only a major issue among adults, but also among children and adolescents as well. Public school teachers for example, have previously noted that weight related bullying from peers was one of the most salient forms of bullying occurring within schools[7]. Moreover, adolescents with overweight and obesity face weight bias from their teachers as well, often manifesting in the form of lowered expectations and attitudes regarding their academic, physical, and social capabilities compared with their normal weight counterparts[8]. There is evidence that weight stigma is also common within the home environment. In a retrospective study, 53% of women with obesity stated that they regularly encountered weight stigma from their mothers when growing up[9]. Overall, the negative consequences of weight stigma from close family members and society are long lasting and may track into adulthood[4].

Stigmatization has been heralded as a possible means of reducing obesity rates. It was assumed that individuals with overweight and obesity would change their eating behavior and lose weight when stigmatized[10]. While successful in reducing smoking prevalence (i.e. anti-smoking campaigns), researchers have found this approach to be ineffective as it relates to weight management. Rather than assisting individuals in losing weight, weight stigma has been shown to work in a manner contrary to expectations—increasing psychological stress and leading to increased weight gain among adults[11–13]. Among adolescents, researchers have observed that increased weight stigma in the form of internalized stigma—the belief that one deserves the stigma and discriminatory treatment they experience because of their weight[2]—was also associated with increased levels of psychosocial distress[14].

One pathway by which weight stigma works to increase stress is through what is described as social identity threat—wherein individuals with obesity are viewed negatively by society, and as a result they themselves may begin to embody those same societal views. Originating from social identity theory, social identity threat occurs during a social situation or event where an individual feels threatened, judged, or discriminated against because of an identity or stereotype that they may or may not uphold[15]. Those who are stigmatized and bullied can internalize the negative judgements of others, resulting in increased stress. The influence of social identity threat is clearly seen in studies such as the one conducted by Major and colleagues[13]. This identity threat not only affects an individual's biological stress response[16], but also likely alters one's behavioral responses as well.

It has been proposed by Major and colleagues as well as several others, that weight stigma is a stressor that not only leads to increased psychological stress but also decreased self-regulation in individuals with overweight and obesity, which in turn leads to increased eating and ineffective attempts to lose weight[13]. Weight stigma not only affects eating behavior, but there is evidence that it affects physical activity behavior as well. Exposure to weight stigma has been shown to be associated with lowered physical activity engagement and motivation to exercise, and reduced sports enjoyment in children and adolescents[17,18]. Previously, in a study of weight victimization among adolescents, Puhl and Luedicke found that those reporting negative emotions resulting from weight-based victimization were more likely to use maladaptive and avoidant coping strategies such as avoiding physical activity and physical education class[18]. Among fifth through eighth grade youth, it was observed that weight stigma in the form of weight criticism *during* physical activity, was related with reduced sports enjoyment and reduced self-reported mild-intensity physical activity participation[17]. In a sample of college-aged females with normal weight, overweight, and obesity, it was observed that *previous* weight stigma experiences were related with increased avoidance of physical activity regardless of BMI[19]. Similar findings regarding previous experiences of weight stigma and teasing with that of reduced interest in participating in physical activity in adolescents have been seen with other investigators[20–22]. Together, these studies provide preliminary evidence of the negative effect of previous and in-task weight-stigma on physical activity participation and motivation. However, given the lack of studies assessing how weight stigma will influence *future* physical activity intentions and behaviors among adolescents, research addressing this concern among adolescents is needed.

To address this research gap, the purpose of this study was twofold: (1) to confirm cross-sectional associations between prior weight stigma experiences, physical activity intentions (positive intent and avoidance), and objectively measure physical activity behavior [moderate-to-vigorous physical activity (MVPA), total physical activity (TPA)] among young undergraduate college students; and (2) to examine the effects of an acute weight stigma exposure on future physical activity intentions and behaviors in this age group. It was hypothesized that higher levels of prior weight stigma experiences would be associated with lower physical activity intentions and behaviors, and that an acute exposure to weight stigma would be associated with short-term declines in physical activity intentions and behaviors.

Methods

Study participants

Undergraduate students between the ages of 18-21 were recruited from the University of Michigan Ann Arbor campus to participate in the Attitudes Regarding Eating and Activity (AREA) study. Participants were recruited via: (i) flyers posted on the University campus, local libraries, and churches, (ii) word of mouth, (iii) through the University research paid subjects pool, (iv) and social media platforms. Participants with normal-, overweight-, and obese-weight statuses were recruited and screened for clinical diagnosis of depression, anxiety, or other mental health disorders that may influence mood, emotions, or perceptions of stress as well as prior treatment or diagnosis of an eating disorder. As a result of the high prevalence of these mental disorders within college-aged populations[23], participants were not excluded due to diagnosed anxiety or depression, but merely screened. Participants were however, excluded from the AREA study on the following basis: i) if they were not between the ages of 18-21 years, ii) if they had a prior diagnosis of a psychiatric disorder (not including anxiety and depression), iii) if they were not a current student at the university, iv) or if they had a mental disability that could hinder them from answering survey questionnaires. Data collection began January 2020 and ran until March 2020. Data collection was stopped in March 2020 because of the COVID-19 pandemic and the halt of in person data collection across the University. Data collection was conducted online using the University Qualtrics Survey software and in-person at the Childhood Disparities Research Laboratory (CDRL). Participants were compensated 20 dollars for the completion of all surveys, and an additional 20 dollars upon completing a weeklong physical activity measurement. Prior to all data collection, written informed consent was obtained from all participants. All experimental procedures, inclusion and exclusion criteria, risks and benefits, confidentiality, compensation, and all pertinent research protocol were approved by the University Institutional Review Board (HUM 00170449) before the start of the study.

Data collection procedures

The description of the study timeline for the AREA study can be seen in Figure 1. At least one week prior to their first in-person visit, participants completed the baseline survey that consisted of demographic and psychological questions including questions regarding prior weight stigma experiences and perceptions regarding their weight. Participants then came to the CDRL for their first in-person visit, where they completed the experimental condition and were given an accelerometer to take home following the visit. A week following the experimental condition, participants returned to the CDRL to return the accelerometer and to complete a follow-up survey pertaining to their physical activity participation over the past week. A debriefing statement and discussion with the participants detailing the true purpose of the study was done at the end of the second in-person visit.

Randomization

Upon completion of the baseline survey, participants were randomized into either a weight stigmatizing or non-stigmatizing control condition. A simple randomization method was conducted, using a random number generator for each participant's group/condition assignment.

