THE EFFECT OF FOOD MARKETING AND ATTENTIONAL BIASES ON EATING BEHAVIORS IN AFRICAN AMERICAN ADOLESCENT GIRLS

by

Omni Cassidy

Dissertation submitted to the Faculty of the
Medical and Clinical Psychology Graduate Program
Uniformed Services University of the Health Sciences
In partial fulfillment of the requirements for the degree of
Doctor of Philosophy 2019

Distribution Statement

Distribution A: Public Release.

The views presented here are those of the author and are not to be construed as official or reflecting the views of the Uniformed Services University of the Health Sciences, the Department of Defense or the U.S. Government.

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
SCHOOL OF MEDICINE GRADUATE PROGRAMS
Graduate Education Office (A 1045), 4301 Jones Bridge Road, Bethesda, MD 20814

April 2, 2019

MEMORANDUM FOR: **DR. MARIAN TANOFSKY-KRAFF**
DR. TRACY SBROCCO
DR. ANDREW WATERS
DR. PATRICK DELEON
DR. JENNIFER ROBERTS

SUBJECT: Appointment to the Final Doctoral Examination Committee for Omni Cassidy

In accordance with the School of Medicine Graduate Program Guidelines, you are formally appointed to the Final Examination Committee of Omni Cassidy, doctoral student in the Department of Medical and Clinical Psychology.

This Committee will evaluate the scope and quality of the student's Dissertation and certify the document's acceptability.

In addition, the Committee will conduct an Oral Examination of the student's scientific knowledge, as well as the knowledge and understanding of the thesis research project and Dissertation. This examination constitutes the Private Defense.

The Private Defense for Omni Cassidy will take place on Thursday, April 25, 2019 at 12:00 PM in room G256.

Dr. Marian Tanofsky-Kraff is appointed Chair of the Examination Committee. Dr. Tanofsky-Kraff will oversee the examination. Passage of the Private Defense will be by majority vote. If corrections to the Dissertation are needed, the Chair may oversee this process and sign-off after the corrections are made.

The Committee Chair should return the signed and dated Final Examination/Private Defense Form and the Dissertation Approval Form to the Graduate Education Office.

GEO thanks you in advance for your participation in the SOM Graduate Program process.

[redacted]

Gregory Mueller, Ph.D.
Associate Dean for Graduate Education

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
SCHOOL OF MEDICINE GRADUATE PROGRAMS
Graduate Education Office (A 1045), 4301 Jones Bridge Road, Bethesda, MD 20814

FINAL EXAMINATION/PRIVATE DEFENSE FOR THE DEGREE OF DOCTOR OF PHILOSOPHY
IN THE DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

Name of Student: Omni Cassidy

Date of Examination: April 25, 2019
Time: 12:00 PM
Place: Room G256

DECISION OF EXAMINATION COMMITTEE MEMBERS:

	PASS	FAIL
[redacted]	✓	
Dr. Marian Tanofsky-Kraff DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY Committee Chairperson		
[redacted]	✓	
Dr. Tracy Sbrocco DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY Dissertation Advisor		
[redacted]	✓	
Dr. Andrew Waters DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY Committee Member		
[redacted]	✓	
Dr. Patrick DeLeon GRADUATE SCHOOL OF NURSING & DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY Committee Member		
[redacted]	X	
Dr. Jennifer Roberts UNIVERSITY OF MARYLAND SCHOOL OF PUBLIC HEALTH Committee Member		

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
SCHOOL OF MEDICINE GRADUATE PROGRAMS
Graduate Education Office (A 1045), 4301 Jones Bridge Road, Bethesda, MD 20814

APPROVAL OF THE DOCTORAL DISSERTATION IN THE DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

Title of Dissertation: "The Effect of Food Marketing and Attentional Biases on Eating Behaviors in African American Adolescent Girls"

Name of Candidate: Omni Cassidy
Doctor of Philosophy Degree
April 25, 2019

DISSERTATION AND ABSTRACT APPROVED:

DATE:

[redacted] 5/30/19
Dr. Marian Tanofsky-Kraff
DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY
Committee Chairperson

[redacted] 5/30/19
Dr. Tracy Sbrocco
DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY
Dissertation Advisor

[redacted] 4/25/19
Dr. Andrew Waters
DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY
Committee Member

[redacted] 4/25/19
Dr. Patrick DeLeon
GRADUATE SCHOOL OF NURSING & DEPARTMENT OF MEDICAL & CLINICAL PSYCHOLOGY
Committee Member

[redacted] 4/25/19
Dr. Jennifer Roberts
UNIVERSITY OF MARYLAND SCHOOL OF PUBLIC HEALTH
Committee Member

ACKNOWLEDGMENTS

What a ride this has been. There are so many people to acknowledge who supported me every step of the way. First off, I would like to acknowledge my advisors, my dream team: Dr. Marian Tanofsky-Kraff, Dr. Tracy Sbrocco, and Dr. Patrick DeLeon. Each one of them had a hand in helping me develop into the student and person I am today. Marian gave me my first opportunity as a research assistant and was the first person to invest in my future as a scientist. It was through her that I met Tracy who became my primary advisor in graduate school. Tracy "got me" and gave me the space to grow and develop into the type of scientist who focused on more just grants and publications, but was dedicated to purposeful science. Meeting Pat when I first became interested in exploring the world of policy truly changed the trajectory of my career forever. Being introduced the policy and being able to use all I had learned from Marian and Tracy to put towards the work of make this world a better place through work at the policy level was my true calling. These three individuals help make me who I am, and I am forever grateful. I would also like to thank my other committee members: Dr. Andrew Waters and Dr. Jennifer Roberts, both of which invested so much time and effort to make my project the best it could be. I would also like to thank Dr. Gloria Ramsey who became my informal mentor throughout graduate school, helping me navigate the academic world as a woman of color. I would also like to thank Dr. Cara Olsen who assisted me with statistics, as well as Dr. Marie Bragg and Dr. Jerome Williams who both served as consultants on my F31/NRSA. I would like to thank Dr. Natasha Schvey who started to invest in me even more she knew me—assisting me with my essays into graduate school back when she was still a graduate student herself. I would also like to thank Dr. Robyn

Osborn who was another person who supported and invested in me as a developed as a young graduate student. I also would like to thank my 2012 cohort who I was in the trenches with for 7 whole years! I would like to thank my department chair, DCT, and the other faculty members, students, and administrative staff of MPS.

I would like to give a very special thank you to my tribe throughout graduate school, the fabulous four: (newly minted!) Dr. Raquel Martin, Sade Soares, and Katrina Powell Brown…also know as the "Fab4." The Fab4 have been my rocks!! These girls have helped keep me afloat, helping me not only as a student (talking me down of ledges, keeping me from anxiety spirals), but also as a woman, aspiring wife and mother, and as black woman. Our monthly girls' nights gave me all the life! I cannot express my gratitude enough for what they mean to me and how unbelievably happy I am go have them in my life. I would also like to thank my friend, former roommate, and newly minted graduate, Dr. Maggie Baisley whose conversations and dialogue helped keep me on my toes. I would also like to thank Joanna Sells who I could also lean during the hard times. I have to also thank all of my friends near and far who supported me a gave me strength throughout this entire journey, particularly Danielle Arnold, Latasha Kinnard, Tiffany Johnson, (soon to be Dr.) Marquisha Lawrence Scott, Darin Allen, and Nicholas Brooks.

Last, but certainly not list, I would like to thank my incredible family. My parents, Milton and Teresa, and my brother, Josh, as well as my extended family members. My parents are literally the best parents anyone could ever ask for. Even if they did not understand my choices or did not quite understand what in the world I was doing with my time, they provided unwavering support and encouragement, and never stopped lifting

me high up. They have literally been here from the beginning, and my love and appreciation for them runs deeper than I can even imagine. I love you all across the universes.

DEDICATION

For Pearline “Bigmama” Jones. You always knew I would be a Doctor someday. I hope you’re looking down with a smile.

COPYRIGHT STATEMENT

The author hereby certifies that the use of any copyrighted material in the dissertation manuscript entitled: “The effect of food marketing and attentional biases on eating behaviors in African American adolescent girls” is appropriately acknowledged and, beyond brief excerpts, is with the permission of the copyright owner.

Omni Cassidy

Department of Medical and Clinical Psychology

Uniformed Services University of the Health Sciences

August 6, 2019

DISCLAIMER

The views presented here are those of the author and are not to be construed as official or reflecting the views of the Uniformed Services University of the Health Sciences, the Department of Defense or the U.S. Government.

ABSTRACT

The effect of food marketing and attentional biases on eating behaviors in African American adolescent girls

Omni Cassidy, M.S., 2019

Thesis directed by: Tracy Sbrocco, Ph.D., Major Advisor, Professor, Department of Medical and Clinical Psychology

Rates of overweight/obesity among Black/African American (hereafter referred to as African American) adolescent girls are greater than rates in other racial/ethnic groups, highlighting a critical health disparity. African American youth with obesity are more likely to remain obese throughout adulthood and to develop health consequences, such as type 2 diabetes and hypertension. Therefore, preventing overweight/obesity in this group is critical. Yet, prevention and intervention efforts must account for the multiple systems that influence obesity risk, specifically cultural, environmental, and individual factors. Given cultural food preferences, African American individuals may be more vulnerable to environmental factors, such as food marketing, compared to other groups. Additionally, African American adolescents with obesity may be more prone to over-attention toward food-related stimuli (i.e., attentional biases [ABs]) increasing the potential role of food marketing. Together, these cultural, environmental, and individual factors may lead to overconsumption of unhealthy foods and increase the risk of obesity

onset. Data were analyzed from 41 African American adolescent girls who were 12-17 years old (M, SD = 14.93, 1.67). Participants viewed a television (TV) episode clip of the *Big Bang Theory* embedded with either four 30-sec food commercials or four 30-second neutral/non-food commercial, based upon prior randomization. ABs towards food-related stimuli were assessed using an AB visual dot probe task. Participants ate *ad libitum* from a laboratory test meal array. Additional variables, such as anthropometrics, mood, and disordered eating were also examined.

The hypotheses were three-fold: 1) Participants in the food marketing condition would consume more total energy compared to participants in the neutral marketing condition, with a stronger relationship among those with obesity; 2) participants with initial and sustained ABs would consume more total energy compared to participants without ABs, with a stronger relationship among those with obesity; and 3) among participants in the food marketing condition, those with greater ABs would consume more total energy and energy from fat, sugar, and sodium compared to all others. Analyses of variance (ANOVAs) and multiple linear regressions were conducted using SPSS, Versions 24 and 25. There were no significant main effects of marketing condition or weight status on total Calories consumed (*ps* = ns). However, adolescents with obesity consumed 239.75 kcal more than non-overweight adolescents, which resulted in a small-to-large effect size (η^2 = .09). There was not a significant marketing condition by weight status interaction on total Calories consumed (*p* = ns). However, adolescents with obesity who were in the food marketing condition consumed 399.58 kcal more than non-overweight adolescents compared to adolescents with obesity who were in the neutral marketing condition who consumed 37.94 kcal more than non-overweight adolescents.

These differences resulted in a small-to-medium effect size (η^2 = .06). *Post hoc* analyses of weight analyzed continuously or examining the impact of ethnic identity were not significant (*p*s = ns).

There were no significant main effects of initial or sustained biases on total Calories (*p*s = ns). BMI*z* did not significantly moderate the relationship between total Calories and initial biases towards high palatable food (versus to nonfood items; HF-NF), initial bias towards low palatable food (versus nonfood items; LF-NF), or initial bias towards high palatable foods (versus low palatable foods; HF-LF; *p*s = ns). BMI*z* also did not significantly moderate the relationship between total Calories and sustained HF-NF bias, sustained LF-NF bias, or sustained HF-LF bias (*p*s = ns). BMI*z* did not significantly moderate the relationship between fat intake and initial HF-NF bias, sustained HF-NF bias, initial HF-LF bias, or sustained HF-LF bias (*p*s = ns). However, sustained HF-LF significantly predicted arcsin fat after controlling for BMI*z* (Change in R^2 = .33, $F(1, 21)$ = 10.51, p = .004)), and the overall model was also significant (Overall R^2 = .35, $F(3, 20)$ = 3.53, p = .03; (*p*s = ns). The overall initial HF-LF by BMI*z* significantly moderated the relationship between initial HF-LF bias and sugar (Overall R^2 = .24, p = .02), and initial HF-LF bias and salt intake (Overall R^2 = .19, p = .04), although the overall models did not reach significance (p = ns). Sustained HF-LF bias significantly predicted arcsin fat after controlling for BMI*z* (Change in R^2 = .33, p = .004, $F(2, 21)$ = 5.37, p = .01), and the overall model was also significant (Overall R^2 = .35, $F(3, 20)$ = 3.53, p = .03). Sustained HF-LF significantly predicted sugar intake after controlling for BMI*z* (Change in R^2 = .17, p = .04), although the overall model did not reach significance (*p*s = ns). There were no significant effects for sustained HF-LF and BMI*z* on salt (p = ns).

Marketing condition significantly predicted fat intake after controlling for initial HF-NF bias (Change in $R^2 = .18, p = 05$), initial LF-NF bias (Change in $R^2 = .17, p = 05$), and initial HF-LF bias (Change in $R^2 = .19, p = 04$), although the overall models did not reach significance (*p*s = ns). Initial HF-NF bias did not significantly moderate the relationship between initial HF-NF bias and intake of sugar or salt (*p*s = ns). Sustained HF-NF bias nor sustained LF-NF bias significantly moderated the relationship between marketing condition and intake of total Calories, fat, sugar, or salt (*p*s = ns). Sustained HF-LF did not significantly moderate the relationship between marketing condition and intake of total Calories or salt (*p* = ns). Sustained HF-LF predicted intake of fat ($R^2 = .33$, $F(1, 22) = 10.83, p = .003$), and the overall model was also significant (Overall $R^2 = .38$, $F(3, 20) = 4.16, p = .02$). Sustained HF-LF did not significantly moderate this effect (*p* = ns). Sustained HF-LF also predicted intake of sugar ($R^2 = .21$, $F(1, 22) = 5.67, p = .03$), although the overall model did not reach significance (*p* = ns). Data must be interpreted cautiously due to the small sample size and limited statistical power. Findings provide support for future work to better elucidate the associations among culture, environmental, and individual factors that may contribute to increased intake and excessive weight gain among African American adolescent girls.

TABLE OF CONTENTS

LIST OF TABLES xvi

LIST OF FIGURES xvii

CHAPTER 1: BACKGROUND 1

Introduction 1
The Problem of Obesity 1
The Socio-Ecological Model and Unhealthy Eating 2
The Proposed Project 3
The Obesity Epidemic 4
Prevalence 4
Obesity-Related Health Consequences 5
Etiology of Obesity 7
Energy Imbalance 7
Eating Behaviors 8
Energy Consumption among African American Adolescents 10
Total Energy 10
Fat 11
Sugar 11
Sodium 12
Brief Summary 13
Socio-Ecological Factors Contributing to Unhealthy Eating 14
Modern Developments of Socio-Ecological Models of Obesity Risk 15
Socio-Ecological Factors of Unhealthy Eating in the Proposed Study 23
Culture 23
African Americans and Food 23
Environment 26
The Current Food Environment 27
Purpose and Procedure of General Marketing 27
Food Marketing 29
Food Marketing to Youth and Racial/Ethnic Minorities 29
African American Adolescents as Highly Vulnerable to Food Marketing 31
Food Marketing, Unhealthy Eating Behaviors, and Obesity Risk 32
Cognition: Attentional Biases as a Potential Mechanism 36
ABs, Eating, and Weight 38
ABs and Eating 38
ABs and Body Weight 40
Measuring the Effect of Food Marketing and Food-cue ABs on Energy Intake 41
Summary 43
Innovation and Policy Impact 44

Current Study 44
Aims and Hypotheses 45
CHAPTER 2: METHODS 49
Study Design 49
Recruitment 49
Inclusion and Exclusion Criteria 51
Study Procedure 52
TV Episode with Embedded Commercials 55
Selection of TV Episode 55
Selection of Commercials 56
Measures 59
Demographics 59
Anthropometrics 60
Hunger 60
Food-related ABs 60
Food Preferences 61
Observed Energy Intake 62
Disordered Eating Behaviors 63
Mood 63
Parent Reported Child Behavior 64
Ethnic Identity 64
Advertisement Attitudes and Degree of Targetedness 65
Perception of Relationships Depicted on the TV Episode 66
Data Analytic Plan 66
A Priori Sample Size Estimation 66
Statistical Analyses 67
Preparing AB Data 68
Specific Aim 1: Food Marketing and Weight on Total Energy Intake 69
Hypothesis 1a 69
Hypothesis 1b 70
Specific Aim 2 70
Hypothesis 2a 70
Hypothesis 2b 71
Specific Aim 3 71
Hypothesis 3a-3b 71
CHAPTER 3: RESULTS 73
Sample Characteristics 74
Meal Consumption 76
Ratings of Hunger and Craving 77
Results of Specific Aim 1: Food Marketing and Weight on Total Energy Intake 79
Specific Aim 1a: Food marketing on total energy intake 79
Exploratory post hoc analyses: Marketing condition on fat, sugar, and salt intake 80

Specific Aim 1b: Food Marketing and Weight on Energy Intake 81
Total Food Intake by Marketing Condition and Weight Status 81
Exploratory Post Hoc Analyses 82
Examining weight as a continuous variable 82
Examining the impact of ethnic identity on energy intake 83
Results of Specific Aim 2: Attentional Biases and Weight on Total intake 84
Specific Aim 2a: Attentional Biases on Total Energy Intake 84
Biases in Initial Attention and Energy Intake 84
Biases in Sustained Attention and Energy Intake 86
Specific Aim 2b: Attentional Biases, Weight, and Total Energy Intake 87
Biases in Initial Attention, Weight, and Energy Intake 87
Biases in Sustained Attention, Weight, and Energy Intake 87
Post Hoc Exploratory Analyses 94
Examining the impact of biases towards highly palatable foods and weight on fat, sugar, and salt intake 94
Results of Specific Aim 3: Food marketing and attentional biases on Total energy, Fat, Sugar, and Salt intake 98
Specific Aim 3a: Marketing, Initial Attentional Biases, and Intake 98
Specific Aim 3b: Marketing, Sustained Attentional Biases, and Intake 112

CHAPTER 4: DISCUSSION 125

Summary & Discussion 125
Strengths 135
Limitations 135
Future Directions and Conclusion 138

REFERENCES 142

APPENDIX 161

Appendix A. Demographics Questionnaire 161
Appendix B. Body Measurements Questionnaire 162
Appendix C. Hunger ratings Questionnaire 163
Appendix D. Food Preferences Questionnaire 166
Appendix E. Eating Disorder Examination, Overeating Section 169
Appendix F. Positive and Negative Affect Scale 188
Appendix G. Brunel Mood Scale 189
Appendix H. Child Behavior Checklist 190
Appendix I. Cross Racial Identity Scale 194
Appendix J. Advertisement Attitudes and Degree of Targetedness 197
Appendix K. Relationship and Communication Skills Questionnaire 206
Appendix L. Referrals 209
Appendix M. Participant Instructions 227
Appendix N. Attentional Bias Computer Task 228
Appendix O. Explanation of True Study Intent 229
Appendix P. *Post Hoc* Analyses 230
Appendix Q. Missing/Excluded Variables 264

LIST OF TABLES

Table 1. Estimated Caloric needs and limits on intake of fat, sugar, and sodium in pre-adolescent and adolescent females. 13
Table 2. Sample characteristics based upon condition. 75
Table 3. Meal consumption 77
Table 4. VAS hunger ratings 79
Table 5. Initial HF-NF bias and BMI*z* on total Calories 88
Table 6. Initial LF-NF bias and BMI*z* on total Calories 89
Table 7. Initial HF-LF bias and BMI*z* on total Calories 90
Table 8. Sustained HF-NF bias and BMI*z* on total Calories 91
Table 9. Sustained LF-NF bias and BMI*z* on total Calories 92
Table 10. Sustained HF-LF bias and BMI*z* on total Calories 93
Table 11. Sustained HF-LF bias and BMI*z* on arcsin fat 96
Table 12. Sustained HF-LF bias and BMI*z* on sugar 97
Table 13. Marketing condition and initial HF-NF bias on total Calories 100
Table 14. Marketing condition and initial HF-NF bias on arcsin fat 101
Table 15. Marketing condition and initial HF-NF bias on sugar 102
Table 16. Marketing condition and initial HF-NF bias on salt 103
Table 17. Marketing condition and initial LF-NF bias on total Calories 104
Marketing x LF-NF Bias§ 104
Table 18. Marketing condition and initial LF-NF bias on arcsin fat 105
Table 19. Marketing condition and initial LF-NF bias on sugar 106
Table 20. Marketing condition and initial LF-NF bias on salt 107
Table 21. Marketing condition and initial HF-LF bias on total Calories 108
Table 22. Marketing condition and initial HF-LF bias on arcsin fat 109
Table 23. Marketing condition and initial HF-LF bias on sugar 110
Table 24. Marketing condition and initial HF-LF bias on salt 111
Table 25. Marketing condition and sustained HF-NF bias on total Calories 113
Table 26. Marketing condition and sustained HF-NF bias on arcsin fat 114
Table 27. Marketing condition and sustained HF-NF bias on sugar 115
Table 28. Marketing condition and sustained HF-NF bias on salt 116
Table 29. Marketing condition and sustained LF-NF bias on total Calories 117
Table 30. Marketing condition and sustained LF-NF bias on arcsin fat 118
Table 31. Marketing condition and sustained LF-NF bias on sugar 119
Table 32. Marketing condition and sustained LF-NF bias on salt 120
Table 33. Marketing condition and sustained HF-LF bias on total Calories 121
Table 34. Marketing condition and sustained HF-LF bias on arcsin fat 122
Table 35. Marketing condition and sustained HF-LF bias on sugar 123
Table 36. Marketing condition and sustained HF-LF bias on salt 124

LIST OF FIGURES

Figure 1. African American Collaborative Obesity Research Network (AACORN) obesity research paradigm (147). 16
Figure 2. Proposed obesity research schematic depicting obesity determinants and drivers spanning the environmental, behavioral, and physiological domains. 19
Figure 3. Proposed framework to conceptualize obesity risk in African American adolescent girls 22
Figure 4. Proposed mechanism by which attentional biases moderate the effect of food marketing on overeating. 43
Figure 5. Study timeline for one-time visit 53
Figure 6. Laboratory test meal (approximately 4,400 kcal) 63
Figure 7. Study flow 73
Figure 9. *Post hoc* analyses of differences between food and neutral marketing condition on (a) fat, (b) sugar, and (c) salt consumed. *Back transformed to assist interpretation 81
Figure 10. Difference in consumption of Calories among obese and non-overweight adolescents in the food and neutral marketing conditions. 82
Figure 11. Initial biases on total Caloric intake. 85
Figure 12. Sustained biases on total Caloric intake. 86

CHAPTER 1: BACKGROUND

INTRODUCTION

The Problem of Obesity

Obesity is a major public health concern (197), particularly within the Black/African American (hereafter referred to as African American) community and among African American adolescents girls (85; 200). Obesity places youth at risk of myriad adverse health outcomes, including cardiovascular disease and type 2 diabetes (66; 295). During adolescence, obesity also places African American girls at risk of remaining obese into adulthood (85; 93) underscoring the need to prevent excessive weight in this vulnerable group.