Experimental conditions

Participants came to the CDRL to complete either the weight-stigmatizing condition or the control condition. The weight-stigmatizing condition consisted of watching a 10-minute video compilation that was created to induce feelings of weight related devaluation[1]. The stigmatizing video was a compilation of several short clips from popular television shows and comedy movies that depicted individuals with overweight and obesity in stereotypical and derogatory ways. The control condition consisted of watching a neutral, low affect 10-minute video compilation that included clips of advertisements for furniture, car insurance, and clips from shows such as Blue Planet and Planet Earth. Both videos were created by and have been previously used by Schvey and colleagues[1]. Additionally, both videos have been previously used to examine the physiological impact of weight stigma exposure in adult women with overweight[1]. To confirm that a negative affective response occurred as a result of the weight-stigmatizing condition, the short form of the Positive and Negative Affect Schedule (PANAS) scale was used as a measure of positive and negative affect[24]. The PANAS scale is a commonly used self-reported 20-item scale to assess both positive and negative affect. The PANAS has been previously used in a sample of adolescents and young adults ages 11-21 years[25] and showed good internal consistency among the present sample (Cronbach $\alpha=0.825$).

Dependent variable

Positive physical activity intentions

Participants' positive physical activity intentions were assessed using the Exercise Intention Scale[26]. The Exercise intention scale is a three item scale that measures intentions to exercise over the course of the upcoming week, with Likert-type responses on a scale anchored from 1 to 7[26]. An example question from the Exercise Intention Scale was: *"In the next two weeks, my goal is to exercise*," with responses anchored by *not at all* (1) to *every day* (7). The exercise intention scale showed excellent internal consistency among the present sample (Cronbach α=0.89). The exercise intention scale has been previously used among adult samples[26].

Physical activity avoidance

Intentions to avoid physical activity was measured using the Exercise avoidance-motivation scale[19]. The exercise avoidance-motivation scale is an eight item, seven-point Likert-like scale that assesses an individual's reaction to experiences dealing with 'negative situations related to their weight' particularly in the domain of exercise. Each item on the scale is anchored by *not at all true* (1) to *completely true* (7). The exercise avoidance-motivation scale demonstrated excellent internal consistency among the sample population (Cronbach α=0.87) and has been previously used in a young undergraduate sample[19].

Objectively measured physical activity

MVPA and TPA participation was measured using the GT3X ActiLife accelerometers (Actigraph LLC., Pensacola, FL). All participants were asked to wear the 3-axial accelerometers for 7 days snugly on their right hip using an adjustable belt. At home, participants were also asked to complete a non-wear/sleep diary in tandem with the accelerometer. Participants were advised to remove the accelerometers during bathing, water-based activities, contact sports, and while they slept. All accelerometers were initialized to collect raw data at a frequency of 30 Hz. Raw accelerometer data were downloaded and integrated into 60s epochs via ActiLife software version 6.11.8 using Puyau cut points[27]. The Puyau cut-points were used to derive moderate-to-vigorous activity[27]. A wear time of > 600 minutes/day was used as the criteria for a valid day, and ≥ 4 valid wear-days was used as the criteria for an accurate baseline measure, in line with established accelerometry recommendations[28]. Consecutive periods of zero counts for > 60 minutes were also defined as non-wear time[29]. Non-wear times were excluded from the analyses with only valid wear-time data used to determine intensities of daily activity intensities in minutes.

Independent variable

Prior weight stigma experiences

The brief-form of the stigmatizing situations inventory (SSI-B), is a commonly used inventory used to measure and assess an individual's past experiences with weight stigma[30]. This brief form consists of a 10-item, Likert-type scale anchored by 0 (never) to 9 (daily) that assesses whether, and how often, individuals have experienced weight-stigmatizing situations[31]. An example item from the SSI-B includes: *"Having a doctor recommend a diet, even if you did not come in to discuss weight loss.*" The SSI-B has been previously validated as an efficient tool for assessing experiences with weight stigma[31]. The SSI-B showed good internal consistency within the sample (Cronbach α=0.79).

Covariates

Anthropometric measures of height and weight were measured in the CDRL by trained study staff members following the completion of the last study survey during each participant's second in-person visit. Height (cm) was measured to the nearest 0.1 cm using ShorrBoard ® (Weigh and Measure, LLC. Olney, MD) with standardized procedures. Body weight (kg) was measured to the nearest 0.1 kg, using an electronic scale (Doran Scales, Inc, Batavia, IL). Race and ethnicity of each participant was self-reported along with each participant's biological sex.

Statistical analysis

All data analysis was conducted using Stata 16.0 (StataCorp LP, College Station, TX). T-tests were used to compare the mean values of negative affect between those who were randomized to the weight-stigmatizing condition and those in the control condition. Multivariable linear regression models were used to examine the association between the independent variable (prior experiences of weight stigma) and dependent variables (physical activity intentions and objectively physical activity behavior), adjusting for all covariates [age, sex, race, and body mass index (BMI)] in each model. To see whether involvement in the stigma condition predicted physical activity intentions and behaviors, similar multivariable linear regression analyses were conducted with a dichotomous independent predictor variable (condition). T-tests were used to assess the acute effects of the weight-stigmatizing exposure on physical activity intentions and behaviors across conditions. A p-value of $p<0.05$ was set *a priori* to determine significance for all models.

Results

Participant characteristics

Participant characteristics are displayed in mean and standard deviation in Table 1. A total of 49 participants completed the study. In total, study participants were: 81.6% female (18.4% males), 59.2% White (40.8% non-White), with a BMI of 23.9±4.0 kg/m^2 (35% classified as having overweight/obesity), and a mean age of 19.6±1.1 years. Of the 49 participants, there were 21 participants who were not asked about any prior diagnosed anxiety or depression. Out of the 28 participants who were asked about their history of anxiety and depression, 5 participants disclosed that they had been diagnosed with anxiety, depression, or both. There were no differences in participant characteristics across both experimental conditions (age, race, BMI, height, weight, prior weight stigma experiences; all ps>0.05). The average rating (score) for the Stigmatizing Situations Inventory was 0.39 (SD=0.49), indicating that the participants in this study on average rarely experienced weight stigma. Mean scores ranged from 0.1 (at least once before) to 0.21 (over several times in their lives), and all participants indicated having experienced at least one incidence of weight stigma in their lifetime. The mean score for prior weight stigma experiences is in line with a previous study conducted among a sample that included college-aged participants across the weight spectrum[19]. On average, participants engaged in 34.0±22.5 minutes of MVPA per day, with a range from 2.7 minutes per day to 101.9 minutes per day, suggesting that on average, participants met and even exceeded the national recommendations of at least 30 minutes of MVPA per day. Mean TPA per day was 99.0±34.6 minutes, with a range of 23.3 to 198.6 minutes of activity per day. Mean positive exercise intention score was 12.98 (SD=4.4) out of a possible score of 21, whereas average exercise avoidance score was 15.7 (SD=8.5) out of a possible score of 56.

Confirmation of a negative affective response

Negative affect was significantly different across experimental conditions, with higher negative affect among those in the weight-stigmatizing condition compared with those in the non-stigmatizing control condition (weight-stigma: 18.7±0.9 vs control: 11.2±0.4; p<0.001). No significant difference in positive affect was seen (weight-stigma: 17.5±1.0 vs control: 20.5±1.6; p= 0.12), however, positive affect was lower among those in the weight-stigmatizing condition. Together these results confirmed the increase in negative emotional distress caused by the weight-stigmatizing condition.

Prior weight stigma experiences, physical activity intentions, and behaviors

Linear regression analysis examining the association between prior weight stigma experiences, physical activity intentions, and physical activity behaviors is reported in Table 2. Prior weight stigma experiences were associated with physical activity avoidance (B= 12.1±2.7; $p<0.001$), with greater weight stigma experiences predicting higher avoidance. Prior weight stigma experiences were not associated with positive physical activity intentions, nor with MVPA, or TPA (*p*s>0.05).

Acute effects of a weight stigma exposure on physical activity intentions and behaviors

The analysis for the acute effects of the weight-stigmatizing exposure on physical activity intentions, and physical activity behaviors across experimental conditions can be seen in Figure 2. There were no differences in positive physical activity intentions (weight-stigma: 13.2±1.0 vs control: 12.9±0.89), avoidance (weight-stigma: 16.2±1.6 vs. control: 15.2±1.9), MVPA (weight-stigma: 36.6±5.1 min/day vs. control: 31.4±4.7 min/day), or TPA (weight-stigma: 98.6±8.2 min/day vs. control: 99.5±7.1 min/day) across experimental conditions (*p*s>0.05). When linear regression was used to examine whether the experimental condition predicted physical activity intentions and behaviors, it was observed that experimental condition did not predict positive physical activity intentions, physical activity avoidance, MVPA, or TPA (all *p*s>0.05; Table not shown).

Discussion

In the present study, greater prior experiences of weight stigma was associated with higher avoidance of physical activity, but not positive physical activity intentions nor objectively measured physical activity behaviors (accelerometry). Exposure to an acute weight stigma condition was also not associated with changes in physical activity intentions or behaviors.

Together, the present study provides confirmation of the detrimental effects of prior weight stigma on decreasing future physical activity motivations. Given that the sample population reported relatively low prior experiences of weight stigmatization, the present findings are significant, in that low to mild levels of weight stigmatization can lead to the avoidance of physical activity. This may, in turn, expedite decreases in future participation in physical activity among older adolescents and young college-aged students.

Previously, Vartanian and Shaprow explored the impact of weight stigma on exercise motivation in a sample of college-aged females with normal weight, overweight, and obesity. The authors noted that weight stigma experiences were related with increased avoidance of physical activity regardless of BMI[19]. Among younger adolescents in grades 9-12th, Puhl and Luedicke observed that those reporting negative affect in response to experiencing weight stigma were more likely to avoid engaging in physical activity and avoid going to physical education class[18]. In the adult literature, Vartanian and Novak also found that increased experiences of weight discrimination was associated with avoidance of physical activity[32]. Seacat and Mickelson also tested to see whether exposure to negative weight-related stereotypes (stereotype priming) affected personal exercise and dietary health behaviors in women with overweight. They observed that those primed to think about their weight reported lower levels of exercise and dietary intentions in comparison to those who were not primed[33]. Together, the present study in addition to those by prior investigators demonstrates how the stressor of weight stigma, increases avoidance of physical activity and decreases intentions to engage in physical activity. This in turn may have long-term implications for weight gain and healthy living, particularly as the negative consequences of weight stigma are long lasting and may track into adulthood[4].

Although higher levels of prior weight stigma was associated with higher avoidance of physical activity, an acute laboratory exposure to weight stigma was not related with changes in physical activity intentions including avoidance of physical activity. Although this finding did not support our hypothesis, other research points to mixed findings in the association between weight stigma and physical activity. For example, Pearl et al., suggested that exposure to weight-stigmatizing media may actually lead to greater reports of exercise intentions, motivation, and behavior, in part because of a pathological drive for thinness encouraged by the media content[34]. In their study exploring past experiences of weight stigma and a present stigmatizing media exposure on physical activity intentions, motivation, and behavior among women ages 18-59 years, they observed that those randomized to the stigma condition who had higher prior weight stigma experiences, reported greater exercise intentions and behavior, and also had a higher drive for thinness. Although there were no significant differences in prior experiences of weight stigma across conditions in our present sample, it is plausible that the null findings may be due in part to not exploring interaction factors such as disordered eating and a drive for thinness that may amplify the effects of weight stigma above and beyond an acute exposure. Because of the small sample size however, such complex analysis would have had insufficient power. Post-hoc power analyses with the current sample of 49 participants, an alpha level of 0.05 (two-sided) showed that we had more than 78% observed power to detect a large effect (d=0.8), and more than 40% observed power to detect a medium effect size (d=0.5) between experimental groups. Thus the current findings should be considered exploratory. Future studies employing such experimental designs should consider the role of moderators such as prior weight stigma experiences and body shape concerns when examining the effects of an acute weight stigma exposure on behavioral intentions. Furthermore, it is also possible that there were not enough participants within our sample with negative self-perceptions of their body size, as only 3 of the 49 participants viewed themselves as being either underweight, overweight, or very overweight. Similarly, only 4 of the 49 participants thought others perceived their body size as either underweight or overweight. The vast majority of the sample perceived their weight and others perception of their weight as normal. This generally positive self-perception of weight by the participants may have played a role in our null findings, given that most viewed their weight to be normal and thus the likelihood of experiencing social identity threat from watching the weight-stigmatizing video is lessened.

Contrary to our hypotheses, neither prior experiences of weight stigma, nor an acute weight stigma exposure was associated with or led to changes in future objectively measured physical activity engagement. Our findings however, are similar to Vartanian & Novak who observed that weight stigma (measured via the stigmatizing situations inventory) was not significantly related with mild, moderate, or strenuous activity measured via a self-reported questionnaire[32]. Likewise, Vartanian & Shaprow also did not find an association between weight stigma and past self-reported mild, moderate, and strenuous activity in a sample of undergraduate females[19]. Although other investigators have previously found an association between an acutely induced weight stigma experience and physical activity engagement[33], it is plausible that the effects of weight stigma are more chronic rather than acute and that it is the cumulative experiences of weight stigma over time that may impact physical activity intentions and behaviors. Additionally, the use of objectively measured physical activity behavior, demographic differences in the current sample (e.g. racial diversity, varied BMI status), as well as timing of measures may have also contributed to the lack of a relationship between weight stigma and physical activity behavior. Given these plausible explanations and the limitation of self-reported measures of physical activity—particularly the overestimation of energy expenditure[35], additional longitudinal research incorporating device-based measurements are needed.