Unhealthy eating behaviors may contribute to the high obesity rates (187; 261) and obesity-related health risk (262) among African American adolescent girls. Although reports must be interpreted cautiously, some data suggest that African American individuals consume more overall energy compared to other racial/ethnic groups (131; 166) and foods high in saturated or *trans* fat (hereafter referred to as fat); simple sugars, including added sugar and sugar-sweetened beverages (hereafter referred to as sugar); and sodium (47; 131; 134; 298). The most effective targets of nutritional interventions for African American adolescent girls remain unclear (163) and has led to limited success (e.g., 157). Thus, determining most effective intervention targets is a major research and clinical inquiry (164). Kumanyika and colleagues (164) have recommended a broader sociocultural approach to understanding obesity risk within African American communities (**Figure 1**). Indeed, unhealthy eating practices that may lead to obesity (125) likely result from various socio-ecological factors (128; 290) spanning several

spheres, including political, environmental, cultural, community, familial, and individual factors.

However, most research to date—in an effort to maintain scientific rigor—have primarily focused upon very specific research variables that may not account for multi-level systems of influence (128; 190). The proposed study extends the current literature by examining three socio-ecological factors influencing unhealthy eating behaviors, and subsequent obesity risk, among African American adolescent girls: cultural, environmental, and individual.

The Socio-Ecological Model and Unhealthy Eating

The socio-ecological perspective (37) has recently resurged and is viewed as a valuable and necessary line of research to capture the collection of drivers of human behavior (128; 164; 290). Theories suggest that unhealthy eating behaviors result from various socio-ecological factors spanning the cultural, environmental, and individual domains (164; 253). For instance, cultural food preferences may intersect with one component of the "obesogenic" environment (38; 252; 253): food marketing (for review, see 34). African American youth are inundated with food advertisements that may promote unhealthy eating behaviors (216; 219). Researchers have purported that food marketing targeted towards African American individuals are designed to appeal to cultural food preferences for foods high in fat, sugar, or sodium (4; 106; 292). Such food advertisements may exacerbate unhealthy eating behaviors and obesity risk in this group (116; 292). Examining food marketing as an environmental risk factor for unhealthy eating in African American adolescent girls is necessary to elucidate determinants of obesity risk and identify appropriate targets for prevention**.**

Individual maladaptive cognitive processes may elucidate the association between food marketing and unhealthy eating behaviors in African American adolescents girls. Not only are African American youth exposed to more food marketing advertisements compared to other groups (118), they often reside in communities with a disproportionately high density of food cues (20; 219). Among African American youth with obesity (35; 193), external food cues may become highly salient and promote overeating due to attentional biases (ABs; 89; 193; 280). Thus, food-related ABs may be a potential mechanism by which food marketing impacts food consumption and increased weight gain in African American adolescent girls.

The Proposed Project

This dissertation project will investigate the role of the three socio-ecological factors potentially impacting unhealthy eating behaviors in African American adolescent girls: cultural food preferences, food marketing, and food-related ABs. Data will be used to test the proposed mechanism whereby food marketing may serve as a stimulus that precipitates craving and a motivation to alleviate craving by seeking and consuming food, particularly among individuals with ABs. It is hypothesized that food marketing will significantly impact food consumption among African American adolescent girls and that food-cue ABs will moderate the effect. Ultimately, these data have the potential to build scientific support towards important regulatory policies to limits on food marketing towards youth and racial/ethnic minorities and encourage alignment of marketing with nutritional guidelines. Each of the concepts regarding obesity, unhealthy eating, food marketing, and ABs will be reviewed in more detail below. The methodology of the proposed study will also be described.

THE OBESITY EPIDEMIC

Prevalence

Obesity has been identified as a high priority health issue among Americans, particularly individuals (85; 197; 200). According to data from the National Health and Nutrition Examination Survey (NHANES), almost 70% of U.S. adults and over 30% of youth are either overweight (body mass index [BMI][1] = 85th – 94th percentiles for age and sex)or obese (BMI > 95th percentile for age and sex) (56; 85; 200). Although rates in some groups have stabilized over the last 10 years (52; 85), there have generally been steady increases for both adults and youth since 1960 (85; 97; 198-200). Compared to approximately 35% of men, over 40% of women have obesity (85). Prevalence rates of obesity are considerably higher among African American women underscoring a critical health disparity (85). Approximately 57% of African American women have obesity, which is significantly higher than other groups, including White women (38%), African American men (38%), and White men (35%; 85). Thus, preventing high rates of obesity among African American individuals, particularly women, is paramount.

Targeting adolescence, a potentially vulnerable developmental period for excessive weight gain among African American women, is essential. Adolescents (12 – 19 years of age) have the highest obesity rates of all age groups—21% of adolescents have obesity compared to 18% of 6 – 11 year olds and 12% of 2 – 5 year olds (201). Approximately 23% of African American adolescents have obesity (200) with higher rates of obesity in African American adolescent girls (28%) (201). Although differences in obesity rates between African American and White adolescents are not statistically

[1] Calculated by dividing one's height (m) and weight (kg) using the following formula: kg/m^2

significant, evidence suggests that African American girls experience higher weight increases over time as compared to their counterparts (94). In their examination of racial/ethnic differences in 30-year weight trends among pre-adolescents and adolescents, Freedman and colleagues (94) found that African American adolescent boys and girls demonstrated a significantly higher increase in BMI-*z* scores compared to White adolescents (0.72 versus 0.32). What's more, African American girls demonstrated a significantly higher increase in BMI-*z* scores compared to White girls (0.71 versus 0.25) over the 30-year period. Further, significantly more African American girls have obesity that tracks into adulthood compared to their White counterparts (93). While researchers did not report weight changes and trends in African American adolescent girls, specifically, these data suggest that African American adolescent girls may be at particularly high risk of excessive weight gain that continues into adulthood. Recent NHANES data indicating significantly higher rates of obesity in African American women compared to other groups provide support for this conclusion (85; 198). The racial/ethnic disparities in the tracking of obesity into adulthood may indicate other socio-ecological factors contributing to excessive weight gain in African American adolescent girls (16; 30; 48). Identifying the relevant risk factors in order to prevent further weight gain and associated obesity-related health consequences in African American adolescent girls is critical.

Obesity-Related Health Consequences

Prevention of obesity during adolescence was once initiated to prevent health consequences in adulthood (95). However, many of the health conditions previously believed to only affect adults are now affecting youth (66). Mounting evidence

demonstrates that obesity during youth is itself associated with various obesity-related co-morbidities affecting the majority of the bodily systems (66; 295). Some evidence also suggests higher incidence of adverse health outcomes in African American individuals. While data are mixed (53), research suggests that African American adults are at increased risk of cardiovascular disease—the leading cause of death among adults (51)—compared to other groups (98; 144; 179; 186). African American individuals may also develop cardiovascular disease earlier and have higher mortality rates compared to their White counterparts (144; 186). In a prospective examination of heart failure among over 5,000 African American and White adults, African adults were 20 times more likely to develop heart failure compared to White adults (Cumulative incidence, 1.1% among African American women, 0.9% among African American men, 0.08% among White women, 0.0% among White men, 0.0%) (19). Thus, not only may African American girls be at increased risk of excessive weight gain, they might be at higher risk of developing cardiovascular disease.

In addition to cardiovascular disease (66; 92; 95; 295), evidence suggests that obesity places youth at increased risk of type 2 diabetes (65; 173; 211) and other components of the metabolic syndrome, a term that refers to a cluster of symptoms that increase risk for cardiovascular disease (e.g., dyslipidemia; 66; 143). Metabolic changes with obesity may also place individuals at increased risk for certain cancers (100). According to prior data, individuals with obesity are also at increased risk of developing pulmonary (22; 139; 259), gastrointestinal (158; 159), and musculoskeletal (66; 154; 206; 260) health conditions. Obesity during adolescence has also been associated with neurocognitive disturbances (168) and psychological difficulties, including depressive

symptoms and disordered eating behaviors, such as binge or loss of control eating (83; 238; 242). Taken together, African American adolescent girls with obesity are at significantly higher risk of developing health issues (295). Therefore, prevention in this highly vulnerable group is critical. However, in order to determine appropriate preventative strategies, examining the relevant socio-ecological factors that may contribute to the onset and etiology of obesity in this group is paramount (37; 162; 164; 290).

ETIOLOGY OF OBESITY

Energy Imbalance

While various socio-ecological factors contribute to obesity and continued weight gain (128), it is well-established that the development of obesity is the physiological result of energy imbalance, namely positive energy balance—when energy intake exceeds energy expenditure (125). Energy balance comprises of two components: *energy intake* and *energy expenditure*. Energy intake refers to the amount of food consumption measured in Calories as a measurement of energy. Expenditure refers to the amount of energy expended through physiological processes and physical activity. A "neutral" or homeostatic state is accomplished when energy intake and expenditure are equitable, which leads to weight maintenance or stabilization. When expenditure exceeds intake, a "negative" energy balance ensues, which leads to weight loss. In contrast, when intake exceeds expenditure, a "positive" energy balance occurs, which leads to weight gain.

Notably, expenditure and subsequent weight regulation is the result of coordination across various systems, including the metabolic, behavioral, endocrine, and nervous systems (188; 230). Collectively, these systems maintain weight based upon an

ideal “set point” for body fat storage, which is defined by the central nervous system (230). Reports suggest that, following weight loss, the body decreases metabolism and energy expenditure, known as metabolic adaptation or metabolic thermogenesis, in order to re-establish body fat storage (188; 230). Such metabolic adaptation is theorized to contribute to the well-known difficulties in sustaining weight loss over time (230). Persistent metabolic adaptation may further complicate sustained weight loss. In a prospective study of metabolism and body weight in a small sample of individuals with obesity seeking weight loss, metabolism not only significantly decreased from baseline (average of 2,607 kcal/d) to immediately following the weight loss intervention (average of 1,996 kcal/d), but such metabolic adaptation appeared to persist even after 6 years (30-week follow-up versus 6-year follow up; an average of 1,996, 358 kcal/d versus 1,903 kcal/d). Further, while there were no significant associations between metabolism and weight regain at 30-week follow-up, weight regain was significantly correlated with slowed metabolism at 6-year follow-up (91). Taken together, these data provide support for the association between metabolic adaptation and weight loss, which may contribute to weight regain. Reports underscore the critical need to identify factors that may prevent excessive weight gain prior to the onset of obesity. While energy intake and expenditure are both responsible for influencing excessive weight gain over time (125), this review and the subsequent proposed study, will focus upon the role of energy intake (254; 255).

Eating Behaviors

Evidence suggests that the majority of Americans, including adolescents, fail to meet nutritional guidelines for intake of total energy, fat, sugar, and sodium, which may increase their risk of developing diet-related illnesses (137; 161; 192; 240; 270; 281).

According to the U.S. Department of Health and Human Services/U.S. Department of Agriculture (USDA)'s *2015-2020 Dietary Guidelines for Americans*, adolescents should consume an adequate amount of energy (also referred to as "Calories"; based upon age, sex, body weight, and activity level) and limit consumption of fat, sugar, and sodium (270). Guidelines suggest that females 12 – 17 years of age with sedentary or moderate activity levels should consume 1,800 to 2,000 Calories per day, respectively (Table 1; 270). Additionally, adolescents are instructed to consume less than 10% of their daily Calories from fat, less than 10% of their daily Calories from sugar, and no more than 2,200 – 2,300 milligrams (mg) of sodium per day (Table 1; 270).

However, literature suggests that adolescents typically exceed recommendations for intake of total energy, salt, sugar, and sodium (78; 79; 172; 223; 240). Slining and Popkin examined energy intake in over 20,000 youth 2 – 18 years of age from 1994 – 2010 (240). Although pre-adolescents and adolescents consumed fewer Calories from fat between 1994 and 2010 (21% versus 19%), consumption continued to exceed both current (270) and prior dietary guidelines (271). Furthermore, reports suggest that 92 – 98% of pre-adolescents and adolescents exceed recommendations for sugar intake (161). While youth's consumption of sugar significantly declined between 1994 and 2010 (18% versus 14%), estimates continued to exceed dietary guidelines (215; 270). Evidence also suggests that 12 – 19 year old girls consume more sugar compared to their 6 – 11 year old and 2 – 5 year old counterparts (70; 78). Data regarding sodium intake are similar (137). Reports indicate that approximately 93% of youth 2 – 18 years of age exceeded recommendations for consumption of sodium(137). Given the limitations of self-reported dietary recalls (43; 213), consumption estimates among adolescents may be higher than

those reported. Regardless, consumption of total energy, fat, sugar, and sodium appears high and likely places adolescents at increased risk of obesity (124; 125; 142) and obesity-related comorbidities (23; 47; 151; 166; 262; 298).

Energy Consumption among African American Adolescents

Total Energy

Although data evaluating racial/ethnic differences in energy consumption are limited, some (107; 120; 134; 148), but not all (57; 148; 155) reports suggest that African American youth have poorer diets compared to other groups. Haughton and colleagues (120) found that African American pre-adolescents and adolescents were less likely to meet nutritional guidelines for consumption of fruits and vegetables, as well as sugar-sweetened beverages, compared to their White counterparts (adjusted odds ratio [OR], 95% confidence interval [CI], 1.82, 1.04 – 3.17). What's more, in their examination of changes in energy intake among youth, Slining and Popkin (240) found that while total energy consumption in pre-adolescents and adolescents declined from 2,115 Calories per day during 2003 – 2004 to 1,914 Calories per day during 2009 – 2010, a similar decline in consumption was not found among African American youth 12 – 18 years of age(199; 200). On the other hand, some studies have found similar (57; 148) or lower (298) consumption of total energy among African American youth compared to other racial/ethnic groups. While data are mixed, African American adolescent girls may consume increased amounts of total energy, which may precipitate obesity onset or excessive weight gain in this group.

Fat

Data regarding fat intake in youth are also mixed (148; 155). Although differences were not found in total energy intake, Kant and Graubard (148) found that African American youth consumed a higher percentage of fat compared to their White counterparts (34.1% versus 32.6%). By contrast, Kirkpatrick and colleagues (155) found that significantly more African American pre-adolescents and adolescents met guidelines for fat consumption compared to White youth (4.0% versus 2.9%) suggesting that fewer African American youth consumed high amounts of fat. Other reports have found similar percentages of fat consumption between African American and White youth (57; 298). Notably, while differences in the aforementioned studies are statistically significant, differences may not be clinically relevant. Nevertheless, based upon these reports, African American adolescents girls may consume high amounts of fat effectively increasing total energy intake and obesity risk.

Sugar

Some evidence suggests that significantly fewer African American youth meet recommendations for sugar consumption compared to their White counterparts (9.6% versus 10.3%) suggesting higher consumption of sugar among African American youth (155). Another report found that African American youth had significantly higher consumption of sugar-sweetened beverages compared to their White counterparts (225). Similar trends have been demonstrated in other reports (78; 134). However, other reports have found lower consumption of sugar in African American youth (74; 215). Taken together, African American adolescents girls likely consume high amounts of sugar increasing risk for excessive Caloric consumption and subsequent weight gain.

Sodium

Although differences have been shown in adults (298), evidence suggests that African American youth consume similarly high amounts of sodium compared to their White counterparts (13; 126; 137; 298). In their examination of African American and White adolescents 14 – 18 years of age, Zhu and colleagues (298) did not find any significant differences between sodium consumption among African American and White adolescents (an average of 3,197 mg versus 3,359 mg). Similarly, Jackson and colleagues (137) reported that 92% of African American youth 9 – 13 years of age and 87% of African American youth 14 – 18 years of age exceeded recommendations for consumption of sodium. African American adolescents 14 – 18 years of age consumed similar amounts of sodium compared to White adolescents (an average of 86.6 mg versus 94.6 mg). Data from other reports are consistent (13; 126). While consumption in youth is similar, African American youth may be at increased risk of sodium-related health consequences, such as hypertension (99; 102). Therefore, USDA dietary guidelines recommend lower sodium intake for African American youth compared to their counterparts (271).

Table 1. Estimated Caloric needs and limits on intake of fat, sugar, and sodium in pre-adolescent and adolescent females.

Intake[a]	Age		
	9 – 1 3 y	12 – 17 y	14 – 18 y
Total Energy (kcal per day)[b]	2,000	1,600 – 2,000	2,000
Saturated Fat (% kcal)	<10%	–	<10%
Sugar (% kcal)	<10%	–	<10%
Sodium (mg)	2,200	–	2,300

Note: Based upon recommendations from the *2015-2020 USDA Dietary Guidelines for Americans* (270); y = years; kcal = kiloCalories; mg = milligrams; Estimates that were not provided are denoted by a dash (–).

[a]Includes ranges across age groups, as appropriate.

[b]Estimated based upon moderate activity levels.

Brief Summary

Rates of overweight and obesity (199; 200) and risk of tracking obesity into adulthood (93) are high among African American adolescent girls creating a critical health disparity. Obesity status places African American adolescent girls at increased risk of obesity-related comorbidities, such as type 2 diabetes and cardiovascular disease (19; 66). At the physiological level, obesity onset and continued weight gain results from positive energy balance, including increased energy intake (125). Reports suggest that African American adolescents consume a high amount of total energy, fat, sugar, and

sodium (13; 240), which may contribute to high obesity rates in this group. Current reports highlight the socio-ecological factors that may contribute to obesity risk in this group (270). Thus, examining these factors is essential to determining appropriate targets of prevention.

SOCIO-ECOLOGICAL FACTORS CONTRIBUTING TO UNHEALTHY EATING

Given the multi-factorial influence on obesity risk, utilizing a socio-ecological framework to both conceptualize and contextualize risk for excessive weight gain among African American individuals may be particularly useful (150; 164; 167; 249; 290). The socio-ecological model originally developed by Urie Bronfenbrenner (37) was one of the first to posit the ways in which humans interact with, and thus, are altered by their environments. Bronfenbrenner's socio-ecological model (37) outlined four nested systems integral to the understanding of human behavior and development—the microsystem, mesosystem, exosystem, and macrosystem—with the sphere of influence progressively broadening with each subsequent system. Notably, factors within each system may influence factors within the same system or factors in other systems. Thus, the socio-ecological model attempts to capture the fluid nature of human development (37).