There are many strengths of this study that must be noted. Strengths of the study include the racial and ethnic diversity of the study participants, an experimental design utilizing a validated weight stigmatizing video created by experts in the field of weight stigma, and the inclusion of individuals across the weight spectrum were notable strengths of this study. Furthermore, the use of objectively measured physical activity in the current study adds to the literature by filling a gap that has often been missing in existing studies. Nevertheless, a few limitations should also be mentioned. Of note, the study recruited participants who were student members of a Midwestern university, limiting the generalizability of the study findings to other groups outside of the Midwest and university environments. This, however, was done as a means for allowing for a tighter sample demographic, in turn, improving internal validity. In that limiting the sample to students from a single Midwestern university limits the influence of other demographic factors and variables that may be seen outside of this location and group. Additionally, the cross-sectional nature of the findings from the regression analysis limits interpretation of causality. Given the small sample size of the study as well, it is possible that we may not have had adequate power to detect changes in physical activity intentions and behavior across conditions. Lastly, because of the online and in-person nature of the study design, the confounding influence of the research assistant(s)'s social identities (race, gender, weight status) may have influenced accuracy of respondents' answers. Despite these limitations, we were able to observe a significant association between greater experiences of prior weight-stigma and higher avoidance of exercise and physical activity among older adolescents/young college-aged students.

Conclusion

Considering the paucity of research on weight stigma and future physical activity intentions and objectively measured behavior among adolescents and young college-aged students, this study adds to the extant literature by demonstrating the harmful association between weight-stigma and greater avoidance of physical activity. Given the importance of physical activity on mental and physical wellbeing, and the uniqueness of this transitional period into early adulthood, where there is greater autonomy in decision making[36], the present findings shed insight into the destructive nature of weight stigma on positive health enhancing intentions. Especially as the participants rarely experienced weight stigma yet there was still an association with physical activity avoidance. Given that the negative consequences of weight stigma are long lasting and track into adulthood[4], it is critical for families, public health professionals, researchers, and all other stakeholders to find strategies to mitigate the negative consequences of weight stigma on health enhancing behaviors. Strategies that include but are not limited to increasing positive physical activity antecedents such as physical activity enjoyment through interventions focused on the intrinsic value of physical activity, as well as using positive portrayals of individuals with overweight/obesity in the media. Attributing more positive traits to an individual with obesity has been seen to improve and increase participation in daily exercise, as well as lower caloric intake[37]. This can be an important interventional facet in addressing obesity and its associated comorbid conditions during adolescence and emerging adulthood. Thus, encouraging and depicting enjoyment of physical activity among individuals of all weight statuses, may be an important interventional tactic to reduce the negative impact of weight stigma on health enhancing behavior.