The microsystem contains the developing human and includes environments that have an immediate impact on functioning, such as the home, school, or place of worship. The mesosystem, an intermediary system, highlights the effect of the aforementioned environments on behavior. The macrosystem includes the formal and informal social structures that influence environments within the exosystem. According to Bronfenbrenner (37), while the exosystem does not directly influence human behavior, it

creates a “blueprint” that defines the ways environments function within the microsystem. The macrosystem encompasses both implicit and explicit institutional structures that impact the broad perspectives and values of cultures and subcultures. Similar to the exosystem, the author purports that the macrosystem does not directly affect individual behavior, but may significantly influence broader cultural ideals and perspectives (37). These structures include economic, social, educational, legal, and political arenas. Bronfenbrenner (37) theorized that the microsystem, mesosystem, and exosystems are manifestations of the patterns created by the macrosystem.

A strength of the model includes the model’s ability to provide a framework for broadly conceptualizing multi-factorial risk factors of health behaviors. However, it is limited in its ability to distinguish factors within and across systems, which is crucial to determining appropriate research or intervention targets. The definitions of each system lead to diverse interpretations. Further, each factor must be individually examined to determine the most appropriate system of influence. For instance, whether economic regulations surrounding food production are best understood within the exosystem or macrosystem depends greatly upon interpretations of the definition of the macrosystem and the particular regulation. Therefore, the model may not be able to directly inform intervention or research. Regardless, Bronfenbrenner’s seminal work (37) has prompted further evaluations socio-ecological factors influencing health risk, including the proposed project.

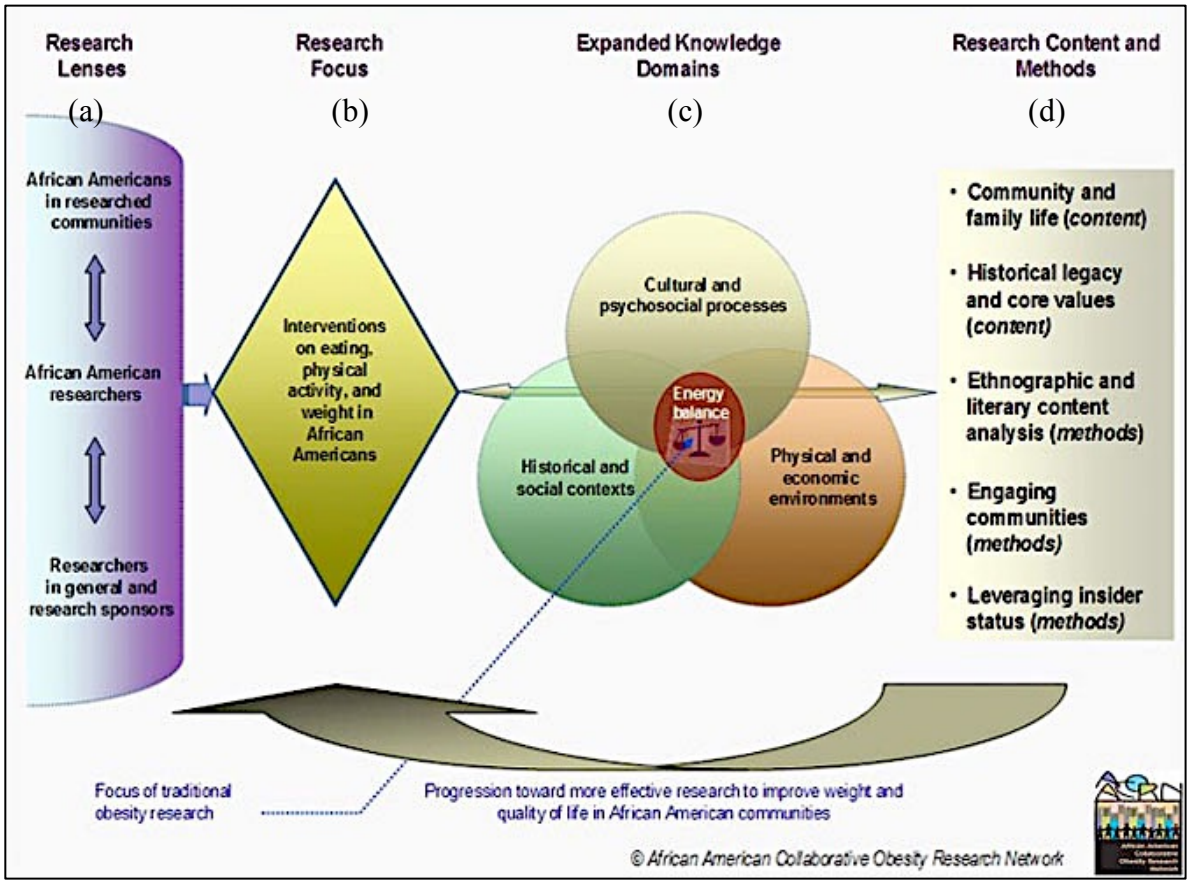
Modern Developments of Socio-Ecological Models of Obesity Risk

Since the development of Bronfenbrenner’s socio-ecological model (37), researchers have developed similar models to more precisely capture risk factors of

obesity and excessive weight gain in both adults and youth (128; 164; 248; 253; 290). Two models will be highlighted that will serve as the foundation of the proposed project: Kumanyika and colleagues' (164) African American Collaborative Outreach Network's (AACORN) obesity research paradigm and Swinburn and colleagues' (253) framework to describe determinants of and solutions for obesity. Both of these models were created to guide conceptualizations of obesity and obesity intervention endeavors from a socio-ecological perspective.

The AACORN paradigm was developed to illuminate areas of research focus within the African American community that may best capture the African American human experience and related obesity risk (164). For the purpose of orientation to the figure, a description is included: In the AACORN paradigm (164), presented in **Figure 1**, authors highlight research lenses (Figure 1a), research focus (Figure 1b), expanded knowledge domains (Figure 1c), and research content and methods (Figure 1d) most suitable for research endeavors. Within the research lenses column (Figure 1a), Kumanyika and colleagues underscore collaborations among African American individuals within

Figure 1. African American Collaborative Obesity Research Network (AACORN) obesity research paradigm (164).



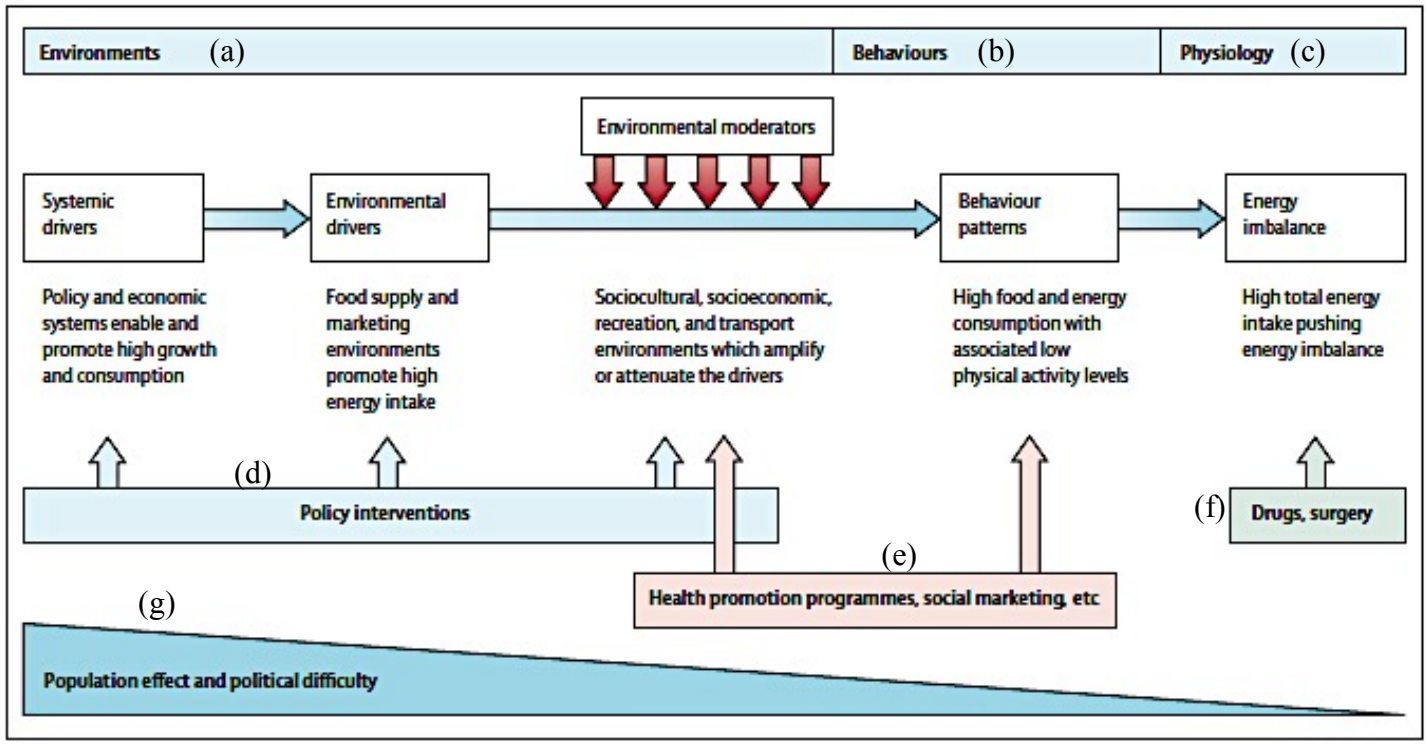
the communities being investigated, African American researchers, and general researchers to accomplish common weight-related research focus. This research focus—interventions targeting eating, physical activity, and weight (Figure 1b)—is based upon an expanded paradigm that not only includes energy expenditure, but cultural and psychosocial processes, historical and social contexts, and physical and economic environments that may promote or create barriers for weight loss (Figure 1c). In turn, these domains may clarify appropriate content to investigate (community life, family life, historical legacy, and core values), in addition to appropriate research methods (ethnographic and literary content analysis, engaging communities and leveraging insider status; Figure 1d) (164).

Unlike other models, Kumanyika and colleagues incorporate the historical context, specifically the existence of slavery that has likely shaped cultural perspectives regarding obesity and obesity-related behaviors. Authors highlight the core values of trust and distrust that have been transmitted through generations of individuals that may affect attempts to alter obesity-related behaviors. Authors also underscore the influence of collective trauma, also referred to as minority stress (205), on obesity-related behaviors. According to researchers (164), long-term exposure to sociocultural and socioeconomic distress among Africans Americans may lead to generational experiences similar to post-traumatic stress, precipitating maladaptive coping strategies, like overeating. In this way, Kumanyika and colleagues (164) provide a framework that captures the historical influences on the existence of African American individuals, particularly as it relates to the development of obesity. While not specifically highlighted in this model, others have

also pointed to specific obesity-related behaviors in the form of cultural food preferences that may also be linked to slavery (4).

In another model, Swinburn and colleagues (253) highlight potential determinants of obesity across several domains, which is presented in **Figure 2.** For the purpose of orientation to the figure, a description is included: The model depicts obesity "drivers" that span the environmental (Figure 2a), behavioral (Figure 2b), and physiological domains (Figure 2c). Within the environmental context (Figure 2a), systemic drivers (e.g., policy and economic infrastructures) coupled with environmental consequences (e.g., increases in the food supply and proliferation of food marketing strategies) collectively influence obesity-related behaviors. The behavioral domain (Figure 2b) includes food consumption, subsequently precipitating the physiological imbalance of energy that leads to obesity (Figure 2c). The model is used to highlight various policy (Figure 2d), health programming (Figure 2e), and medical interventions (Figure 2f) that coincide with the environmental, behavioral, and physiological domains, respectively. The model also highlights the population effect, as well as the political difficulty, that decreases across the domains (Figure 2g).

Figure 2. Proposed obesity research schematic depicting obesity determinants and drivers spanning the environmental, behavioral, and physiological domains.

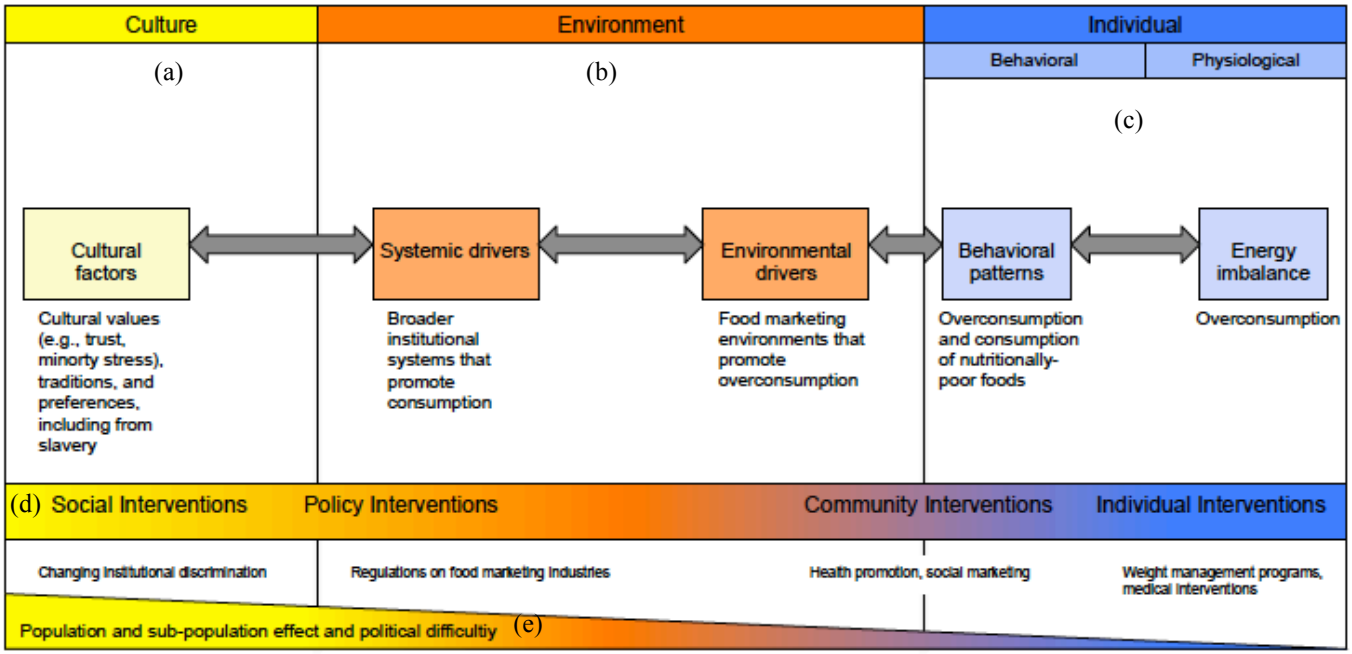


One notable strength of this paradigm is its inclusion of broader systemic factors, such as food marketing, that influences obesity risk. More specifically, the authors review systemic alterations that have coincided with the substantial increase in global obesity rates over the last several decades (253). Swinburn and colleagues note that elements of cultural traditions or food preferences (4; 164) have remained relatively static over time, and thus, are unlikely *primary* drivers of the recent obesity epidemic (253). However, other drivers, like food marketing, have become increasingly influential and ubiquitous in ways that coincide with increases in obesity rates. Thus, food marketing and other systemic drivers may more sufficiently explain the rise in obesity rates (97; 200).

This proposed project will utilize a framework based upon paradigms by Kumanyika and colleagues (164) and Swinburn and colleagues (253) to guide conceptualization of obesity risk among African American adolescent girls. This framework will also guide investigations within the proposed project. The framework, presented in **Figure 3**, highlights three primary domains of influence on obesity risk: culture (Figure 1a), environment (Figure 1b), and individual (Figure 1c), which include both behavioral and physiological mechanisms. Cultural factors encompass the broader historical influences of slavery on cultural values, like trust, distrust, collective trauma/minority stress as described by Kumanyika and colleagues (164), in addition to food practices and body shape/weight. Within the environmental domains, system-level drivers refer to broader institutional systems (e.g, policy, economic) that promote overconsumption of energy. Additionally, environmental-level drivers refer to elements of the environment that promote overconsumption of consumption of foods of poor nutritional value. Behavioral patterns include the patterns of overconsumption or

consumption of foods of poor nutritional value. Lastly, the physiological domain includes energy imbalance that occurs at the metabolic level. Notably, in line with the Bronfenbrenner's original model (37), influence of each of these factors on subsequent domains is bi-directional. Appropriate interventions aimed at targeting the aforementioned factors coincide with the domain (Figure 1d). Social interventions (e.g., changing institutional discrimination) are the primary methods required to precipitate social change, although some policy interventions (e.g., regulations marketing industries) may effectively address both cultural and environmental changes. Based upon the particular interventions, community interventions (e.g., health promotion, social marketing) may be effective tools to intervene at both the environmental and individual levels. Lastly, individual interventions are most well suited for altering behavioral and physiological factors. Notably, as described by Swinburn and colleagues (253), the potential effect on populations, as well as sub-populations, and similarly, the difficulty within the political realms decreases across domains (Figure 1e). The impact of the cultural, environmental, and individual domains in this particular group is described in more detail below.

Figure 3. Proposed framework to conceptualize obesity risk in African American adolescent girls.



Socio-Ecological Factors of Unhealthy Eating in the Proposed Study

Culture

The first element of the adapted framework highlights the role of culture in the development of behaviors, including obesity-related behaviors (164). The below review will describe the potential role of cultural food practices on obesity risk in this population.

African Americans and Food

In addition to examples provided by Kumanyika and colleagues (164), a core part of African American culture and tradition has been passed down through the preparation of foods (130). Food culture, also referred to as foodways, has been described as the means of expressing cultural or subcultural heritage through the identification of specific foods, preparation of foods, and eating patterns (292). Foodways not only assist with cultural expression, but also serve as ways to recount important events in African American history, such as the enslavement of Africans in America (292). While variability exists, historical accounts suggest that slavery has significantly influenced contemporary African American cuisine, also referred to as "soul food" (130; 292). One way slavery likely influenced food practices is through choices of specific foods that retained connections to African heritage. For instance, slaves' use of roots (e.g., okra roots) and seeds (e.g., watermelon, sesame) assisted in preserving connections to countries of origins and have remained essential items of the soul food repertoire (130; 292). Other ways slavery impacted food cultures are through the limitations on African slaves' ability to obtain certain foods and their inaccessibility to appropriate preparation

methods and proper food storage (68; 292). Reports suggest that slaves were often given foods slave masters and their families discarded, such as chitterlings, pigs' feet, pigs' ears, and giblets (4; 68). Thus, spices, seasonings, or fillings (e.g., salt, sugar, fat) were used to improved the palatability of such foods (68). Similarly, since food storage facilities were rarely available, salt was often used for food preservation or to conceal the taste of spoiled foods (68). The inaccessibility to food storage and desire to avoid wasting foods also prompted food sharing among slaves, which became another important component within African American food culture (130). It is also suggested that slavery impacted eating patterns (130). According to one account, the deprived state of slaves prompted the use of consuming food as a means of escaping oppression, depression, and distress (130). Taken together, cultural food preferences and practices shaped by slavery may partially explain unhealthy eating patterns and current barriers for healthy eating among African American individuals (4; 68; 130; 200; 292), including adolescent girls.

While younger generations may not recognize the larger historical context of eating practices, these practices may influence the familial environment surrounding food and eating (3; 4). African American adolescents' consumption of foods high in fat, sugar, and sodium African American individuals is supported by data evaluating dietary practices among youth (13; 137; 240). Further, qualitative reports provide support for the transmission of food as a means of cultural and social connection among African American families (3; 4). In a seminal qualitative report by Airhihenbuwa and colleagues (4) examining cultural food patterns among African American adults and youth, participants described eating as a means of intimacy with close family and friends and highlighted protectiveness surrounding the preparation and eating process as a way to

protect the African American food culture. In another qualitative study among African American families to examine the intergenerational transmission of foods values, Ahye and colleagues (3) investigated the meaning grandmothers, mothers, and daughters ascribed to food practices. Notable themes included food as a way to care for others and a means of social connectivity. Taken together, food traditions transmitted from slaves might influence food and eating among African American adolescent girls (164). According to Williams and colleagues (292), food marketing may target individuals by appealing to these food preferences.

Role of Epigenetics

Although a history of enslavement likely plays a key role in culturally based eating patterns, such influences may have also impacted the ways African American individuals respond to food and nutrients at the genomic level. This area of *nutritional epigenetics* refers to the genetic process of developing unique gene expressions that result from external mechanisms *independent* of one's original DNA sequence (250; 251). One external mechanism that may be responsible for modifications to gene expression is related to the gene-environment interaction (204; 250). That is, one's exposure to certain foods and nutrients can lead to DNA mutations, modifications to the ways certain nutrients are absorbed or utilized within the body, changes to gene expression, or even reprogramming of entire genomes, which can be passed down to offspring. For instance, research has demonstrated that adaptations to one's genes can occur as early as *in utero* (a process known as *metabolic imprinting* or *metabolic programming*). Such a process is purported to increase the survivability of the embryo, but can also place infants at increased risk of developing some chronic diseases in adulthood (283).

The ways in which the process of enslavement has led to changes in DNA among people of African descent through the process of epigenetics is both complex and controversial (171). One of the most notable theories regarding African enslavement and epigenetics is referred to as the *slavery hypertension hypothesis* (24; 64). Researchers hypothesized that an innate ability to retain salt was genetically selected in African-born natives due to the extremely high temperatures and limited salt and water supplies in the West African climate. It was purported that while this genetic predisposition towards salt retention was adaptive in that climate, it became maladaptive once enslaved and exposed to milder temperatures *and* increased access to high salt foods. This theory later evolved to suggest that mortality during the migration selected African individuals who could best conserve salt (64). These theories have been heavily scrutinized over the last several decades as researchers have challenged the accuracy of the historical account of West African climates and studied salt retention among West Africans currently living in West African countries (171). Authors have also noted the genotypic and phenotypic heterogeneity of individuals of African heritage (171). Other researchers have chosen to focus on the ways in which the *trauma* and *stress* of enslavement and racism has the potential to change gene expression and increase risk of developing chronic illnesses, such as hypertension (136). Although data are evolving, research in this area provides support for gene-environment interactions that may elucidate both eating patterns and chronic disease risk among African American individuals.

Environment

In the adapted framework, food marketing is highlighted as one environmental component that may promote obesity or create barriers to maintaining healthy weight,

particularly within African American individuals. The below review will describe the potential role of food marketing on obesity risk in this population.

The Current Food Environment

The current "obesogenic" food environment refers to a food environment that is replete with opportunities for overconsumption or consumption of foods of poor nutritional value (38), both of which may increase risk of obesity (125; 253). Researchers have investigated alterations in the environment that have coincided with the substantial increase in global obesity rates over the last several decades (253). Among several important drivers, Swinburn and colleagues (253) identified increasingly influential and ubiquitous food marketing strategies as a primary driver of the increased obesity rates (109; 156; 200). A large proportion of food marketing strategies promote overconsumption and consumption of nutritionally poor foods (38; 217; 218). This influence may be exacerbated by other cultural factors, including food preferences and food culture (164; 253). Thus, food marketing may play a key role in promoting obesity, particularly in vulnerable groups (105; 106).

Purpose and Procedure of General Marketing

Before discussing the role of food marketing in promoting overconsumption and consumption of nutritionally poor foods, it is important to review the purpose and function of the general marketing of commercial products. Marketing refers to the process of communicating the value of a particular product to encourage voluntary purchase by consumers (160). Marketers attempt to determine the best ways to advertise a product to meets the demands of particular markets (160). The various concepts involved in determining these *demands* and *markets* are described below.

The foundation of marketing is based upon the concepts of human needs, wants, and demands (160). Within the marketing industry, human *needs* often guide desires to pursue necessities, such as food and shelter (160). These needs transform to *wants* once individuals identify how their preferences and desires will be met. For instance, a person may *need* food, but the type of food or place to receive that food is based upon a *want*. Once individuals have a desire to purchase items that fulfill their wants, these wants become *demands*. Manufactures, in turn, seek to meet the demands that appear in the marketplace—the place in which products, goods, and services are exchanged for profit (160).

After manufactures identify marketplace demands, an important next step is to identify the particular *market*—all current and potential buyers of a particular product—to serve. Due to the realization that one company cannot satisfy all wants, needs, and demands of the entire market, the majority of marketing is targeted towards specific groups (160; 163). Thus, the market is divided into types of customers, known as *marketing segmentation*. Afterwards, *target marketing* ensues to determine the best segment to pursue. Target markets may be chosen based upon geographic location, sex/gender, age-and-life cycle, or race/ethnicity (160).

Following the identification of the targeted groups, an integrated *marketing mix* is developed that is most suited for the target market (274). A marketing plan includes a mix of strategies related to *promotion*, *product*, *price*, and, *place*. Although promotion, also known as advertising, is the most recognized form of marketing, the goods and services being offered (product), the avenues where the product will be available (place), and the dollar amount required to obtain the product (price) are all critical components of

a marketing mix. This proposal will focus on promotion as a marketing strategy, specifically the promotion of nutritionally-poor foods (274). The identification of youth and racial/ethnic minorities as target markets of food marketing will also be reviewed.

Food Marketing

Food advertising refers to the process of communicating the value of particular food products to encourage voluntary purchase by consumers (160; 163; 291). In 2012, fast food restaurants spent an estimated $4.6 billion on several marketing platforms to promote food products, including television (TV), social media, radio, print, news media, in-store marketing/promoting, premiums, and in-school marketing (116; 216). McDonald's ® is the largest spender of food advertising accounting for nearly one-fourth of the total yearly expenditure of all food advertisements (116). While various forms of marketing are increasing in popularity (e.g., social marketing), TV remains the most common form of marketing accounting for 35% of all advertising platforms and almost 90% of all monetary expenditures of food advertising (113; 216). Thus, TV advertising remains the primary means of marketing food products (216).

Food Marketing to Youth and Racial/Ethnic Minorities

Companies spend a total of $13.4 billion annually on food marketing across all platforms, and $2 billion on advertisements targeted towards youth (216; 219). A total of $333 million is spent each year advertising foods and beverages to African American youth. Between 2013 and 2017, companies increased their expenditures by more than 50% ($217 million in 2013). Food marketing towards youth includes a combination of persuasive strategies (33; 106) incorporated into food positioning and packaging, as well as digital and TV advertisements (96; 115; 178; 245; 272; 273; 285). Food marketing

attempts to engage youth by incorporating celebrities (36), cartoons (77; 115), games (245), or music (105) into advertisements. Advertisements targeted toward racial/ethnic minorities often utilize culturally salient cues that appeal to cultural food preferences and practices, such as African American actors or activities with a high percentage of African American participants (e.g., basketball; 105; 106; 292).

According to the 2019 report from the University of Connecticut Rudd Center for Food Policy and Obesity, African American youth are exposed to approximately 17 TV advertisements per day totaling approximately 6,000 TV food advertisements annually (113). Compared to their White counterparts, African American children and adolescents were exposed to 86% and 119% more advertisements, respectively, (118; 121; 165; 219). Increased exposure in African American youth is partially related to high media consumption in this group (113; 134; 217). Other key contributing factors are the placement of food-related advertisements on African American-centered programming (113; 203) and in communities populated by primarily African American individuals compared to White individuals (219).

Such exposure may be harmful since over 90% of food advertisements promote foods that are high in fat, sugar, and sodium (218), which misaligns with nutritional recommendations (270). Furthermore, content analyses of the nutrient composition of advertised foods indicate that advertisements targeting African American communities include more foods high in fat and sugar compared to those appearing in White communities (121; 203; 218). According to reports by the Nielsen group, one of the leading investigators of consumer behavior, African American adolescents view more advertisements from Popeye's ® (African American: White ratio, 1.81), Domino's ®

(1.78), Wendy's ® (1.75), and Burger King ® (1.75) compared to their White counterparts (as referenced by 116). Researchers have purported that such advertisements are designed to appeal to African American food preferences (116; 292). Examining food marketing as an environmental risk factor for unhealthy eating in African American adolescent girls is necessary to elucidate determinants of obesity risk and identify appropriate targets for prevention.

African American Adolescents as Highly Vulnerable to Food Marketing

African American youth may be highly vulnerable to targeted food marketing (105; 292). Prior research (105) provides the below definition of consumer vulnerability (p. 350):

1) "Particular susceptibility to harm based on product use (e.g., given a disproportionate burden of product-related disease *and*
2) Perceived susceptibility to the marketing techniques (e.g., inability distinguish advertising from the entertainment in which it is imbedded."

Based upon these criteria, African American adolescent girls may be more vulnerable to the effects of food marketing due to 1) the high prevalence of overweight and obesity demonstrated in this group and 2) their cognitive and identity development during adolescence.

First, overweight and obesity prevalence rates are high in African American adolescent girls (199; 200). They may also be at increased risk of obesity that tracks into adulthood (93) and of obesity-related comorbidities (19). Thus, promotion of nutritionally poor foods through food marketing may be particularly more harmful compared to other

groups (105; 116; 174). Secondly, African American adolescent girls may be particularly vulnerable to the negative effects of food marketing as their higher-order cognitive functioning and identities are still developing (208). Under-developed executive functioning often leads to a lack of impulse control and an inability to delay gratification. Therefore, adolescents may have a limited capacity to combat the powerful psychological techniques marketing uses to promote unhealthy food products (112; 114). Further, adolescents often look for social cues to help shape their developing identities (208). As described by Pechmann and colleagues (208), advertisements typically use "consumption symbols," the purchasing of products for image attributions or value rather than functional attributes, designed to highlight "psychosocial benefit" or "image benefit" of products by implying that consumption will improve social connection, mood, or attractiveness. During the adolescent period, youth may be more likely to look to such consumption symbols to help identity and clarify their identity and role, making them more likely to use consumer symbols to guide their behavior (54; 208). This process may be particularly salient among African American youth as they may be more likely to utilize the media to assist in understanding the definition of ethnic group membership and the accepted group behaviors, thereby reinforcing ethnic identity development (76). Taken together, reports suggest that African American individuals may be more exposed to marketing of nutritionally poor foods and may be more susceptible to its effects on unhealthy eating and subsequent obesity risk.

Food Marketing, Unhealthy Eating Behaviors, and Obesity Risk

Food marketing has been shown to influence unhealthy eating behaviors among youth. Thus far, studies examining the impact of direct food marketing on eating

behaviors have primarily been conducted in children (e.g., 5 – 11 years of age), but fewer in adolescents (12 – 17 years of age). In meta-analyses examining the effect of exposure to food marketing (including advergames) and dietary intake, authors reported that you exposed to food marketing consumed an average of 50-60 kcal more than youth not exposed to food marketing. Additionally, youth with obesity consumed an average of 125.5 kcal more than non-overweight youth (232). Effect sizes have ranged from small-to-medium to large (Cohen's d = .03 – 2.08) (34).

There have been several key studies highlighting the effect of food marketing on intake. Dovey and colleagues (72) examined the effect of TV food advertisements on eating in children 5 – 7 years in the United Kingdom (UK). Authors randomized children to view a 14-minute episode clip of the cartoon, "The Secret Show" embedded with 2 minutes of either healthy food advertisements, unhealthy food advertisements, or toy advertisements (neutral). Advertisements were matched on time rather than the number of advertisements, and thus, number of advertisements in each category varied. Children could eat *ad libitium* from an assortment of healthy (e.g., carrot sticks) and unhealthy snack foods (e.g., Cadbury's chocolate buttons) immediately following exposure to the cartoon. Authors found that children in the unhealthy food advertisement condition consumed significantly more total energy (an average of 461.2 g) compared to children in both the healthy food advertisement condition (an average of 410.8 g) and the neutral toy advertisement condition (an average of 400.3 g). In a similar study examining the effect of food advertisements on snack consumption in children 7 – 11 years, Harris and colleagues (111) randomized children to view a 14-minute TV episode clip of "Disney's Recess" embedded with either four 30-second food commercials or non-food

commercials inserted during advertisement breaks. Children could eat *ad libitum* from a bowl of cheddar cheese "gold fish" snacks while watching the episode clip. Authors found that children exposed to food advertisements consumed significantly more snacks (an average of 28.5 g) compared to children in the control condition (an average of 19.7 g). Additionally, Harris and colleagues (117) examined exposure to food advergames (internet games embedded with advertisements) on energy intake in children. Authors found that children exposed to the unhealthy food marking condition consumed significantly more nutritionally poor foods (an average of 31.9 g) compared to youth in the healthy food marketing condition and the control (an average of 20.5 g). Similar results have been found in other studies (5-7; 31; 88; 108).

In addition to consumption, food preferences have also been examined to underscore the additional impact of food marketing on food perceptions. Borzekowski and Robinson (28) examined the impact of food advertisements on food preferences in children aged 2 – 6 years old. Researchers found that children exposed to advertisements were significantly more likely to choose advertised products compared to the control condition. In another report, Borzekowski and Robinson (28) found that children preferred significantly more foods from the general food preferences questionnaire after watching the food commercial compared to the toy commercial, specifically carbohydrates and fats. Similar results have been found in other studies (32; 69; 210). Taken together, scientific evidence suggests that exposure to food marketing influence eating behaviors among children.

However, there is a paucity of data on the effect of food marketing on eating behaviors in adolescents. Among reports indicating an effect of food marketing on eating

behaviors (5; 236; 289), results have primarily relied upon self-report measures, used TV as a proxy for exposure to food marketing, or have not accounted for relevant covariates (e.g., body mass index [BMI]). Scully and colleagues (236) found that adolescents who self-reported watching the most commercial TV (more than 2 hours per day) had a significantly greater odds of requesting (adjusted OR, 95% CI, 1.48, 1.27 – 1.71) and trying advertised food products (adjusted OR, 95% CI, 1.61, 1.38 – 1.88). Significant results were also found for youth who self-reported being exposed to the most advertisement print, transport, school, and digital advertising sources in the past month. Similarly, in a sample examining the relationship between self-reported TV viewing (a proxy for food marketing) and self-reported energy intake among pre-adolescents and adolescents, Wiecha and colleagues (289) found that self-reported TV viewing correlated with daily energy consumption. However, these reports were based upon commercial TV as a proxy for food marketing and self-report measures. Commercial TV includes other images youth may view that could influence energy intake and confound results. Additionally, self-reported measures on energy intake may not accurately account for overall consumption attributed to food marketing exposure. Further, with the exception of studies evaluating self-reported intake, studies have not investigated the influence of marketing exposure on an array of foods youth typically see in their environment. To date, no study has examined the impact of food marketing in African American girls.

There have been several psychological mechanisms proposed to elucidate the effect of food marketing on attitudes and behaviors (112; 114). One potential mechanism is classical conditioning (60; 207). Classical conditioning refers to a learning process where a neutral stimulus that does not elicit a sensation and an unconditional stimulus

that naturally elicits some sensation are frequently paired, and eventually the neutral stimulus elicits the same sensation as the unconditional stimulus. These can be seen in food commercials where messaging is frequently paired with food images that elicit feelings of hunger, whereby the messages themselves eventually elicit feelings of hunger. Another proposed mechanism involves the information processing theory (176), which purports that food commercials influences behaviors in successive stages, beginning with an attending to the commercial, the comprehending the messages of the commercial, then encoding those messages, and then eventually agreeing with the message encode. The social learning theory (12) purports that social behaviors and attitudes are learned by observing like others. In food commercials, individuals observe others, such as other African American individuals, engaging in interactive and fun activities while consuming the advertised food product. Other mechanisms involve the elaboration likelihood/heuristic-systemic theory (75; 212), which theorizes that changes in one's attitude can occur either directly through rationally considering the information or indirectly through processing other factors, such as the uplifting messages. Although the proposed mechanisms have each attempted to describe the underlying attitudes and cognitive processing contributing to behaviors, they have failed to identify which components of one's cognitions are activated. Examining maladaptive cognitive processes may provide a valuable theoretical framework for investigating the association between food marketing and eating behaviors in AA adolescents girls.

Cognition: Attentional Biases as a Potential Mechanism

African American adolescents who are exposed to ubiquitous food cues in the current environment (38; 235) and have obesity (50) may be at increased risk of food-

related attentional biases (ABs), a maladaptive cognitive vulnerability toward overeating, subsequently promoting excessive weight gain (35; 50; 193; 195). ABs involve distinct, yet related processes of selective attention (59), including *attention orienting*, a component of automatic processing (193). Attention orienting refers to the ease or speed with which attention is drawn towards a particularly salient stimulus (e.g., food cue) compared to other non-salient stimuli (e.g., household items). Attention orientating may be further differentiated into two categories: *initial attention capture*, which is related to attention engagement, and *sustained attention*, which is related to attention disengagement. Initial attention capture refers to the unconscious capturing of attention that is evoked by salient stimuli (e.g., food cues). Sustained attention towards those salient stimuli occurs when the stimulus impairs the shifting of attention away from the specific cue. Both initial attention capture and sustained attention can be conceptualized as either 1) automatic, or *bottom up*, cognitive processing or 2) strategic, or *top down*, cognitive processing. Bottom up cognitive processing occurs in response to presented stimuli and ensues outside of one's awareness. Top down processing is more goal-driven and intentional. ABs are measured using several different methods, including reaction time, eye-tracking, brain activation, eye-tracking and electroencephalography (EEG) (246).

Incentive-sensitization theory has been put forth to elucidate the mechanism involved in ABs (228). This theory purports that repeated exposure of the stimulus can lead to an exacerbated reward response in vulnerable individuals. This response subsequently increases salience and establishes strong cravings for the stimulus. As the incentive salience for the food item increases, seeking out and consuming the food item

to alleviate craving becomes an important goal, overriding homeostatic feeding drives. With the increased food availability, such drives may lead to overeating in the current environment (38; 235).

While all individuals exhibit ABs towards food-related cues when hungry (50; 169; 184), individuals repeatedly exposed to food cues (228) and those with obesity (50) may be particularly vulnerable. Not only are African American youth exposed to more food marketing advertisements compared to other groups (118), they often reside in communities with a disproportionately high density of food cues (20; 219). Among African American youth with obesity (35; 193), external food cues may become highly salient, and promote overeating due to ABs (89; 193; 280).

ABs, Eating, and Weight

Literature examining the effect of ABs on eating and weight has been mixed (84). This section provides an exhaustive review of research examining the association of AB and food consumption, body weight, and weight gain among youth and a selective review among adults. Prior research has examined the effect of AB on consumption in non-clinical samples, as well as clinical samples of individuals with restrictive and binge eating/loss of control eating patterns. Few studies have examined these effects in adolescents, so evidence from both young adults and children will be reviewed.

ABs and Eating

Several studies have demonstrated that ABs significantly affect food consumption (29; 146; 153). The primary method researchers have evaluated the association between AB and eating has been through manipulating attention via computerized training programs and measure subsequent food intake. In one study using this methodology,

researchers randomized approximately 150 undergraduate women to conditions that trained them to 1) attend to chocolate or 2) avoid chocolate, then measured their intake of chocolate and blueberry muffins immediately after the training session, at 24-hour follow-up, and at a one week follow up (152). Participants who were trained to attend to chocolate over multiple sessions consumed significantly more chocolate muffins compared to the participants trained to avoid chocolate 24 hours following the training and at one-week follow up. Similar results were found in a clinical sample of youth with overweight and obesity who met criteria for eating in the absence of hunger during a feeding paradigm (29). In this study, participants were randomized to a condition where 1) attention was trained either away from food words to neutral words or 2) a condition where attention was trained toward neutral words and half of the time to food words. Authors found that youth whose attention was trained to neutral/food words consumed significantly more calories from the laboratory meal compared to those in the other condition. Similarly, Kakoschke and colleagues (146) randomized approximately 150 undergraduate women to 1) a condition where they were trained to attend to healthy foods (and avoid unhealthy foods) or 2) a condition where they were trained to attend to unhealthy foods (and avoid healthy foods). They found that participants who were trained to attend to healthy foods consumed significantly more healthy foods compared to those who were trained to attend to unhealthy foods.

However, when the same authors examined associations between AB, inhibitory control, and intake in another sample of undergraduate women, they found a significant interaction between AB and inhibitory control on food intake, although they did not find any direct effects of ABs on food intake (147). Similarly, although Folkvord and

colleagues (89) found a significant interactional effect on total intake among youth who were exposed to food advertising and demonstrated certain ABs, there was no direct effect of AB on intake. Hardman and colleagues (110) randomized 60 undergraduate students to conditions that trained them to either 1) attend to cake, 2) avoid cake, or 3) undergo no training (control). They did not find any significant effects of the training conditions on intake of cake. Mixed results may be due to the various methodologies in the measurement of ABs, as well as the utilization of control conditions (246). It is possible that different measures reveal similar, yet distinct neurological processes (122), making comparisons more challenging. Although data are mixed, these studies highlight a potential relationship between AB and food intake.