Figures and Tables

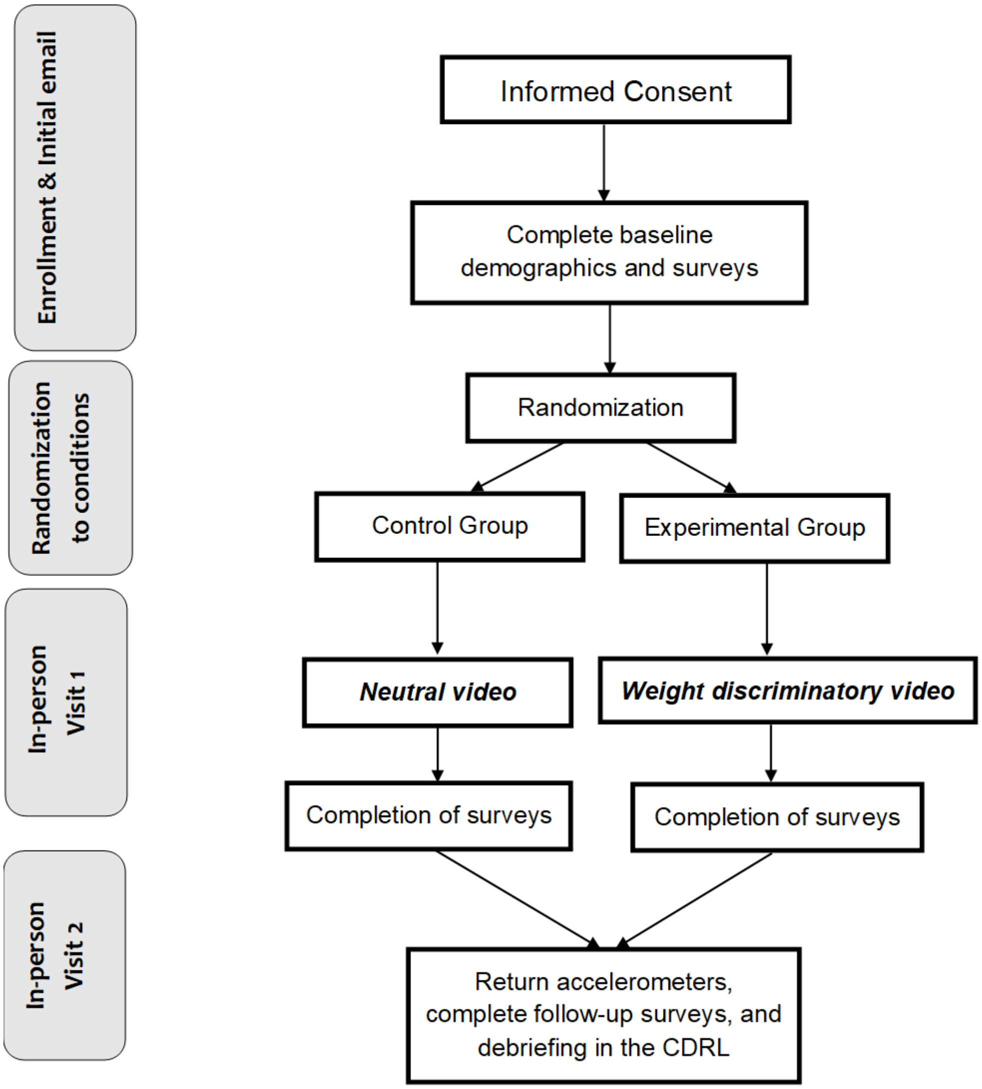

Figure 5.1. AREA study timeline

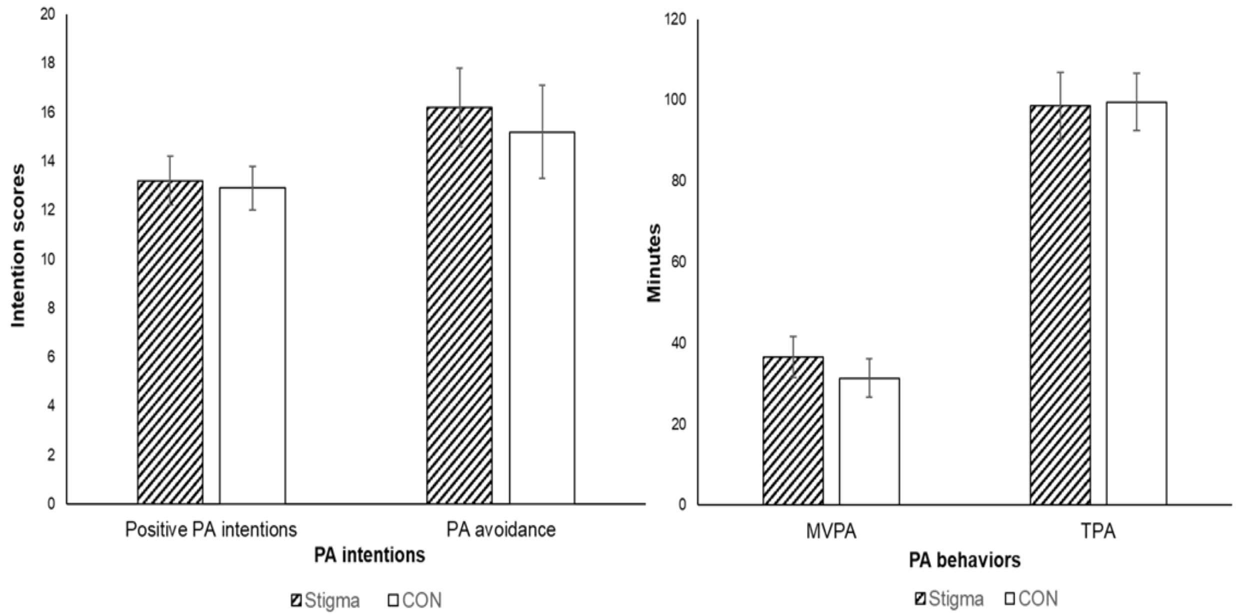

Figure 5.2. Physical activity intentions and behaviors across experimental conditions. CON= control; PA= physical activity; MVPA= moderate-to-vigorous physical activity; TPA= total physical activity.

Table 5-1. Participant characteristics. Statistics are in reported in either mean and standard deviation (SD) or frequency (%).

Variables	**N**	**Mean (SD) or Frequency (%)**
Age (years)	49	19.6 (1.1)
Gender (% female)	49	81.6
Race (% non-White)	49	40.8
Non-Hispanic White	29	59.2
Non-Hispanic Black	5	10.2
Asian or Pacific Islander	13	26.5
Other	2	4.1
BMI (kg/m^2)	49	23.86 (3.96)
MVPA (min/day)	42	34.0 (22.5)
TPA (min/day)	42	99.03 (34.6)
Exercise intention score	49	12.98 (4.4)
Negative Affect	49	15.0 (5.2)
Prior experiences of weight stigma (SSI-B average score)	49	0.39 (0.49)

BMI; body mass index. MVPA=moderate-to-vigorous physical activity; TPA=total physical activity. SSI-B= Stigmatizing Situations Inventory-Brief.

Table 5-2. Linear regression output for association between weight-stigma and physical activity intentions and behaviors.

	Coefficient (*B*)	Standard Error	p-value
Exercise avoidance			
SSI	12.1	2.7	<0.001*
Positive intentions			
SSI	-0.31	1.82	0.86
MVPA			
SSI	-2.94	9.23	0.75
TPA			
SSI	-15.34	14.69	0.30

B=Unstandardized beta-coefficient. SSI= Stigmatizing Situations Inventory. MVPA= moderate to vigorous physical activity. TPA= total physical activity. All models controlled for covariates race, gender, BMI, and age.

References

1. Schvey NA, Puhl RM, Brownell KD. The Stress of Stigma: Exploring the Effect of Weight Stigma on Cortisol Reactivity. *Psychosomatic Medicine*. 2014;76(2):156-162. doi:10.1097/PSY.0000000000000031

2. Alberga AS, Russell-Mayhew S, von Ranson KM, McLaren L. Weight bias: a call to action. *Journal of Eating Disorders*. 2016;4(1):34. doi:10.1186/s40337-016-0112-4

3. Pont SJ, Puhl R, Cook SR, Slusser W. Stigma experienced by children and adolescents with obesity. *Pediatrics*. 2017;140(6).

4. Christensen S. Weight Bias and Stigma in Children. *Journal of Pediatric Surgical Nursing*. 2018;7(3):72-74. doi:10.1097/JPS.0000000000000178

5. Puhl RM, Andreyeva T, Brownell KD. Perceptions of weight discrimination: prevalence and comparison to race and gender discrimination in America. *International Journal of Obesity*. 2008;32(6):992-1000. doi:10.1038/ijo.2008.22

6. Tomiyama AJ, Carr D, Granberg EM, et al. How and why weight stigma drives the obesity 'epidemic' and harms health. *BMC Medicine*. 2018;16(1):123. doi:10.1186/s12916-018-1116-5

7. Bradshaw CP, Waasdorp TE, O'Brennan LM, Gulemetova M. Teachers' and Education Support Professionals' Perspectives on Bullying and Prevention: Findings From a National Education Association Study. Suldo S, ed. *School Psychology Review*. 2013;42(3):280-297. doi:10.1080/02796015.2013.12087474

8. Peterson JL, Puhl RM, Luedicke J. An Experimental Assessment of Physical Educators' Expectations and Attitudes: The Importance of Student Weight and Gender. *Journal of School Health*. 2012;82(9):432-440. doi:https://doi.org/10.1111/j.1746-1561.2012.00719.x

9. Puhl RM, Brownell KD. Confronting and coping with weight stigma: an investigation of overweight and obese adults. *Obesity*. 2006;14(10):1802-1815.

10. Callahan D. Obesity: Chasing an Elusive Epidemic. *Hastings Center Report*. 2013;43(1):34-40. doi:https://doi.org/10.1002/hast.114

11. Tomiyama AJ. Weight stigma is stressful. A review of evidence for the Cyclic Obesity/Weight-Based Stigma model. *Appetite*. 2014;82:8-15. doi:10.1016/j.appet.2014.06.108

12. Sutin AR, Terracciano A. Perceived Weight Discrimination and Obesity. *PLOS ONE*. 2013;8(7):e70048. doi:10.1371/journal.pone.0070048

13. Major B, Eliezer D, Rieck H. The psychological weight of weight stigma. *Social Psychological and Personality Science*. 2012;3(6):651-658.