ABs and Body Weight

Data examining the associations between AB and body weight have also been mixed (71; 177). When examining ABs and body weight cross-sectionally, Yokum and colleagues (296) examined ABs in adolescent girls utilizing functional magnetic resonance imaging (fMRI). In their examination, authors found a significant correlation between BMI and activations in brain regions associated with attention. They also found that higher BMI was significantly associated with ABs (initial attention) to both "appetizing" and "non-appetizing" food cues. In an examination of treatment-seeking pre-adolescents and adolescents with obesity and matched non-overweight controls, other authors have found that youth with obesity were significantly more likely to experience interference while completing a modified Stroop task of food-related and neutral words compared to controls (35). In a study examining the relationship among ABs, weight, and eating in a sample of overweight and non-overweight children, authors found that

children with overweight were more affected by the food cue exposure compared to non-overweight children (138). However, in a study examining 60 children with and without obesity, Werthmann and colleagues (287) did not find any significant differences in ABs to food cues based upon BMI, which was similar to other findings (177).

In the one study examining the relationship between AB and weight gain longitudinally in youth, Yokum and colleagues (296) did not find that ABs predicted BMI change after one year. However, some, but not all researchers examining this relationship in adult have found significant results. In a longitudinal study examining the effect of AB (as measured by the emotional Stroop and dot probe tasks) towards unhealthy and healthy foods among 102 undergraduate students, Calitri and colleagues (45) found that ABs towards unhealthy food cues significantly predicted a 0.46 unit increase in BMI at one year follow-up. Those with ABs towards healthy foods measured on the same task showed a 0.32 decrease in BMI at 1-year follow-up. There were no significant changes in BMI when authors measured AB using the dot probe task. Kaisari and colleagues (145) investigated whether ABs predicted changes in weight gain after one year. While "bottom-up" ABs (i.e., biased attention when merely exposed to food cues) did not differ between the two groups, "top-down" ABs (i.e., biased attention when asked to remember food information while completing the task) significantly predicted a 0.26 unit increase in BMI at one-year follow up. Similar to examinations in eating, different methodologies may be contributing to different results.

Measuring the Effect of Food Marketing and Food-cue ABs on Energy Intake

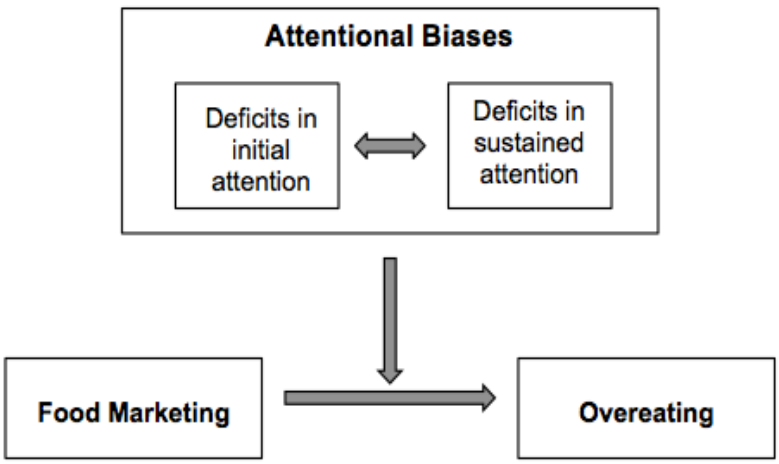
Studies elucidating the potential link between food marking and ABs on energy intake have primarily been examined in adults (147; 293). However, there has been one

with one study examining exposure to food advertisements and energy intake objectively (89), while the other (279) utilized self-report measures. In former study by Folkvord and colleagues (89), children (grades 2 through 4) were randomized to an advergame (i.e., online video game promoting particular products) embedded with either a food advertisement or a non-food advertisement. Following the advergame, children were invited to consume snack foods *ad libitum*. Using eye tracking to measure ABs, scientists measured three components of visual AB: gaze frequency, gaze duration, and the latency of initial gaze towards the cue. While not all analyses were significant, children who played the advergame promoting snack foods consumed significantly more snack foods compared to children in the control condition. Among children who played the food advergame, gaze duration was associated with significantly more intake of one of the advertised snack foods. Results remained consistent after controlling for age, sex, and hunger. Velazquez and colleagues (279) tested the associations between attention to food-related cues using eye-tracking and self-reported food preferences and choices in 102 youth 8 – 15 years old. Youth were exposed to 40 food and beverage commercials and eye-tracking examined the total time youth viewed the commercial, fixation length, and fixation count. Both fixation length and fixation counter were significantly associated with "unhealthy" food and beverage preferences. However, results did not remain significant after controlling for sex, age, and BMI*z*. Although data are mixed and covariates attenuate results, data suggest that ABs may moderate the effect of food marketing on eating behaviors. However, more research is needed to elucidate this mechanism, particularly in African American adolescent girls and using a variety of foods youth may see in the current food environment.

Summary

Obesity among African American adolescent girls is highly prevalent (199; 200). Further, they may be at a disproportionately high risk for adult obesity (93) and related comorbidities (19; 66). African American adolescent girls may also consume high amounts of fat, sugar, and sodium exacerbating their risk of excessive weight gain (13; 240). While prevention is critical, most methods have been met with limited success, which warrants more targeted approaches. Several socio-ecological factors may contribute to unhealthy eating behaviors, and subsequent obesity risk, in this group, including cultural food preferences, food marketing, and food-related ABs (164; 233; 253; 292). Indeed, African American adolescents are exposed to significantly more food advertisements that may appeal to cultural food preferences by promoting nutritionally poor foods (217). Food cue ABs may increase the likelihood of seeking and consuming foods thereby exacerbating the effect of food marketing among these vulnerable youth (89). Further, food marketing may serve as a stimulus that precipitates craving and a motivation to alleviate craving by seeking and consuming food, particularly among individuals with ABs (**Figure 4**).

Figure 4. Proposed mechanism by which attentional biases moderate the effect of food marketing on overeating.



Innovation and Policy Impact

The worsening rates of chronic diet-related illnesses calls for "strategic science" that has the capacity to fill knowledge gaps that can inform policies and have the potential to improve food environments (39). Although the food industry has made varying commitments to improving nutritional quality of foods promoted on TV and other platforms (191), the majority of foods promoted still fail to meet nutritional guidelines for healthful living (294). Research that helps uncover key questions and mechanisms that can identify levers of changes may greatly assist capacity building for regulations or other means of promoting health in the current environment (180). Ultimately, these data have the potential to build scientific support towards important regulatory policies to encourage alignment of marketing with nutritional guidelines and limits on food marketing towards youth and racial/ethnic minorities.

CURRENT STUDY

The purpose of this proposed study is to examine the effect of food marketing and ABs on eating behaviors in African American adolescent girls with and without obesity (12 – 17 years of age). This study is a between-subjects cross-sectional experimental design to examine the role of food marketing and ABs on total energy, fat, sugar, and salt intake in African American adolescent girls. Participants were randomization to one of two marketing conditions (food marketing, neutral marketing). ABs were evaluated using a visual probe task. The primary outcomes were total energy, fat, sugar, and salt consumption from a laboratory test meal.

Aims and Hypotheses

Rationale for Specific Aim 1: Evidence suggests that food advertisements may negatively impact eating behaviors in youth. According to prior data, over 90% of food advertisements targeting adolescents promote foods that are high in fat, sodium, and sugar (218). Youth-targeted food marketing has been shown to influence consumption of *both* advertised and non-advertised foods (72; 111) and food preferences (28; 32). Examining food marketing as an environmental risk factor for unhealthy eating in this group is necessary to elucidate determinants of obesity risk and identify appropriate targets for prevention.

Specific Aim 1: To investigate the effect of food marketing on energy intake.

Hypothesis 1a: Girls in the food marketing condition will consume more total energy compared to girls in the neutral marketing condition.

Hypothesis 1b: Girls with obesity who are in the food marketing condition will consume the most energy compared to all other conditions (girls with obesity in the neutral marketing condition, non-overweight girls in the food marketing condition, and non-overweight girls in the neutral marketing condition).

Rationale for Specific Aim 2: African American youth often reside in communities with a disproportionately high density of food cues (20; 219). Given the ubiquity of available foods and food cues in the current environment (38; 235), ABs to food stimuli may indicate a vulnerability towards overeating, consequently promoting obesity (35; 50; 193; 195). Some data suggests that youth with obesity may demonstrate increased difficulties in attention capture (35; 195) followed by exacerbated sustained attention following initial allocation (288). Although data are mixed (241), some research (286) has demonstrated an association between these cognitive deficits and overeating in response

to external food stimuli. Youth who exhibit ABs toward food-related stimuli may be at increased risk of overeating (89). ABs to food cues may be a potential mechanism by which food marketing impacts food consumption and increased weight in African American adolescent girls.

Specific Aim 2: To examine ABs (initial, sustained) towards food cues and energy intake.

Hypothesis 2a*:*

Hypothesis 2ai: Greater initial ABs towards food cues will be associated with more total energy consumption.

Hypothesis 2aii: Greater sustained ABs towards food cues will be associated with more total energy consumption.

Hypothesis 2b:

Hypothesis 2bi: Girls with obesity and greater initial ABs will consume the most total energy compared to all other conditions (girls with obesity and fewer initial ABs, non-overweight girls with greater initial ABs, and non-overweight girls with fewer initial ABs).

Hypothesis 2bii: Girls with obesity and greater sustained ABs will consume the most energy compared to all other conditions (girls with obesity and fewer sustained ABs, non-overweight girls with greater sustained ABs, and non-overweight girls with fewer sustained ABs).

Rationale for Specific Aim 3: Some data suggest that youth who exhibit ABs toward food-related stimuli may be at increased risk of overeating (89), particularly when exposed to food advertisements (89). In a study examining ABs among children exposed

to the food marketing condition, food-related AB was associated with significantly more snack intake (89). Such results indicate that ABs may moderate the effect of food marketing on eating behaviors.

Specific Aim 3: To examine the role of food marketing and ABs on total energy intake.

Hypothesis 3a:

Hypothesis 3ai: Among girls in the food marketing condition, greater initial ABs will be associated with higher total energy consumption compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3aii: Among girls in the food marketing condition, greater sustained ABs will be associated with higher total energy consumption compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3b:

Hypothesis 3bia: Among girls in the food marketing condition, greater initial ABs will be associated with higher consumption of foods high in fat compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3bib: Among girls in the food marketing condition, greater sustained ABs will be associated with higher consumption of foods high in fat compared to

all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3biia: Among girls in the food marketing condition, greater initial ABs will be associated with higher consumption of foods high in sugar compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3biib: Among girls in the food marketing condition, greater sustained ABs will be associated with higher consumption of foods high in sugar compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3biiia: Among girls in the food marketing condition, greater initial ABs will be associated with higher consumption of foods high in sodium compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

Hypothesis 3biiib: Among girls in the food marketing condition, greater sustained ABs will be associated with higher consumption of foods high in sodium compared to all other conditions (girls in the neutral marketing condition with greater ABs, girls in the neutral marketing condition with fewer ABs, and girls in the food marketing condition with fewer ABs).

CHAPTER 2: METHODS

STUDY DESIGN

This study is a between-subjects cross-sectional experimental design to examine the role of food marketing and ABs on eating behaviors African American adolescent girls. Participants were randomization to one of two marketing conditions (food marketing, neutral marketing). ABs were evaluated using a visual probe task. The primary outcomes were total energy (Calories), percent of fat, grams of sugar, and mg of salt from a laboratory test meal.

RECRUITMENT

Participants were recruited from Washington, DC metropolitan area. Recruitment efforts were targeted towards parents/guardians of African American girls between the ages of 12 and 17 years, and included a number of methods found to be highly effective in our prior studies:

1. Direct mailings to families with African American adolescent girls between the ages of 12 and 17 years with contact information and study flyer.
2. Flyers posted at the Uniformed Services University (USU), local facilities (e.g., libraries, supermarkets), and churches with permission.
3. Adolescents enrolled in previous studies who were eligible based on their age, race/ethnicity, and sex.
4. Adolescents not eligible for previous studies who expressed their interest in participating in other protocols in the future.
5. Contacting school listservs in Washington, D.C., Maryland, and Virginia.

Advertisements targeted parents/guardians and included language indicating that only adolescent girls (12-17 y) were being recruited. Given that the effect of food

marketing is a primary outcome of the study, the primary goal of the study was concealed in accordance with prior research (e.g., 111). Participants were told that the primary aim of the study was to learn more about African American adolescent consumers' opinions about how relationships and communication styles were depicted in the TV show. Interested participants were informed that the purpose of the study was to understand how African American adolescent girls view relationships and communication depicted on TV. For parents who were interested in the study and who had questions about study participation, all recruitment materials included a study email address and a "Google Voice" telephone number for participants to call where they could reach the Principal Investigator (PI) or research team directly. Participants were debriefed immediately following the study visit. During the consent, adolescents were told that they would have a session at the conclusion of the study visit to provide their feedback about the study. During this session, study team members asked participants an open-ended question to elicit their opinions about the study (e.g., "What did you think about the study?"). The study team member then asked, "now that you have completed the study, what did you think was the purpose?" to assess whether the deception was successful or unsuccessful. The deception was deemed "unsuccessful" if participants stated that the study was related to marketing/commercials, African Americans and commercials/marketing, and/or observing eating behaviors. The deception was deemed "successful" if participants provided any other response (e.g., how relationships are depicted on TV [the advertised study purpose], adolescents and relationships, African American girls or communities in general). The study team member provided verbal and written information regarding the true nature of the study (**Appendix 13**). Given the history of mistrust within this community, particularly pertaining to medical research, study

team members were careful to provide this information sensitively. Study team members also engaged in a brief follow up discussion once participants perceived this information on deception and true purpose to assess whether they experienced any distress.

Inclusion and Exclusion Criteria

A telephone preliminary screen was conducted with potential participants to determine if they met the following inclusion criteria:

1) Female,
2) Self-identify as African American or Black,
3) 12 to 17 years of age at the start of the study,
4) Presence of non-overweight, overweight, or obesity by virtue of a BMI ≥ 5th percentile for age and sex (55), *and*
5) English-speaking.

Potential participants were excluded if they met the following criteria:

1) Self-reported presence of a major chronic medical illness: renal, hepatic, gastrointestinal, endocrinologic (e.g., Cushing syndrome, hyper- or hypothyroidism), hematological problems or pulmonary disorders (other than asthma not requiring continuous medication),
2) Self-reported major depressive disorder, psychoses, current substance or alcohol use disorder, or any other psychiatric disorder that, in the opinion of the investigators, would impede competence or compliance or possibly hinder completion of the study,
3) Self-reported current, regular use of prescription medications that affect appetite, mood, or body weight: currently prescribed serotonin re-uptake

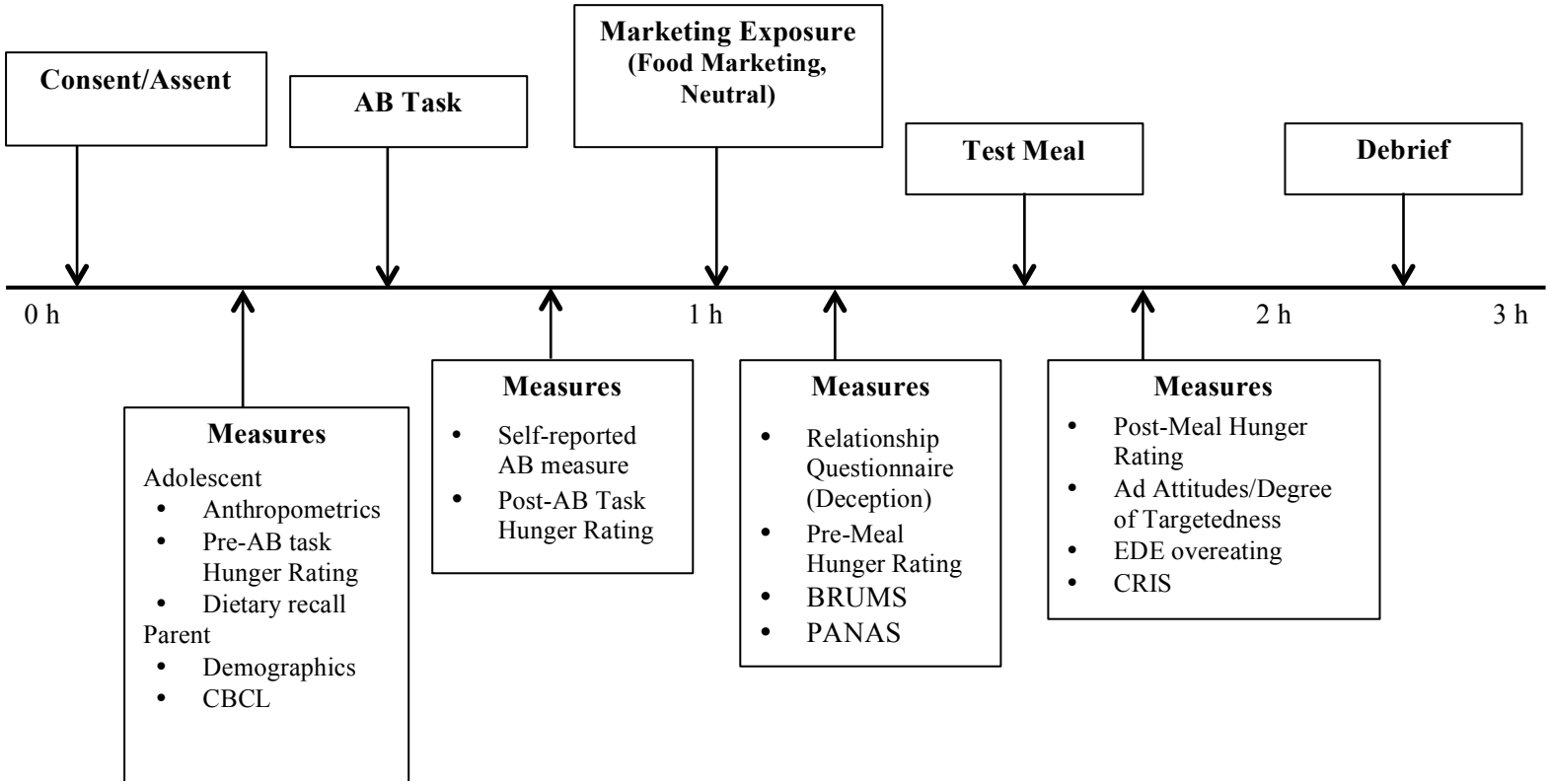
inhibitors (SSRIs), neuroleptics, tricyclics, stimulants, or any other medication known to affect appetite, mood, or body weight. Oral contraceptive use will be permitted, provided the contraceptive has been used for at least two months before the study visit, *or*

4) Failure to provide a rating of 6 or more on at least 50% of food items on the Food Preferences Questionnaire (Appendix 4);
5) Presence of food allergies or restrictions if food items could not be replaced with items of equivalent macronutrient content.

STUDY PROCEDURE

A schematic of the study timeline is included in **Figure 5**. A telephone screen was conducted with potential participants to determine preliminary eligibility. If a potential participant appeared appropriate based upon a pre-screening, she was scheduled for a one-time visit to participate in the study. Adolescents were asked to fast and not engage in any exercise two hours prior to their visit. Potentially eligible families were seen at USU.

Figure 5. Study timeline for one-time visit



Upon arrival for the appointment, the purpose, procedures, risks, and b
the study were explained to adolescents and their parent(s)/guardian(s). Potenti
participants were informed of the right to withdraw from the protocol at any p
the study. All questions were answered. Parents/guardian signed consent and a
signed prior to completing screening. Parents completed questions on adolesce
demographics (TV use, parents/caregiver occupations) and the Child Behavior
Checklist. Height, weight, and waist circumference were measured, and then p
consumed the standardize snack (granola bar, 100 kcal) followed by completin
recall. Approximately 5 minutes after consuming the snack, adolescents compl
questionnaire to assess hunger. After completing the questionnaire, adolescents
completed the visual dot-probe AB task, followed by a self-report AB measure
another hunger questionnaire. Afterwards, all participants were viewed of the
episode clip of *The Big Bang Theory* (embedded with commercials; to view ins
provided to participants, see **Appendix M**).

Following the episode, adolescents completed the questionnaire about t
relationship and communication styles of the couples. Afterwards, adolescents
another hunger questionnaire and mood ratings, and then were invited to eat *ad*
from a multi-item test meal array. They were provided up to 30 minutes to eat
ad libitum. Following the meal, adolescents completed another hunger questio
addition to questionnaires related to behavior, eating behaviors, ethnic identity
advertisement attitudes, and perceived degree of targetedness. All girls receive
form of a MasterCard© gift card for participating in the study.

TV Episode with Embedded Commercials

Selection of TV Episode

The author coordinated with grant consultant, Marie Bragg, Ph.D., to identify appropriate TV episode and advertisements based on her previous research experiences. All participants viewed a 14-minute episode clip of *The Big Bang Theory* (Season 9, Episode 19, "The Soldier Excursion Diversion"; 220). A comedy was used to remain consistent with the genre used in prior research (111). In consultation with Dr. Bragg, the *Big Bang Theory* was selected because of its documented popularity among adolescents (62). Although other shows that may resonate with African American adolescents were discussed, only anecdotal data on their popularity existed at the initiation of the project in 2016. Ways to mitigate this challenge is discussed in the Summary/Discussion. A relatively recent (versus older) episode season was chosen (Season 9, aired September 2016) to reduce the likelihood that adolescents would question the selection of the particular episode, while also minimizing the chance that adolescents had recently viewed the episode. Episode 19 was selected because of the limited references to food, eating, or restaurants and the ability to easily remove any remaining references without affecting the storyline or episode flow. Explicit language or behaviors were also removed. The digital rights management (DRM) copy protection, which is designed to prevent illegal copying of proprietary and copyrighted works, was removed using Tuneskit for Mac (Version 3.3.4) (265). Removal of DRM copy protection allowed for editing for non-commercial, research purposes (267). The episode was edited using Apple Final Cut Pro X (Version 10.3.4; Cupertino, CA).

In the episode, Leonard and Howard attend a movie premier without their wives' (Penny and Bernadette, respectively) knowledge. Leonard and Howard share their decision with their mutual friend, Raj, who discloses the truth to Penny and Bernadette. After returning from the movies, Leonard and Howard voluntarily share the truth of their outing with their wives and request forgiveness. The scenes in this storyline take place in Leonard's and Howard's research laboratory and at a movie theater. In the secondary storyline, Amy offers to purchase her boyfriend, Sheldon, a new laptop. Her request leads to a dialogue with Sheldon about his discomfort with discarding personal items. These scenes take place in Sheldon's home, car, and storage unit.

Selection of Commercials

The TV episode was embedded with four 30-second food commercials or neutral commercials at minute 1:01, 4:03, 6:56, and 10:05. Two advertisements promoted hamburgers from McDonald's and Burger King, and two advertisements promoted fried chicken from Kentucky Fried Chicken (KFC) and Popeye's. The neutral advertisements promoted cars from Ford and Honda and insurance from Esurance and Geico.

Food advertisements from four different food companies (as opposed to one food company) were included in order to examine the overall effect of food advertisements as opposed to the effect of one particular advertisement. Dr. Bragg and the author discussed the most suitable food companies based upon a study conducted in 2013 reporting that McDonald's, Burger King, Kentucky Fried Chicken (KFC), Popeye's spent the most money on marketing towards racial/ethnic minority youth (116). Identifying neutral commercials was more challenging. It is widely held that control conditions are similar to the experimental condition with the exception of the active component, which—in this

study—was promotion of a food product. This author initially proposed using advertisements related to intake or consumption, such as mouthwash commercials, because they involved a product that was consumed in some form similar to food commercials. However, using such closely aligned commercials in control conditions is not standard practice within food marketing research. Although neutral advertisements, such as games and toys, have been used in studies with children (111), choosing appropriate neutral advertisements for adolescent was more challenging. The majority of nonfood commercials targeting adolescents were related to school or school work, health promotions (e.g., anti-drug, anti-tobacco), appearance (e.g., acne, clothing), or communicating with parents while navigating hormonal changes during adolescents. Such advertisements were considered emotionally activating, and therefore, not suitable for the control condition. Thus, in conjunction with Dr. Bragg, it was determined that neutral advertisements that were unrelated to consumption or adolescents would be best suited for the project. Dr. Bragg and the author agreed upon car and insurance commercials.

Advertisements were selected from Adscope (149), a database that provides subscribers access to advertisements. The Adscope database categorizes commercials using the following criteria: name (e.g., restaurant or company), date aired, media platform (e.g., TV, radio), attributes (demographic specifications), markets (specific geographic regions), language, and length. Eight separate searches for food commercials and neutral was conducted using the following search terms: McDonald's, Burger King, KFC, Popeye's, Ford, Honda, Esurance, and Geico. Searches also included the following search terms (specific category included in parentheses): three years prior to search year

in 2016 (date aired), TV only (media), ethnic targeted/African American (attributes), Baltimore, MD/Washington, DC/Hagerstown (markets), English (language), 30 seconds (length).

The search returned 28-42 commercials for each restaurant or company. Commercials were excluded if they did not advertise the preferred items (e.g., McDonald advertising only chicken products), advertised activities that were unlikely to appeal to adolescents (e.g., advertising toys for younger children), included references to sports or celebrities, were off-season (e.g., references to winter), or promoted a corporation (e.g., Bank of America). For neutral commercials, advertisements that referenced food, eating, or restaurants were also excluded. A total of 15 McDonald's, four Burger King, 10 Popeye's, eight KFC, nine Geico, 10 Ford, eight Esurance, and seven Honda commercials remained. The remaining commercials were reviewed based upon the following categories that were identified in conjunction with Dr. Bragg based upon prior research methodology: number of people in the advertisement, the estimated time persons were on screen, approximated age of persons on screen, gender of persons on screen, estimated time the product was on screen, estimated time the product was on screen, and overall affect. The estimated time the product was on screen and overall affect were determined to be the most critical as more exposure to food products could impact eating behaviors, as well as the potential for an episode to activate different emotions, such as happiness or sadness. Various combinations of four food and four neutral commercials were compared to determine optimal matching on the aforementioned categories.

In the selected McDonald's commercial, a group of primarily African American young adults enter McDonald's to order from the new "mix and match" menu and sit

down to eat together. In the Burger King commercial, an African American man returns to Burger King after his "whopper" was swamped for a McDonald's "big mac." Before the man walks away, the Burger King mascot emerges from behind the counter and gives him the "whopper." In the Popeye's commercial, the African American female spokesperson, Annie, is displayed in the kitchen describing the new Popeye's "tear'n tenderloin chicken." She identified the way Popeye's chicken is similar to New Orleans' style Cajun chicken. In the KFC commercial, a group of predominantly African American young adults are in a recording studio with a tray of KFC "chicken littles." They continuously repeat how being "little" is the new "big."

In the Geico commercial, the Geico gecko is featured in a forest promoting their emergency roadside service. In the Esurance commercial, an elderly White man releases pigeons on a rooftop as a way to send a "tweet" (an error in his reference to the communication process of the social media platform, Twitter). Esurance is promoted as the "new" way of purchasing insurance that replace antiquated methods. In the Honda commercial, the 2016 Honda Fit LX is promoted as a way to stylishly complete errands while remaining in touch with others through the Bluetooth capabilities. In the Ford commercial, an employee is featured in the showroom to promote a sale to provide employee discounts to the general public.

MEASURES

Demographics

Demographic information (**Appendix A**), such as age and parents'/guardians' occupation (proxy for socioeconomic status [SES], hereafter referred to as SES), was collected via self-report. Due to high media use among African American youth (217),

parents also provided information on the adolescents' weekly TV use as a proxy for habitual advertisement exposure.

Anthropometrics

Body weight (2-hour fasting) was measured in triplicate to the nearest 0.1 kg using the Tanita BF 350 Body Composition Analyzer (Tanita Corporation of America, Inc., Arlington Heights, IL). Height (2 hours fasting) was measured in triplicate to the nearest 0.1 cm using a stadiometer. Fasting fat mass and fat-free mass was measured using the COSMED BOD POD® (Life Measurement Inc., Concord, CA), which utilizes air displacement plethysmography. Details of the start date of their last menstrual cycle was also collected to determine menstrual phase. For measure, see **Appendix B**.

Hunger

Participants completed rating scales for hunger, fullness, and food craving on a visual analogue scale ranging from "Not at all" to "Extremely" (scale 0 to 100; 244; 257). Hunger rating were examined pre- and post-AB task, in addition to pre- and post-meal (**Appendix C**).

Food-related ABs

To examine ABs, all participants completed a visual dot probe task (**Appendix N**), which has been widely used as a behavioral measure of food cue incentive salience in overweight samples (50; 170; 195; 288). The computer task consisted of 180 trials. Participants viewed pairs of images on a computer screen for 500 ms or 2000 ms. Image types included 90 images from one of three categories: high palatable foods (HF) (i.e., high-energy dense foods such as pizza and donuts), low palatable foods (LF) (i.e., low-

energy dense foods such as mushrooms and pineapples), and neutral non-food (NF) household items (e.g., clock) as the control stimuli. Energy density was used as a proxy for food palatability, which refers to the pleasantness or liking of foods based upon factors such as taste, texture, and smell (133; 243). Energy dense foods are typically more palatable, but less satiating compared to other foods (25), may uniquely promote overeating and excessive weight gain (73).

Combinations of image pairs included HF-NF, LF-NF, and HF-LF. Dot-probe paradigms assessed ABs by presenting stimulus pairs followed by a probe that required a response. Trials consisted of a mixture of incongruent trials (when the probe replaces the neutral image) and congruent trials (when the probe replaces the HP food image). Sixty pairs of each combination (HF-NF, LF-NF, HF-LP) were presented in randomized order. The spatial location of images and probes were counter-balanced. After the image pair disappeared, a central fixation cross of variable duration (500 ms, 1000 ms, or 1500 ms) served as the inter-trial interval. Youth were instructed to respond with a right or left sided key-press to indicate the direction of the arrow. Two time periods were isolated: initial attention (500 ms after stimulus presentation) and sustained attention (2000 ms after stimulus presentation). For a visual depiction of the task, see Appendix 12. Internal split-half reliability was excellent for mean reaction times (*r*s = .84 - .92), but poor for bias scores (*r*s = -.30 - .40, Mean = .05).

Food Preferences

The food preferences questionnaire (FPQ; **Appendix D**) contained the foods that would comprise the buffet used for the laboratory test meal. These food items were embedded within a larger, general food preference list. The questionnaire evaluates

whether foods used in the test meal array would be acceptable to the participant and has been shown to successfully identify food preferences in children (182). Participants completed a rating scale for foods on a visual analogue scale ranging from 1 ("I hate this food") to 10 ("I love this food"). To qualify for the study, participants had to provide a rating of 6 or more for at least 60% of the food items.

Observed Energy Intake

Following exposure to the advertisement, participants were given instructions to eat *ad libitum* from a multi-array test meal (approximately 4,400 kcal; Fat: 37%, Sugar: 88 g, Salt: 1050 mg; **Figure 6**). Meal times were standardized across four time periods based on the start of the visit: 10:30am, 12:30pm, 2:30pm, and 4:30pm. The test meal included chicken nuggets, white bread, Oreos ©, M&Ms ©, barbecue sauce, peanut butter, jelly, water, apple juice, and 2% fat-free milk. The meal paradigm was based upon a modified meal utilized by Lauren B. Shomaker, Ph.D., Associate Professor at Colorado State University. The test meal did not include foods marketed during the advertisements as intake following exposure to foods advertisements is not necessarily related to advertised food items (111). Consumption was calculated by weighing each item pre- and post-meal. Energy content and nutrition composition was determined by manufacturers. Participants consumed meals at approximately 10:30am, 12:30pm, 2:30pm, and 4:30pm. Participants who consumed meals within approximately 30 minutes of the expected time were added to that time category (e.g., participants who began eating at 10:50am were coded within the 10:30am category). Participants outside of these ranges were placed into separate categories.

Figure 6. Laboratory test meal (approximately 4,400 kcal)

Disordered Eating Behaviors

Disinhibited eating was measured to determine presence and severity of overeating and eating behaviors that may impact energy intake. The Eating Disorder Examination (EDE) Overeating Section, Version 12 (with text edits from versions 14 and 15; 80; 81) is a semi-structured psychodiagnostic interview of eating disorder psychopathology (**Appendix E**). The overeating section was administered to determine the presence and severity of disordered eating behaviors. The EDE is reliable and valid in adolescent samples (103; 258; 276).

Mood

Mood may contribute to eating behaviors. The Positive and Negative Affect Scale (PANAS; 284) and the Brunel Mood Scale (BRUMS) were used to measure state mood (263; 284). The PANAS is 20-item a measurement of positive (e.g., interested, excited)

and negative (e.g., scared, guilty) mood states identified on a 5-point Likert-type scale ranging from 1 ("Very Slightly or Not at all") to 5 ("Extremely"; **Appendix F**). The PANAS has been validated in African American adults (181). The BRUMS is a 16-item measurement of various negative mood items (e.g., angry, sad). Participants indicate to what extent they feel a variety of different mood descriptors (e.g., sad, happy) "right now" (**Appendix G**). The mood descriptors in the BRUMS are rated on a 5-point Likert scales ranging from "Not at all" to "Extremely." The BRUMS is a reliable and valid measure among diverse youth (119; 263; 297).

Parent Reported Child Behavior

The Children's Behavior Check List (CBCL; 2) is the most commonly used measure assessing various internalizing (e.g., anxious, depressive) and externalizing (e.g., aggressive, hyperactive) childhood behaviors (**Appendix H**). Parents were asked to complete the CBCL for their child. Responses were scored with fill-in-the-black and on a 3-point Likert-type scale ranging from 0 ("Not true") to 3 ("Very true or Often true"). The CBCL is reliable and valid in pediatric samples (8; 61; 202; 247). While the CBCL has not been validated in African American youth, CBCL has been used among various racial/ethnic minority samples (18; 67; 231).

Ethnic Identity

Food advertisements embed cultural relevance by utilizing cultural food preferences.(105; 292). Ethnic identity was used as a proxy for identifying individuals who may be more susceptible to such practices. The Cross Racial Identity Scale (CRIS; 275) is a commonly questionnaire used to measure ethnic identity (**Appendix I**). The CRIS is a 40-item measure examining ethnic exploration, ethnic belonging, cultural

knowledge, and ethnic attachment using a 7-point Likert-type scale ranging from 1 ("Strongly disagree") to 7 ("Strongly agree"). The CRIS includes 6 subscales: pre-encounter assimilation (pro-American identity; items 2, 9, 18, 26, 34), pre-encounter miseducation (negative stereotypical views about African Americans), pre-encounter self-hatred (anti-Black, self-hating identity), immersion-emersion anti-white (dislike and distrust of White individuals), internalization afrocentricity (strong belief in Black empowerment and success based on the work of African Americans), and internalization multiculturalist exclusive (Black self-acceptance and acceptance of other cultural groups). The two subscales that indicated strong identification with African American culture—internalization afrocentricity and internalization multiculturalist exclusive—were used in the analyses.

The CRIS has demonstrated excellent reliability (Cronbach's αs = .65 – .88), construct validity (Cronbach's αs = .71 – .87), and convergent validity (Cronbach's αs = .72 – .89) among African American adolescents and young adults (101; 275). In the current sample, pre-encounter assimilation (Cronbach's α = .80), pre-encounter miseducation (Cronbach's α = .87), pre-encounter self-hatred (Cronbach's α = .91), immersion-emersion anti-white (Cronbach's α = .79), internalization afrocentricity (Cronbach's α = .78), and internalization multiculturalist exclusive (Cronbach's α = .75) all demonstrated excellent reliability.

Advertisement Attitudes and Degree of Targetedness

Data suggests that African American youth view advertisements more favorably compared to other groups (44; 104), potentially influencing future purchase. Product attitudes towards each advertisement was measured via a 7-point Likert scale ranging

from 1 (“Very bad/Very unfavorable”) to 5 (“Very good/Very favorable”; **Appendix J**). Further, data indicates that marketers may appeal to cultural food preferences in order to target racial/ethnic minorities (292). The extent to which adolescents felt targeted by advertisements will be measured using three questions regarding advertisement intent, belief that advertisement was created for them, and belief that advertisements was designed to appeal to people like them. Items will be measured using a 7-point Likert scale ranging from 1 (“Disagree completely”) to 7 (“Agree completely”). The measures have been widely used in prior marketing studies (1; 140; 141). Participants completed questions related to advertisement they viewed in their respective conditions.

Perception of Relationships Depicted on the TV Episode

As part of the study deception, adolescents were asked to complete a relationship questionnaire that assessed the relationship and communication styles depicted on the TV episode (**Appendix K).** Adolescents were asked to rate the relationship between members of the TV show on a scale from 1 (“Very good”) to 5 (“Terrible”). They were also asked to determine which communication styles were utilized on the TV show: Using “I feel” statements, putting yourself in another person’s shoes, being willing to compromise, or picking the right time to have a difficult conversation.

DATA ANALYTIC PLAN

A Priori Sample Size Estimation

A sample size of approximately 140 was the *a priori* estimation based upon recommended equations for estimating sample size in some prior studies, expected attrition, as well as *a priori* power analyses. Sample sizes ranged from N = 92 - 118 (Cohen’s d = .35 - .96; Cohen’s f^2 = .06 - .15) in food marketing studies (89; 111); N = 36

- 76 (partial eta-squared = .01 - .054; some effect sizes not reported) in visual probe task studies (46; 237); N = 60 - 177 (eta-squared = .06 - .11; R2 = .04 - .25; some effect sizes not reported) in pediatric laboratory test meal studies (123; 182; 257). *A priori* power analyses were conducted with G*Power, Version 3.1.9.2 (82) with a significance threshold of $\alpha = 0.05$. Separate power analyses were conducted for all planned statistical analyses to determine which analysis required the highest sample size.

For an ANCOVA with two groups, one degree of freedom, and six covariates (Hypothesis 1a), analyses indicated that a minimum sample size of 128 would provide 80% power to detect a medium effect (Cohen's $f = 0.25$). For an ANCOVA with four groups, one degree of freedom, and six covariates (Hypothesis 1b), analyses indicated that a minimum sample size of 128 would provide 80% power to detect a medium effect (Cohen's $f = 0.25$). For multiple linear regressions with 7 predictor variables (Hypotheses 2a and 2b), analyses indicated that a minimum sample size of 103 would provide 80% power to detect a medium effect (Cohen's $f^2 = .15$). For multiple linear regressions with 9 predictor variables (Hypotheses 3a and 3b), analyses indicated that a minimum sample size of 114 would provide 80% power to detect a medium effect (Cohen's $f^2 = 0.15$). Based upon these analyses, a minimum sample size of 128 was indicated. This author planned to recruit a total of 140 to account for potential attrition. Comparison to current sample size is provided in the Discussion.

Statistical Analyses

All analyses were conducted using SPSS, Versions 24 and 25. Data were examined for outliers and screened for normality. To minimize influences on analyses, extreme outliers were recoded to 1.5 times the interquartile range below the 25th

percentile or above the 75th percentile (17). Total energy referred to intake of total Calories (kcal). Fat intake was calculated based upon the percentage of fat consumed; sugar intake was calculated based upon total grams of sugar consumed; and salt intake was based upon total milligrams consumed. Percentage of fat consumed during the test meal was arcsin transformed to satisfy normality assumptions of the analyses. To improve interpretation of fat intake, results were back transformed where possible to assist interpretation. Age, SES, height, fat mass, fat-free mass, mood, menstrual phase, daily intake, craving, TV use, and meal time were considered as covariates, but were not statistically associated with the outcome variables and were not included in the models. Although pre-meal hunger was statistically associated with intake of fat, sugar, and salt, adjusted models did not differ from adjusted models or contribute to statistical interpretation. Thus, pre-meal hunger was not included as a covariate. Differences in demographic variables, overall meal consumption, and hunger ratings were examined using repeated measures analyses of variance (ANOVAs), Pearson's correlations, and chi square tests where appropriate. Participants were excluded from analyses due to the deception being unsuccessful, high error rates completing AB task/lost AB data, overweight status, where appropriate. Differences were considered significant at $p \leq .05$. All tests were two-tailed.

Preparing AB Data

Based upon prior literature, trials were excluded if participants responded too quickly (<200 ms) or too slowly (>2000 ms). Trials were also excluded if participants responded incorrectly. Participants with error rates of 10% or above were excluded from the analyses (9). AB in initial attention capture were calculated for each of the three pair

types (HF-NF, HF-LF, LF-NF) based upon reaction times within trials where probe appears 500 ms after stimulus presentation. AB in sustained attention capture was calculated for each of the three pair types (HF-NF, HF-LF, LF-NF) based upon reaction times within trials where probe appears 2000 ms after stimulus presentation. The reaction time to images determined to be most salient were subtracted from the reaction time to the images determined to be least salient, which resulted in bias scores. Faster reaction times suggest a greater bias towards the stimuli. Negative AB scores will suggest a bias *away* from the salient cue, and positive AB scores will indicate a bias *towards* the salient cue. Therefore, negative initial and sustained AB scores for HP-NF pairings indicate a bias away from high palatable foods, and positive indicate a bias towards high palatable foods. Negative initial and sustained AB scores for LP-NF pairings indicate a bias away from low palatable foods, and positive scores indicate a bias towards low palatable foods. Negative initial and sustained AB scores for HP-LP pairings indicated a bias away from high palatable foods, and positive scores indicate a bias towards high palatable foods. Mean and median reaction times were calculated. To reduce the effect of outliers, median reaction times were used in the analyses. The visual probe was coded using E-prime 2.0.