14. Hand WB, Robinson JC, Stewart MW, Zhang L, Hand SC. The Identity Threat of Weight Stigma in Adolescents. *West J Nurs Res*. 2017;39(8):991-1007. doi:10.1177/0193945917704201

15. Steele CM, Spencer SJ, Aronson J. Contending with group image: The psychology of stereotype and social identity threat. In: *Advances in Experimental Social Psychology*. Vol 34. Academic Press; 2002:379-440. doi:10.1016/S0065-2601(02)80009-0

16. Jackson SE, Steptoe A. Obesity, perceived weight discrimination, and hair cortisol: a population-based study. *Psychoneuroendocrinology*. 2018;98:67-73. doi:10.1016/j.psyneuen.2018.08.018

17. Faith MS, Leone MA, Ayers TS, Heo M, Pietrobelli A. Weight criticism during physical activity, coping skills, and reported physical activity in children. *Pediatrics*. 2002;110(2):e23-e23.

18. Puhl RM, Luedicke J. Weight-Based Victimization Among Adolescents in the School Setting: Emotional Reactions and Coping Behaviors. *J Youth Adolescence*. 2012;41(1):27-40. doi:10.1007/s10964-011-9713-z

19. Vartanian LR, Shaprow JG. Effects of Weight Stigma on Exercise Motivation and Behavior: A Preliminary Investigation among College-aged Females. *J Health Psychol*. 2008;13(1):131-138. doi:10.1177/1359105307084318

20. Losekam S, Goetzky B, Kraeling S, Rief W, Hilbert A. Physical Activity in Normal-Weight and Overweight Youth: Associations with Weight Teasing and Self-Efficacy. *OFA*. 2010;3(4):239-244. doi:10.1159/000319433

21. Hayden-Wade HA, Stein RI, Ghaderi A, Saelens BE, Zabinski MF, Wilfley DE. Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. *Obesity research*. 2005;13(8):1381-1392.

22. Slater A, Tiggemann M. Gender differences in adolescent sport participation, teasing, self-objectification and body image concerns. *Journal of adolescence*. 2011;34(3):455-463.

23. Auerbach RP, Alonso J, Axinn WG, et al. Mental disorders among college students in the World Health Organization World Mental Health Surveys. *Psychol Med*. 2016;46(14):2955-2970. doi:10.1017/S0033291716001665

24. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of personality and social psychology*. 1988;54(6):1063.

25. Roberts KR, Dimsdale J, East P, Friedman L. Adolescent emotional response to music and its relationship to risk-taking behaviors. *Journal of Adolescent Health*. 1998;23(1):49-54. doi:10.1016/S1054-139X(97)00267-X

26. Jones LW, Sinclair RC, Courneya KS. The effects of source credibility and message framing on exercise intentions, behaviors, and attitudes: An integration of the elaboration likelihood model and prospect theory 1. *Journal of applied social psychology*. 2003;33(1):179-196.

27. Puyau MR, Adolph AL, Vohra FA, Butte NF. Validation and calibration of physical activity monitors in children. *Obesity research*. 2002;10(3):150-157.

28. Trost SG, Pate RR, Freedson PS, Sallis JF, Taylor WC. Using objective physical activity measures with youth: how many days of monitoring are needed? *Medicine & Science in Sports & Exercise*. 2000;32(2):426.

29. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. *Medicine & Science in Sports & Exercise*. 2008;40(1):181-188.

30. Myers A, Rosen JC. Obesity stigmatization and coping: relation to mental health symptoms, body image, and self-esteem. *International journal of obesity*. 1999;23(3):221-230.

31. Vartanian LR. Development and validation of a brief version of the Stigmatizing Situations Inventory. *Obesity Science & Practice*. 2015;1(2):119-125. doi:https://doi.org/10.1002/osp4.11

32. Vartanian LR, Novak SA. Internalized Societal Attitudes Moderate the Impact of Weight Stigma on Avoidance of Exercise. *Obesity*. 2011;19(4):757-762. doi:https://doi.org/10.1038/oby.2010.234

33. Seacat JD, Mickelson KD. Stereotype threat and the exercise/dietary health intentions of overweight women. *Journal of Health Psychology*. 2009;14(4):556-567.

34. Pearl RL, Dovidio JF, Puhl RM, Brownell KD. Exposure to Weight-Stigmatizing Media: Effects on Exercise Intentions, Motivation, and Behavior. *Journal of Health Communication*. 2015;20(9):1004-1013. doi:10.1080/10810730.2015.1018601

35. Prince SA, Adamo KB, Hamel ME, Hardt J, Gorber SC, Tremblay M. A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review. *International Journal of Behavioral Nutrition and Physical Activity*. 2008;5(1):56. doi:10.1186/1479-5868-5-56

36. Nelson MC, Story M, Larson NI, Neumark-Sztainer D, Lytle LA. Emerging Adulthood and College-aged Youth: An Overlooked Age for Weight-related Behavior Change. *Obesity*. 2008;16(10):2205-2211. doi:https://doi.org/10.1038/oby.2008.365

37. Carels RA, Young KM, Wott CB, et al. Weight Bias and Weight Loss Treatment Outcomes in Treatment-Seeking Adults. *Annals of Behavioral Medicine*. 2009;37(3):350-355. doi:10.1007/s12160-009-9109-4

Chapter 6

Summary, Implications, and Future Directions

General conclusion and implications

This dissertation focused on expanding the COBWEBS model to fill in gaps within the model related to physical activity, metabolic risk factors that may precede further weight gain, and the psychological constructs underlying physical activity and eating behavior. Specifically, this dissertation builds upon the COBWEBS model by: 1) including and investigating key psychobehavioral constructs that may underlie eating and physical activity behavior; 2) including physical activity—an important health behavior that must be considered as it relates to energy balance; 3) including additional consideration of metabolic risk factors that may precede further weight gain in adolescents with overweight/obesity; 4) including and exploring the influence of weight stigma on health behavioral intentions among older adolescents/young undergraduate students. Together, findings from this dissertation suggest that psychological stress affects behavioral antecedents such as physical activity enjoyment, and that greater susceptibility to the effects of stress on eating behavior is pronounced among those with disordered eating behavior. Additionally, findings from this dissertation suggests that neither physical activity, food intake, nor cortisol contributes to the relationship between psychological stress and T2D risk among non-Hispanic Black and White adolescents with overweight/obesity, rather race (conceptualized as a social construct) plays an important moderating role in the relationship between stress and T2D risk in this group. Lastly, the findings from this dissertation also suggests that the stressor of weight stigma leads to the avoidance of engaging in physical activity. The present findings provide preliminary data for longitudinal studies, and suggests that preventative approaches are needed to address these behavioral precursors, which in turn may help interrupt the cycle of stress and weight gain among adolescents with overweight/obesity.