Specific Aim 1: Food Marketing and Weight on Total Energy Intake

Hypothesis 1a

General linear models were used to test the hypothesis that exposure to food marketing influences total energy intake. One-way analyses of variance (ANOVAs) were used with marketing (food, neutral) entered as the between-subjects independent variable (IV), and Calories as the dependent variables. *Post hoc* analyses used ANOVAs examined the effect of the IV on fat, sugar, and salt intake.

Hypothesis 1b

General linear models were used to test the hypothesis that adolescents with obesity who are also exposed to food marketing would consume to most total energy. Factorial ANOVAs were used with marketing (food, neutral) and weight status (obese, non-overweight) as the between-subjects independent variables, and Calories as the dependent variables. *Post hoc* analyses used multiple hierarchal regressions to examine the effect of marketing and weight entered as continuous variables. Weight (BMI*z*, fat mass, fat-free mass, or waist circumference) was entered as a predictor in the first step in the model, marketing (food, neutral) was entered as a predictor in the second step, and the interaction term (marketing X BMI*z*, marketing X fat mass, marketing X fat-free mass, or marketing X waist circumference) was entered as the predictor in the third step of the model. The outcome variables were intake of Calories, fat, sugar, and salt. Additional *post hoc* analyses used factorial ANOVAs to examine to examine the effect of marketing (food, neutral) and ethnic identity (internalization afrocentricity, CRIS-IA or internalization multiculturalist exclusive, CRIS-IMCE) on intake of Calories, fat, sugar, and salt intake.

Specific Aim 2

In order to examine both *initial* and *sustained* AB towards food cues, two trial periods were examined: trials where the probe appears 500 ms after stimulus presentation (initial) and trials where the probe appears 2000 ms after stimulus (sustained).

Hypothesis 2a

Simple linear regressions were to test the hypothesis that individual with food-related ABs would consume the most total energy. Initial and sustained ABs (HF-LF

bias, HF-NF bias, LF-NF bias) were entered as predictors, and the outcome variables was Calories.

Hypothesis 2b

Multiple hierarchal regressions were used to test the hypothesis that adolescents with obesity and food-related AB would consume the most total energy. Weight status (obese, non-overweight) was entered as a predictor in the first step in the model, AB (initial HF-NF bias, initial HF-LF bias, initial LF-NF bias, sustained HF-NF, sustained HF-LF, sustained LF-NF) was entered as a predictor in the second step, and the interaction term (initial HF-LF bias X weight, initial HF-NF bias X weight, initial LF-NF bias X weight, sustained HF-LF bias X weight, sustained HF-NF bias X weight, or sustained LF-NF bias X weight) was entered as the predictor in the third step of the model. The outcome variable was Calories. *Post hoc* analyses used multiple hierarchal regressions to examine AB for highly palatable foods and weight on fat, sugar, and salt intake. Weight (BMI*z*) was entered as a predictor in the first step, initial or sustained ABs for highly palatable foods (initial HF-NF, initial HF-LF, sustained HF-NF, or sustained HF-LF) was entered as a predictor in the second model, and the interaction term (initial HF-NF X BMI*z*, initial HF-LF X BMI*z*, sustained HF-NF X BMI*z*, or sustained HF-LF X BMI*z*) was entered as a predictor in the third step of the model. The outcome variables were fat, sugar, and salt intake.

Specific Aim 3

Hypothesis 3a-3b

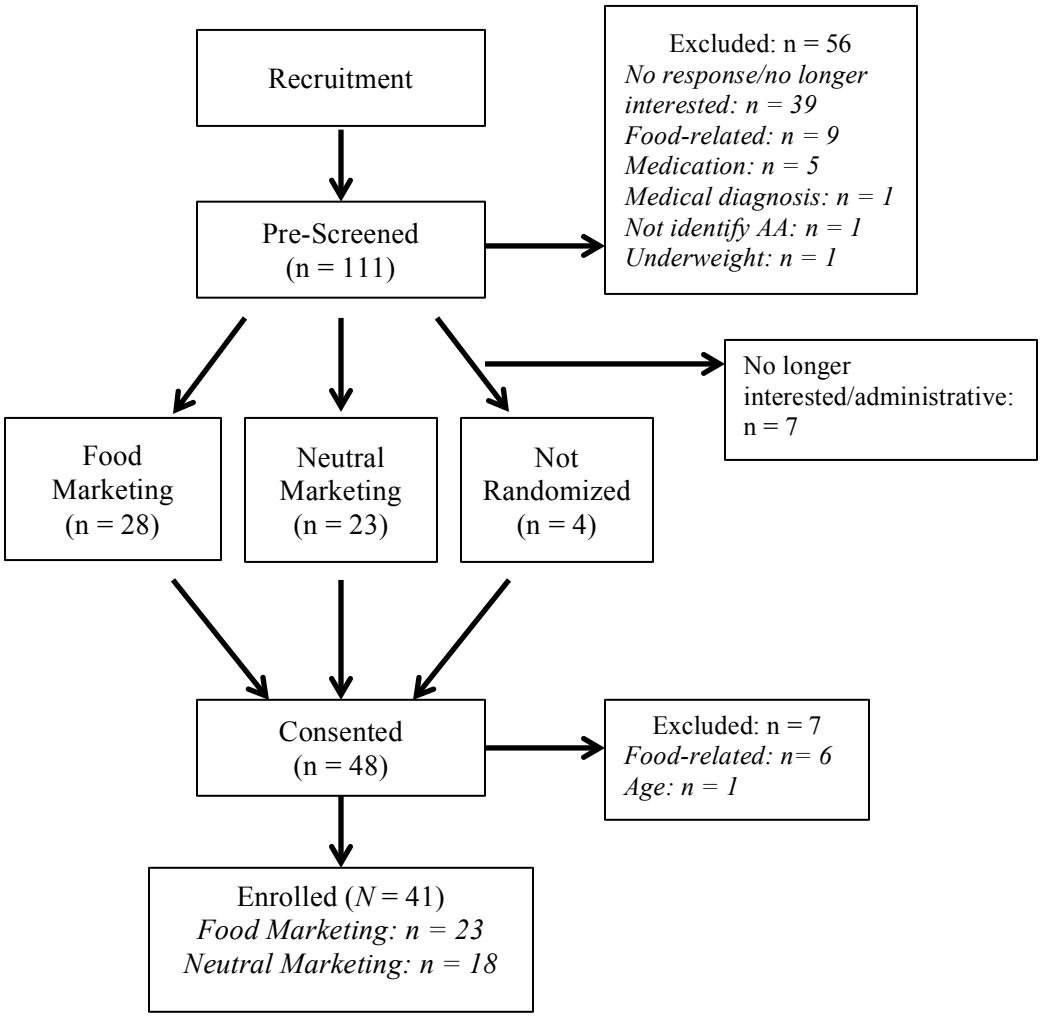
Multiple hierarchal regressions were used to test the hypothesis that ABs moderate the relationship between food marketing and energy intake. AB (initial HF-NF

bias, initial HF-LF bias, initial LF-NF bias, sustained HF-NF, sustained HF-LF, or sustained LF-NF) was entered as a predictor in the first step of the model, marketing condition (food, neutral) was entered as a predictor in the second step, and the interaction term (marketing X initial HF-NF bias, marketing X initial HF-LF bias, marketing X initial LF-NF, marketing X sustained HF-NF bias, marketing X sustained HF-LF bias, or marketing X sustained LF-NF) was entered as the predictor in the third step of the model. The outcome variables was intake of Calories, fat, sugar, and salt.

CHAPTER 3: RESULTS

A total of 111 adolescents were pre-screened to participate in the study (Mailings: 89%, Other studies: 9%, Word of mouth: 3%). Fifty-six adolescents were excluded because they did not meet inclusion criteria during the pre-screen. Fifty-five adolescents were invited to participate in the study. Of these, 14 were excluded because they did not meet inclusion criteria after completing the screening process during the study visit, were no longer interested in participating, or were not scheduled due to administrative decisions. For the complete study flow, see **Figure 7**.

Figure 7. Study flow



SAMPLE CHARACTERISTICS

Total sample was 41 African American females between the ages of 12 and 17 years (M, SD = 14.93, 1.67). BMI*z* ranged from -1.01 to 2.62 (M, SD = 0.95, 1.00), body fat mass ranged from 1.51 to 52.83 kg (M, SD = 20.25, 14.07), fat-free mass ranged from 51.50 to 96.90 kg (M, SD = 73.70, 11.20), and waist circumference ranged from 49.75 to 124.40 cm (M, SD = 88.70, 18.50). Based upon estimated calculations of menstruation, 44.10% of the sample was in the follicular phase, 5.90% was in the ovulation phase, and 41.20% was in the luteal phase of her menstrual cycle. Per parent/guardian report, weekly TV use ranged from 0 to 31 hr (M, SD = 11.54, 7.93). A total of 18 participants (45%; *N* = 40, one excluded due to missing data) reported having at least one loss of control eating (LOC) episode in their lifetime. A total of 11 participants (26.83%; *N* = 41) reported having at least one LOC episode in the past three months. Based on parent report on the Child Behavior Checklist (CBCL), two participants scored in the clinical range for depression, and one scored in the clinical range for oppositional defiant problems. The mood descriptors on the Brunel Mood Scale (BRUMS) were rated on Likert scales ranging from "Not at all" to "Extremely" (scores 0 to 5). Average scores were between 0 and 1 for the anger, tension, and depression subscales. Based upon parents'/guardians' occupations, estimations of household income ranged from $27,950 to $206,760, with eight participants having a household income between $27,950 and $53,910 (25%); eight participants between $61,530 and $95,270 (25%); and 16 participants between $96,950 and $206,760 (50%). The median income was $96,110. There were no differences based

upon condition for any of these variables (see **Table 2**). Information regarding excluded participants can be found in **Appendix Q**.

Table 2. Sample characteristics based upon condition.

	n	Food Marketing (M, SD)	Neutral Marketing (M, SD)	*p*
Age (y)	41	14.65 (1.78)	15.28 (1.49)	.24
BMI*z*	41	1.00 (1.12)	0.88 (0.84)	.71
Body fat mass (%)	38	26.19 (11.55)	26.46 (11.08)	.94
Fat-free mass (%)	38	73.81 (11.55)	73.55 (11.10)	.94
Waist circumference (cm)	39	87.62 (20.74)	89.97 (15.98)	.70
Weekly TV use (hr)	39	11.70 (7.89)	11.28 (8.24)	.88
Estimated Household Income (dollars)	32	102595.56 (55526.65)	94585.71 (31407.18)	.63
			Count (%)	
LOC Eating (ever)	40	8 (20.00)	10 (25.00)	.34
Menstruation Phase: Follicular*	18	10 (56.56)	8 (44.44)	--
Menstruation Phase: Ovulation*	2	2 (100.00)	0 (0.00)	--
Menstruation Phase: Luteal*	17	9 (52.94)	8 (47.06)	--

**N* = 37 for all menstruation data combined. Chi-square analyses could not be conducted because two cells had counts less than 5.

Meal Consumption

Total energy is measured in total Calories (also referred to as "kilocalories"; 1 Calorie = 1 kilocalorie). Fat intake was calculated based upon the percentage of fat consumed (hereafter denoted "fat"); sugar intake was calculated based upon total grams of sugar consumed (hereafter denoted "sugar"); and salt intake was based upon total milligrams consumed (hereafter denoted "salt").

Meal times occurred at 10:30am, 12:30pm, 2:30pm, and 4:30pm. Four participants consumed meals at approximately 10:30am (12.1%), 13 consumed meals at approximately 12:30pm (39.4%), five consumed meals at approximately 2:30pm (15.2%), and seven consumed meals at approximately 4:30pm (21.2%). There were four participants who consumed meals outside of the ranges due to delays to starting the visit at the expected time (12.2%). Two additional meals categories were added to account for these participants: 'between 12:30pm/2:30pm' (included 2 participants) and 'between 2:30pm/4:30pm' (included 2 participants).

Participants consumed an average of 1083.65 kcal during the meal in an average of 17 min (**Table 3**). On average, participants consumed approximately 41.19% fat, 58.06 g of sugar, and 1444.52 mg of salt. Average self-reported daily intake was 363.40 kcal. Meal time did not significantly impact total intake of Calories ($F(5, 27) = 1.21, p = .33$), fat ($F(5, 27) = 0.95, p = .46$), sugar ($F(5, 27) = 1.01, p = .43$), or salt ($F(5, 27) = 2.17, p = .09$).

Table 3. Meal consumption

	n	Minimum	Maximum	Mean	Standard Deviation
Total Calories (kcal)	33	477.30	1730.60	1083.65	351.84
Fat (%)	33	30.20	53.74	41.19	5.58
Sugar (g)	33	11.27	96.85	58.06	20.39
Salt (mg)	33	939.20	1932.72	1444.52	268.71
Meal duration (min)	33	8	27	17.00	5.60
Daily Intake	33	100.00	870.00	363.40	258.40

Ratings of Hunger and Craving

A range of 36-41 participants were included in the analyses (excluded due to lost data; see **Appendix Q**). Participants rated their hunger and craving on a VAS ranging from "Not at all" (0) to "Extremely" (100). Participants were 'a little' to 'somewhat' hungry prior to completing the AB task (M, SD = 38.72, 27.79) and hungrier following the AB task (M, SD = 45.00, 29.83; **Table 4**). Participants were 'somewhat' hungry prior to completing the test meal (M, SD = 50.79, 29.10) and not very hungry following the test meal (M, SD = 2.73, 8.93). There were significant differences in hunger at the various time points (*corrected* $F(2.24, 73.99) = 48.94$, $p = .000$). Pairwise comparisons revealed that participants were least hungry after the test meal compared to before the AB

task (p = .000), after the AB task (p = .000), and before the test meal (p = .000). Pre-meal hunger was significantly associated with intake of fat (r = -.42, p = .01), sugar (r = .39, p = .02), and salt (r = -.35, p = .03), but was not associated with intake of total Calories (r = .16 p = .34). Although pre-meal hunger was statistically associated with intake of fat, sugar, and salt, adjusted models did not differ from adjusted models or contribute to statistical interpretation so only adjusted models are reported.

Participants rated their craving between 'a little' to 'somewhat' prior to completing the AB task (M, SD = 31.14, 29.18), following the AB task (M, SD = 31.40, 24.69), prior to completing the test meal (M, SD = 33.34, 26.58), and following the test meal (M, SD = 3.53, 6.54; **Table 4**). There were significant differences in craving at the various time points (*corrected* $F(2.40, 69.54) = 18.63$, p = .000). Pairwise comparisons revealed that participants craved food the least after the test meal compared to before the AB task (p = .000), after the AB task (p = .000), and before the test meal (p = .000). Craving at the various time points was not significantly associated with intake (ps = .06 - .43).

Table 4. VAS hunger and craving ratings

	n	Minimum	Maximum	Mean	Standard Deviation
Pre-AB hunger	39	0	100	38.72	27.79
Post-AB hunger	41	0	100	45.00	29.83
Pre-Meal hunger	39	0	100	50.79	29.10
Post-Meal hunger	37	0	51	2.73	8.93
Pre-AB craving	37	0	100	31.14	29.18
Post-AB craving	40	0	100	31.40	24.69
Pre-Meal craving	38	0	100	33.34	26.58
Post-Meal craving	36	0	25	3.53	6.54

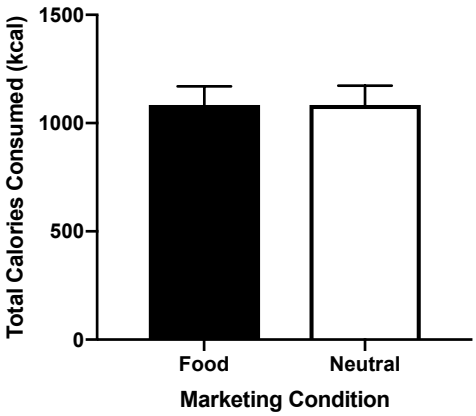
Note: AB = attentional bias. VAS hunger ratings from 0 to 100.

RESULTS OF SPECIFIC AIM 1: FOOD MARKETING AND WEIGHT ON TOTAL ENERGY INTAKE

Specific Aim 1a: Food marketing on total energy intake

A total of 33 participants were included in specific aim 1a and 1b analyses (6 were excluded for unsuccessful deception, 2 for missing meal duration data or meal duration greater than 30 minutes, and 2 for overweight status; 1 excluded participant was in both deception and meal duration categories and 1 excluded participant was in both deception and overweight status categories; **Appendix Q**). *A priori* analyses were conducted to examine the effect of food marketing on Caloric intake. There were no significant differences between adolescents in the food marketing condition compared to those in the neutral marketing condition on total Calories consumed (1084.04 kcal versus 1083.28 kcal, $F(1, 31) = 0.00, p > .99, \eta^2 = .00$; **Figure 8**).

Figure 8. Differences between food and neutral marketing condition on total Caloric intake (kcal).

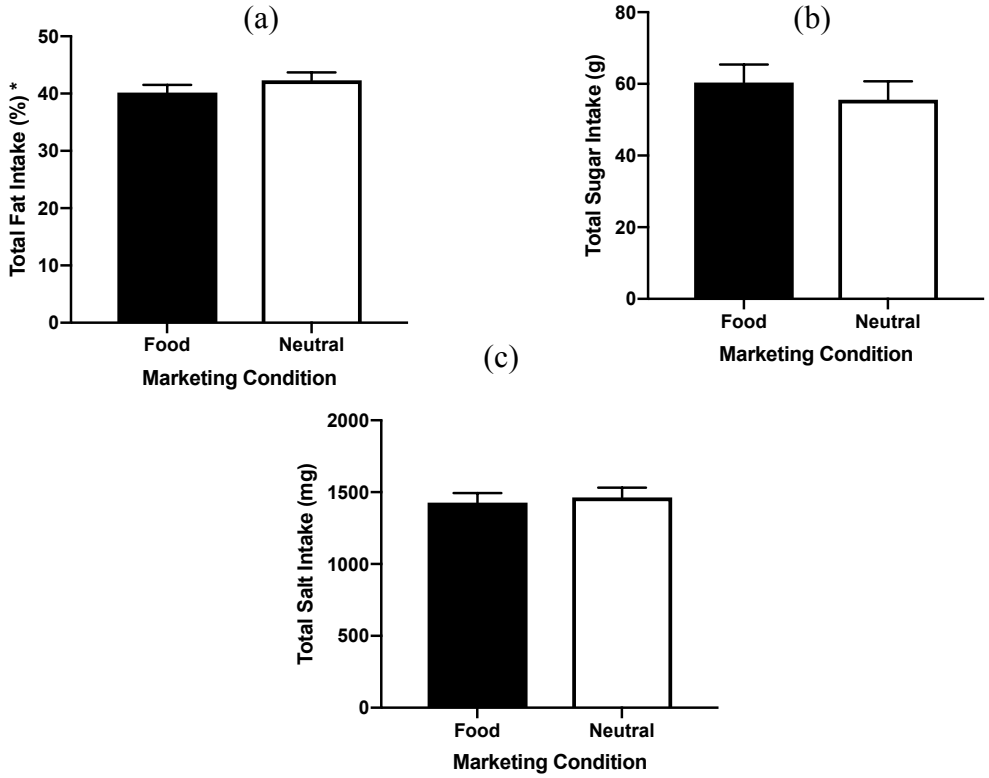


Exploratory post hoc analyses: Marketing condition on fat, sugar, and salt intake[2]

Independent *post hoc* analyses were conducted to examine the effect of food marketing on intake of fat (**Figure 9a**), sugar (**Figure 9b**), and salt (**Figure 9c**). There were no significant differences between adolescents in the food marketing condition compared to those in the neutral marketing condition on consumption of fat (40.17% versus 42.28%[3], $F(1, 31) = 1.16$, $p = 0.29$, $\eta^2 = .04$, sugar (60.39 g versus 55.58 g, $F(1, 31) = 0.45$, $p = 0.51$, $\eta^2 = .01$), or salt (1427.06 mg versus 1463.07 mg, $F(1, 31) = 0.14$, $p = .71$, $\eta^2 = .01$).