Stress and its implications for physical activity enjoyment and engagement

The first study of this dissertation examined the cross-sectional associations of psychological stress on physical activity enjoyment and physical activity participation in adolescents with overweight/obesity. In addition to that, study one of this dissertation also sought to examine the potential moderating effect of physical activity enjoyment on the association between stress and physical activity participation. Using cross-sectional secondary data analysis from the SODA and HCP studies, the key takeaway from this study was that greater psychological stress was associated with lower enjoyment of physical activity among adolescents with overweight/obesity. Contrary to our hypothesis, psychological stress was not a predictor of physical activity engagement, nor was physical activity enjoyment a significant moderator in the relationship between stress and physical activity engagement. Given the importance of physical activity enjoyment to long-term physical activity participation and the high levels of stress often experienced by adolescents with overweight/obesity, study 1 results are notable and point again to the value of adding physical activity to the COBWEBS model. It is plausible that higher levels of stress may further worsen self-perceptions (e.g. insecurity about appearance, confidence) about physical activity among adolescents with overweight and obesity, which may then influence actual participation in physical activity among adolescents with overweight/obesity in the long-term. Not only may stress influence self-perceptions about physical activity, but its negative association with physical activity enjoyment (a predictor of sustained physical activity engagement) further suggests that experiencing stress chronically may also lead to decreased long-term physical activity participation. Thus, in order to break the cycle of weight gain among adolescents with higher weight status, it is imperative to improve self-perceptions regarding one's ability to engage in physical activity. Just as importantly, making physical activity a 'fun' activity that anyone regardless of body size and ability can participate in through gamification for example, may be helpful in combating the immediate upstream effects of psychological stress. Lastly, it is important to equip adolescents with healthy stress coping strategies, such as using physical activity as a coping outlet, but just as important, school administrators and families must foster social environments wherein negative social experiences such as weight-based bullying and stigmatization are not viewed as normal.

Stress and its implications for eating behavior among adolescents with overweight/obesity

The second study of this dissertation examined the cross-sectional associations between acute stress dimensions and caloric intake in adolescents with overweight/obesity, and the potential moderating effect of disordered eating behavior on these associations. Using cross-sectional secondary data analysis from the SODA and HCP studies once again, the key takeaway from this study was that higher frequency and totality of acute daily stressors was associated with higher caloric intake among adolescents with overweight/obesity. Moreover, adolescents who engage in higher levels of disordered eating behavior were the most susceptible to having high food intake. Thus, in acute circumstances or situations where adolescents with overweight/obesity feel overwhelmed with demands beyond that of the resources they have at their disposal, they may turn to increasing food intake as a means of coping. If experiencing these acute stressors in a reoccurring manner over longer periods of time, it is plausible that these adolescents may further gain weight and be predispose to higher risk of cardiometabolic disease. Findings from this study, further confirm the COBWEBS model, and reaffirm the importance of addressing the role of stress and its differing dimensions in propagating food intake and weight gain among youth and adults. Moreover, special attention must be paid to adolescents engaging in high levels of disordered eating behaviors, as this particularly group may already have a dysregulated relationship with food that is then worsen when experiencing bouts of stress. It is important to note that although acute daily stress dimensions were associated with higher food intake, more long-term psychological stress measured using the Perceived Stress Scale (PSS-14) was not associated with food intake in the sample ($p>0.05$). This analysis was done separately and was not included in the second study of this dissertation as it was outside of the scope of acute stress. Considering however, that other studies have demonstrated the impact of chronic stress on increasing food intake[1], more studies are needed exploring specific types of stress and stress appraisal as it relates to food intake among adolescents with overweight/obesity.

Stress and increased metabolic disease among Black adolescents

The third study of this dissertation explored racial differences in the association between psychological stress and measures of T2D risk (S_i, DI, and AIR_g—assessed via an intravenous glucose tolerance test or IVGTT) among non-Hispanic White and Black adolescents with overweight/obesity. Using cross-sectional secondary data analysis from the SODA study, the key takeaway from this study was that psychological stress may have a differential impact on metabolic function and T2D risk among Black and White adolescents with overweight/obesity. More specifically, for non-Hispanic Black adolescents, higher levels of stress was associated with lowered insulin sensitivity, whereas for non-Hispanic White adolescents, higher levels of stress were associated with higher insulin sensitivity. Additionally, in contrast with our hypothesis, stress was not associated with any metabolic markers of risk (S_i, AIR_g, or DI) when data from non-Hispanic Black and White adolescents where analyzed together as a larger sample. Because the different racial association observed between stress and T2D risk was only observed with S_i and not AIR_g or DI, it is plausible that the increased risk of T2D among Black adolescents works through a skeletal muscle insensitivity pathway as opposed to pancreatic beta-cell dysfunction pathway. Given the increased stress exposure experienced by non-Hispanic Black adolescents—through such forms as racial discrimination, community and environmental violence for example, it is likely that the racial difference in T2D risk among Black and White adolescents is driven in part by stress. Nevertheless, because the positive association between stress and S_i among non-Hispanic White adolescents, more studies are needed that examine the racial differences in the association between stress and insulin sensitivity among adolescents with overweight/obesity and the influence of stress appraisal and coping resources. In so doing, our understanding of the factors that buffer or otherwise alter this relationship among the two racial groups will be clearer.

It is important to also note the original aims of the third study of this dissertation; which were to examine biobehavioral pathways that link psychological stress to a metabolic risk factor (insulin resistance) among adolescents with overweight/obesity. In models examining these aims through several linear regression models, cortisol patterning (diurnal cortisol and cortisol awakening response—CAR) alone was observed to be associated with android fat mass (a proxy measure for abdominal fat) (diurnal cortisol: $\beta=0.05\pm0.02$; $p=0.029$ and CAR: $\beta=0.04\pm0.02$; $p=0.031$), respectively. However, cortisol patterning was not associated with stress, S_i, AIR_g, nor DI (all ps>0.05). Additionally, all other possible pathways explored (physical activity, caloric intake) linking psychological stress and T2D risk were also found to be insignificant for the study sample. Although findings from the original aims do not suggest a connection between stress and T2D risk through a physical activity or caloric intake pathway, it is likely that other social-environmental factors are at play connecting stress to T2D risk beyond and/or in addition to physical activity and caloric intake. Thus, additional studies exploring stress exposure types, stress appraisal, and coping resources for example are needed to confirm or refute the results of study 3.

Weight stigma: a prominent stressor and its implications for physical activity

The fourth study of this dissertation explored the associations between prior weight stigma experiences, physical activity intentions (positive intent and avoidance), and physical activity behavior among older adolescents/young undergraduate college students. In addition, the study also examined the acute effects of a weight stigma exposure on physical activity intentions, and physical activity behaviors among the same participants. Using primary data collected from the Attitudes Regarding Eating and Activity (AREA) study, the key takeaway from this study was that greater prior experiences of weight stigma are linked with higher avoidance of physical activity. This finding, although not surprising given evidence from other researchers, is distressing. Distressing because of the well-established links between participation in physical activity and improved physical and mental wellbeing. Because the negative consequences of weight stigma are long lasting and track into adulthood[2], higher exposure to weight stigma may lead adolescents and young adults to avoid engaging in physical activity across their lifespan. And as is well described within the COBWEBS model, if the stressor of weight-stigma is persistent, a cycle of increased weight gain will ensue.

This study adds to our knowledge of the association between weight-stigma and persistent weight gain, by illustrating another plausible pathway by which the stressor of weight stigma leads to weight gain—through avoidance of physical activity. This in turn will have major implications for mental and physical health risks. Thus, intervening to find strategies to lessen or eliminate the negative consequences of weight stigma is crucial. Strategies that include but are not limited to increasing enjoyment of physical activity via interventions focused on the intrinsic value of physical activity, and use of positive portrayals of individuals with overweight/obesity in the media when exercising. Additionally, interventions targeting multiple levels of the social ecological model (e.g. interpersonal, community, and policy levels) to assist and equip individuals with the resources to overcome the demands of the stressor that is weight stigma should be encouraged.

Harkening back to the original aims of the study, part of the purpose of the study was to examine the associations between weight stigma exposure, eating intentions, and eating behavior (measured as caloric intake). In addition to exploring these aforementioned aims, an additional sub-aim to examine the possible moderating effect of stress on the associations between weight stigma and health promoting behavioral intentions (both physical activity and healthy eating intentions) was proposed. As it relates to eating, it was observed that prior weight stigma experiences was not associated with healthy eating intentions (β= -30.7±84.4; p=0.72), caloric intake (β= -24.8±59.8; p=0.68), nor were there any difference in healthy eating intention scores (weight-stigma: 4.72±0.59 vs. 4.75±0.49) or caloric intake (weight-stigma: 2,111±162.3 kcals/day vs control: 1,972±194.5 kcals/day; p=0.59) across experimental conditions. When perceived stress was added as a moderator in the regression analysis, it did not significantly moderate the association between prior weight stigma experiences and caloric intake, nor between prior weight stigma experiences and eating intentions (ps>0.05).

Recommendations for future directions

The majority of the studies conducted as part of this dissertation are cross-sectional in nature, and thus limitations regarding causality must be noted, and longitudinal studies are needed to understand the long-term effects of stress on the outcomes examined throughout this dissertation. Nevertheless, the studies of this dissertation can inform future interventions and also build upon current interventions. Specifically, as it relates to psychological stress, evidence from prior studies as well as this dissertation, make it undoubtedly clear of the negative effects of stress on physiological and psychological homeostasis, as well as multitude of health behaviors. Because adolescence is a critical developmental period where the adolescent brain is maturing, it is an especially vulnerable period to external factors such as stress.

As the findings of this dissertation appropriately highlight, physical activity must not be neglected when considering the effects of stress on weight gain. Not only must physical activity be considered within the COBWEBS model, but physical activity may be one of the solutions to counteracting the effects of stress by alleviating the negative health effects of stress and even improving psychological wellbeing. More specifically, exercise may buffer the negative effects of stress through physiological adaptation to the stress response system—through what is known as the cross-stressor adaptation hypothesis. This hypothesis simply states that regular exercise, leads to biological adaptations which in turn contributes to reduced physiological responses during subsequent exercise and other non-exercise related stressors[3,4]. Habituation of the stress system through regular exercise and increased fitness leads to decreased amplitude of stress responses (e.g. cortisol or alpha-amylase responses). Several studies have shown that acute exercise dampens stress reactivity, in turn protecting against the detrimental effects of increased stress reactivity on cardiovascular health[5,6]. For example, Lambiase and colleagues tested whether a simulated active commute to school would dampen against cardiovascular and psychological stress responses following a laboratory stressor. They found that in comparison to a sedentary control group, that those who had engaged in physical activity (i.e. the active commute group) had lowered cardiovascular reactivity and psychological stress in response to the laboratory stressor[5]. Similarly, Roemmich et al., found that 20 min of interval exercise led to decreased cardiovascular reactivity following a laboratory stressor among children ages 8-12 years old compared with their peers who were in a sedentary control group[6]. In summary, not only can exercise dampen stress reactivity, but it can also play a role in preventing further weight gain when individuals are regularly participating in physical activity. Thus, exercise jointly decreasing the long-term disease risk among adolescents through reductions in stress and weight. As such, future interventions addressing stress exposure among adolescents with overweight and obesity, should not neglect but rather must consider the importance of physical activity as a means of helping reduce stress and increase both psychological and physiological resilience in this group of adolescents. Interestingly however, it was also observed that physical activity, caloric intake, and cortisol did not contribute to the relationship between psychological stress and T2D risk among non-Hispanic Black and White adolescents with overweight/obesity. Rather it was found that race moderated the association between psychological stress and T2D risk among the adolescent participants. It is likely that other factors beyond these behavioral and biological measures are playing a role in the association between stress and T2D risk. Factors such as social support and its numerous dimensions, stress appraisal, and/or more specific measures of stress beyond that of perceived stress or chronic stress measured via cortisol may be contributing or driving the association. Given that race moderated the association between stress and T2D risk, with higher levels of stress associated with lowered insulin sensitivity among non-Hispanic Black participants, it is likely that factors such as racism, and racial discrimination may be underlying and in turn, driving the association between stress and heighted T2D risk among Black participants. As such, it is imperative for researchers and interventionist to create novel programs and interventions to lower stress and increase resilience among non-Hispanic Black adolescents, and for policy makers to enact policies that address the numerous socioenvironmental stressors faced by all adolescents, and especially those experienced by non-Hispanic Black adolescents.

References

1. Adam TC, Epel ES. Stress, eating and the reward system. Physiology & Behavior. 2007;91(4):449-458. doi:10.1016/j.physbeh.2007.04.011

2. Christensen S. Weight Bias and Stigma in Children. Journal of Pediatric Surgical Nursing. 2018;7(3):72-74. doi:10.1097/JPS.0000000000000178

3. Sothmann MS. The Cross-Stressor Adaptation Hypothesis and Exercise Training. Published online 2006.

4. Gerber M, Pühse U. Review Article: Do exercise and fitness protect against stress-induced health complaints? A review of the literature. Scand J Public Health. 2009;37(8):801-819. doi:10.1177/1403494809350522

5. Lambiase MJ, Barry HM, Roemmich JN. Effect of a simulated active commute to school on cardiovascular stress reactivity. Medicine and science in sports and exercise. 2010;42(8):1609.

6. Roemmich JN, Lambiase M, Salvy SJ, Horvath PJ. Protective effect of interval exercise on psychophysiological stress reactivity in children. Psychophysiology. 2009;46(4):852-861.