[2] Pre-meal hunger was considered as a covariate for analyses examining fat, sugar, and salt, but adjusted models did not contribute to statistical interpretation or change results.
[3] Back transformed to assist interpretation

Figure 9. *Post hoc* analyses of differences between food and neutral marketing condition on (a) fat, (b) sugar, and (c) salt consumed. *Back transformed to assist interpretation



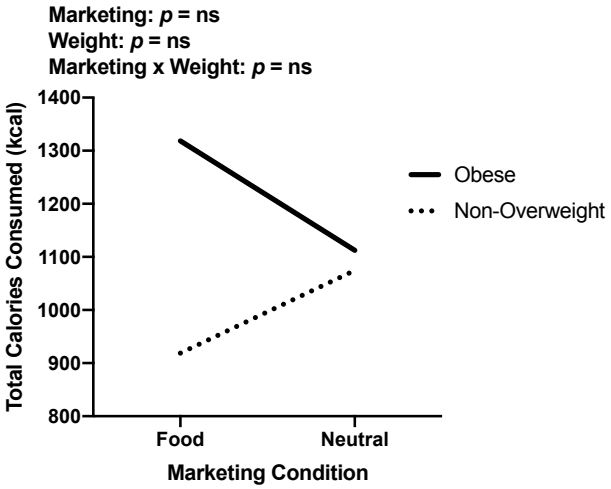
Specific Aim 1b: Food Marketing and Weight on Energy Intake

Total Food Intake by Marketing Condition and Weight Status

There were also no significant main effects of marketing condition ($F(1, 29) = 0.04, p = 0.85, \eta^2 = .00$) or weight status ($F(1, 29) = 2.92, p = 0.10, \eta^2 = .09$) on total Calories consumed (**Figure 10**). Given the small sample size, the effect size was examined for this comparison. Adolescents with obesity consumed 239.75 kcal more than non-overweight adolescents. There was a small-to-medium effect size ($\eta^2 = .09$) for this difference. There was not a significant marketing condition by weight status interaction on total Calories consumed ($F(1, 29) = 2.00, p = 0.17, \eta^2 = .06$). Similarly, given the small sample size, the effect size was examined for this comparison. Compared to

adolescents with obesity who were in the neutral marketing condition who consumed 37.94 kcal more than non-overweight adolescents, adolescents with obesity who were in the food marketing condition consumed 399.58 kcal more than non-overweight adolescents. There was a small-to-medium effect size ($\eta^2 = .06$) for this difference.

Figure 10. Difference in consumption of Calories among obese and non-overweight adolescents with and without obesity in the food and neutral marketing conditions.



Exploratory Post Hoc Analyses

Examining weight as a continuous variable

A total of 30 participants were included in exploratory analyses examining weight continuously (6 were excluded for unsuccessful deception, 2 for missing meal duration data or meal duration greater than 30 minutes, 2 for overweight status, 3 for missing Bod pod data [fat mass/fat-free mass], and 2 for missing waist circumference; 1 excluded participant was in both deception and meal duration categories, 1 excluded participant was in both deception and overweight status categories, and 2 excluded participants were in the Bod pod and waist circumference categories; **Appendix Q**). Independent *post hoc*

analyses were conducted to examine the associations among marketing condition and intake of total Calories, weight, fat, sugar, and salt.[4] Weight was analyzed as a continuous variable using BMI*z*, fat mass, fat-free mass, and waist circumference. To improve readability, only *p*-values are reported for non-significant models. For the complete models, see **Tables P1 – P16** in **Appendix P**.

There were no significant effects when examining the associations among marketing condition and intake of total Calories, fat, sugar, and salt, and weight as a continuous variable. There was not a significant marketing by BMI*z* interaction on intake of total Calories ($p = .22$; **Table P1**), fat ($p = .76$; **Table P5**), sugar ($p = .27$; **Table P6**), or salt ($p = .16$; **Table P7**). There was not a significant marketing condition by fat mass interaction on total Calories ($p = .27$; **Table P2**), fat ($p = .46$; **Table P8**), sugar ($p = .44$; **Table P9**), or salt ($p = .64$; **Table P10**). There was not a significant marketing condition by fat-free mass interaction on intake of total Calories ($p = .34$; **Table P3**), fat ($p = .45$; **Table P11**), sugar ($p = .71$; **Table P12**), or salt ($p = .40$; **Table P13**). There was not a significant marketing condition by waist circumference interaction on intake of total Calories ($p = .39$; **Table P4**), fat ($p = .71$; **Table P14**), sugar ($p = .22$; **Table P15**), or salt ($p = .75$; **Table P16**).

Examining the impact of ethnic identity on energy intake

A total of 33 participants were included in exploratory analyses examining ethnic identity (6 were excluded for unsuccessful deception, 2 for missing meal duration data or meal duration greater than 30 minutes, and 2 for overweight status; 1 excluded participant

[4] Pre-meal hunger was considered as a covariate for analyses examining fat, sugar, and salt, but adjusted models did not contribute to statistical interpretation or change results.

was in both deception and meal duration categories and 1 excluded participant was in both deception and overweight status categories; **Appendix Q**). Additional exploratory analyses were conducted to examine the impact of marketing condition and ethnic identity (internalization afrocentricity, CRIS-IA; internalization multiculturalist exclusive, CRIS-IMCE) on total energy, fat, sugar, and salt (see **Tables P17 – P24** in **Appendix P**). There were no significant effects when examining the impact of marketing condition and ethnic identity. There was not a significant marketing condition by CRIS-IA interaction on intake of total Calories (p = .39; **Table P17**), fat (p = .76; **Table P18**), sugar (p = .95; **Table P19**), or salt (p = .71; **Table P20**). There was not a significant marketing condition by CRIS-IMCE interaction on intake of total Calories (p = .64; **Table P21**), fat (p = .99; **Table P22**), sugar (p = .56; **Table P23**), or salt (p = .93; **Table P24**).

RESULTS OF SPECIFIC AIM 2: ATTENTIONAL BIASES AND WEIGHT ON TOTAL INTAKE

Specific Aim 2a: Attentional Biases on Total Energy Intake

All 37 participants with valid AB data (4 missing due to lost data) were included in preliminary analyses to prepare AB data. Trials with reactions times outside of the 200 – 2000 ms range were excluded (0.92% of the trials excluded). Trials were also excluded if participants responded incorrectly (7.69% of the trials excluded). There were no significant differences between marketing condition and excluded trials based on timing ($t(35) = -1.26$, $p = .22$) or accuracy ($t(35) = -1.29$, $p = .21$).

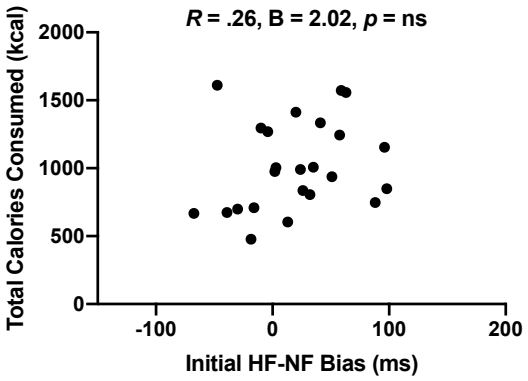
Biases in Initial Attention and Energy Intake

A total of 24 participants were included in the remaining specific aims 2a, 2b, and exploratory analyses (6 were excluded for unsuccessful deception, 2 for missing meal

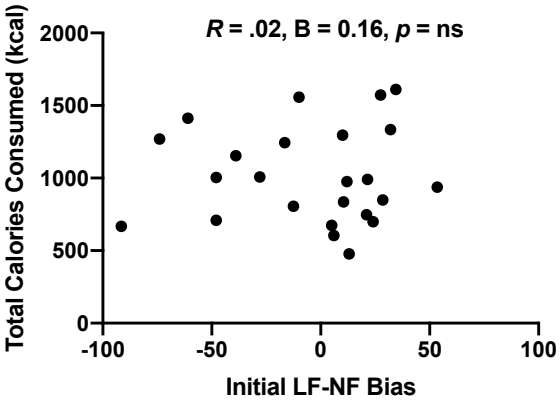
duration data or meal duration greater than 30 minutes, 2 for overweight status, 4 for lost AB data, and 7 for dot probe error rates at or above 10%; 1 excluded participant was in the deception and meal duration categories; 1 excluded participant was in the deception, overweight status, and lost AB data categories; and 1 excluded participant was in the deception and lost AB data categories; **Appendix Q**). Specific aim 2a was to examine the impact of initial and sustained HF-NF, LF-NF, and HF-LF biases on total energy intake. Neither initial HF-NF bias (R = .26, B = 2.02, p = .22; **Figure 11a**), initial LF-NF bias (R = .02, B = 0.16, p = .93; **Figure 11b**), nor initial HF-LF bias (R = .15, B = -1.03, p = .47; **Figure 11c**) statistically predicted total Caloric intake.

Figure 11. Initial biases on total Caloric intake.

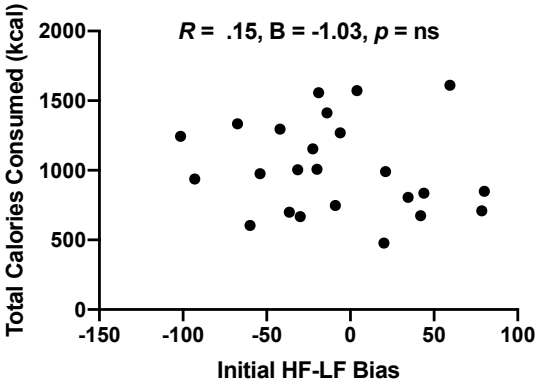
(a) Initial HF-NF bias and Calories



(b) Initial LF-NF and Calories



(c) Initial HF-LF bias and Calories

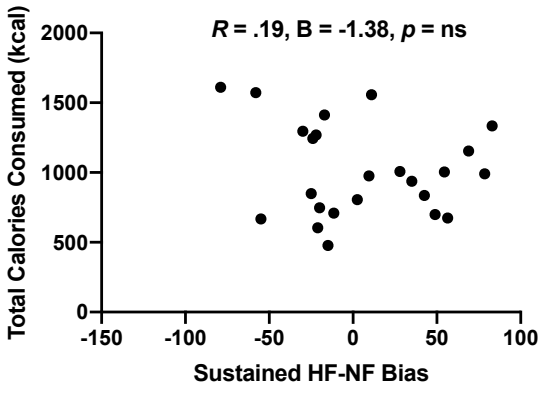


Biases in Sustained Attention and Energy Intake

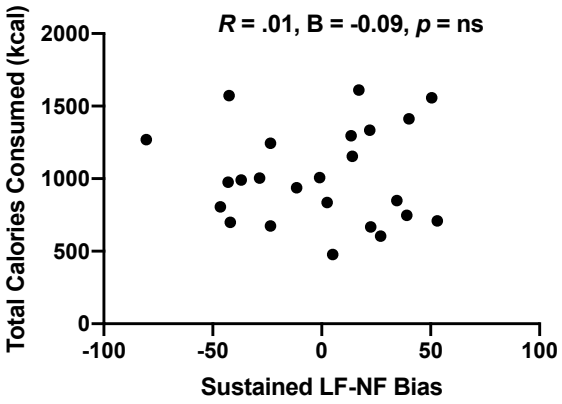
Neither sustained HF-NF (R = .19, B = -1.38, p = .38; **Figure 12a**), sustained LF-NF bias (R = .01, B = -0.09, p = .96; **Figure 12b**), nor sustained HF-LF bias (R = .03, B = 0.18, p = .91; **Figure 12c**) statistically predicted total Caloric intake.

Figure 12. Sustained biases on total Caloric intake.

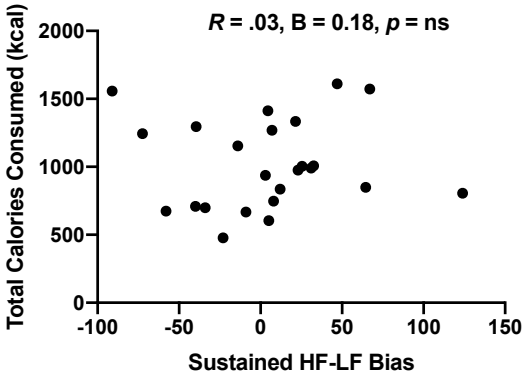
(a) Sustained HF-NF and Calories



(b) Sustained LF-NF bias and Calories



(c) Sustained HF-LF bias and Calories



Specific Aim 2b: Attentional Biases, Weight, and Total Energy Intake

Biases in Initial Attention, Weight, and Energy Intake

Specific aim 2bi was to examine the impact of initial HF-NF, LF-NF, and HF-LF biases and BMI*z* on total energy intake. BMI*z* did not significantly moderate the relationship between initial HF-NF bias and total Calories (Change in $R^2 = .03$, $F(1, 20) = 0.73$, $p = .40$; **Table 5**), initial LF-NF bias and total Calories (Change in $R^2 = .01$, $F(1, 20) = 0.25$, $p = .62$; **Table 6**), nor the initial HF-LF bias and total Calories (Change in $R^2 = .06$, $F(1, 20) = 1.44$, $p = .25$; **Table 7**).

Biases in Sustained Attention, Weight, and Energy Intake

Specific aim 2bii was to examine the impact of sustained HF-NF, LF-NF, and HF-LF biases and weight status on total energy intake. BMI*z* did not significantly moderate the relationship between sustained HF-NF bias and total Calories (Change in $R^2 = .04$, $F(1, 20) = 0.94$, $p = .34$; **Table 8**), sustained LF-NF bias and total Calories (Change in $R^2 = .01$, $F(1, 20) = 0.14$, $p = .72$; **Table 9**), nor sustained HF-LF bias and total Calories (Change in $R^2 = .10$, $F(1, 20) = 2.51$, $p = .13$; **Table 10**).

Table 5. Initial HF-NF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	81.56	72.93	.23	67.01	75.35	.19
Initial HF-NF Bias				1.66	1.63	.21	1.25	1.71	.16
Initial HF-NF Bias x BMI*z*							1.25	1.46	.19
R^2		.08			.12			.15	
Change in R^2		.08			.04			.03	
F for change in R^2		1.83			1.04			0.73	

Note: HF-NF bias = Bias when comparing high palatable food to neutral/nonfood item. BMI*z* = body mass index, *z*-score.

Table 6. Initial LF-NF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	105.68	75.50	.30	115.56	79.41	.33
Initial LF-NF Bias				0.86	1.89	.10	0.13	2.42	.02
Initial LF-NF Bias x BMI*z*							1.31	2.63	.14
R^2		.08			.09			.10	
Change in R^2		.08			.01			.01	
F for change in R^2		1.83			0.21			0.25	

Note: LF-NF bias = Bias when comparing low palatable food to neutral/nonfood item. BMI*z* = body mass index, *z*-score.

Table 7. Initial HF-LF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	88.53	77.46	.25	57.47	80.93	.17
Initial HF-LF Bias				-0.46	1.48	-.07	0.82	1.81	.12
Initial HF-LF Bias x BMI*z*							-1.65	1.38	-.34
R^2		.08			.08			.14	
Change in R^2		.08			.004			.06	
F for change in R^2		1.83			0.10			1.44	

Note: HF-LF bias = Bias when comparing high palatable food to low palatable food. BMI*z* = body mass index, *z*-score.

Table 8. Sustained HF-NF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	104.57	71.66	.30	93.24	72.72	.27
Initial HF-NF Bias				-1.61	1.51	-.22	-2.45	1.75	-.33
Initial HF-NF Bias x BMI*z*							1.86	1.92	.23
R^2		.08			.12			.16	
Change in R^2		.08			.05			.04	
F for change in R^2		1.83			1.13			0.94	

Note: HF-NF bias = Bias when comparing high palatable food to neutral/nonfood items. BMI*z* = body mass index, *z*-score.

Table 9. Sustained LF-NF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	98.02	73.51	.28	103.65	76.62	.30
Sustained LF-NF Bias				-0.36	1.94	-.04	-0.95	2.54	-.10
LF-NF Bias§ x BMI*z*							0.74	2.01	.10
R^2		.08			.08			.08	
Change in R^2		.08			.002			.01	
F for change in R^2		1.83			0.04			0.14	

Note: LF-NF bias = Bias when comparing low palatable food to neutral/nonfood items. BMI*z* = body mass index, *z*-score.

§ Refers to sustained LF-NF

Table 10. Sustained HF-LF bias and BMI*z* on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	96.57	71.48	.28	108.76	76.08	.31	74.26	76.64	.21
Sustained HF-LF Bias				0.82	1.52	.12	2.81	1.93	.40
HF-LF Bias§ x BMI*z*							-2.37	1.50	-.46
R^2		.08			.09			.19	
Change in R^2		.08			.01			.10	
F for change in R^2		1.83			0.29			2.51	

Note: HF-LF bias = Bias when comparing high palatable food to low palatable food. BMI*z* = body mass index, *z*-score.

§ Refers to sustained HF-LF

Post Hoc Exploratory Analyses

Examining the impact of biases towards highly palatable foods and weight on fat, sugar, and salt intake[5]

Independent *post hoc* analyses were conducted to examine initial and sustained biases towards highly palatable foods (initial HF-NF, sustained HF-NF, initial HF-LF, sustained HF-LF), weight (BMI*z*), and fat, sugar, and salt intake. Tables with significant effects are included in the text (**Tables 11-12**). For the remaining models, see **Tables P25 – P34** in **Appendix P**.

Initial and Sustained HF-NF

BMI*z* did not significantly moderate the relationship between initial HF-NF bias and fat ($p = .74$; **Table P25**), sugar ($p = .83$; **Table P26**), or salt ($p = .66$; **Table P27**). BMI*z* did not significantly moderate the relationship between sustained HF-NF bias and fat ($p = .91$; **Table P28**), sugar ($p = .83$; **Table P29**), or salt ($p = .49$; **Table P30**).

Initial and Sustained HF-LF

The overall model fit was not significant for initial HF-LF and BMI*z* on fat ($p = .10$; **Table P31**). BMI*z* significantly moderated the relationship between initial HF-LF bias and sugar intake (Change in $R^2 = .24$, $F(1, 20) = 6.87$, $p = .02$), although the overall model did not reach significance (Overall $R^2 = .30$, $F(3, 20) = 2.88$, $p = .06$; **Table P32**). Similarly, BMI*z* significantly moderated the relationship between initial HF-LF bias and salt intake (Change in $R^2 = .19$, $F(1, 20) = 5.07$, $p = .04$), although the overall model did not reach significance (Overall $R^2 = .30$, $F(3, 29) = 2.34$, $p = .10$; **Table P33**).

[5] Pre-meal hunger was considered as a covariate for analyses examining fat, sugar, and salt, but adjusted models did not contribute to statistical interpretation or change results.

Sustained HF-LF significantly predicted arcsin fat after controlling for BMI*z* (Change in $R^2 = .33, F(1, 21) = 10.51, p = .004$), and the overall model was also significant (Overall $R^2 = .35, F(3, 20) = 3.53, p = .03$; **Table 11**). Sustained HF-LF significantly predicted sugar intake after controlling for BMI*z* (Change in $R^2 = .17, F(1, 21) = 4.61, p = .04$), although the overall model did not reach significance (Overall $R^2 = .21, F(3, 20) = 1.75, p = .19$; **Table 12**). There were no significant effects for sustained HF-LF and BMI*z* on salt ($p = .99$; **Table P34**).

Table 11. Sustained HF-LF bias and BMI*z* on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	0.01	0.01	.08	-0.01	0.01	-.10	-.004	.01	-.07
Sustained HF-LF Bias				-0.001	0.00	-.60	-0.001	0.00	-.68
HF-LF Bias§ x BMI*z*							0.00	0.00	.13
R^2		.01			.34			.35	
Change in R^2		.01			.33			.01	
F for change in R^2		0.15			10.51*			0.23	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to sustained HF-LF.

*$p < .01$.

Table 12. Sustained HF-LF bias and BMI*z* on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
BMI*z*	-4.11	4.70	-.18	-1.21	4.56	-.05	-1.20	4.88	-.05
Sustained HF-LF Bias				0.20	0.09	.44	0.20	0.12	.44
HF-LF Bias§ x BMI*z*							0.00	0.10	.001
R^2		.03			.21			.21	
Change in R^2		.03			.17			.00	
F for change in R^2		0.77			4.61*			0.00	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to sustained HF-LF.

*$p < .05$.

RESULTS OF SPECIFIC AIM 3: FOOD MARKETING AND ATTENTIONAL BIASES ON TOTAL ENERGY, FAT, SUGAR, AND SALT INTAKE

Specific aim 3 was to examine the effect of food marketing and ABs for high palatable foods on intake of total energy, fat, sugar, and salt[6] (**Table 13-36**).

Specific Aim 3a: Marketing, Initial Attentional Biases, and Intake

A total of 24 participants were included in specific aim 3a and 3b analyses (6 were excluded for unsuccessful deception, 2 for missing meal duration data or meal duration greater than 30 minutes, 2 for overweight status, 4 for lost AB data, and 7 for dot probe error rates at or above 10%; 1 excluded participant was in the deception and meal duration categories; 1 excluded participant was in the deception, overweight status, and lost AB data categories; and 1 excluded participant was in the deception and lost AB data categories; **Appendix Q**). The marketing condition by initial HF-NF bias interaction did not statistically predict intake of total Calories (Change in $R^2 = .03$, $F(1, 20) = 0.57$, $p = .46$; **Table 13)**. Marketing condition significantly predicted fat intake after controlling for initial HF-NF bias (Change in $R^2 = .18$, $F(1, 21) = 4.48$, $p = 05$), although the overall model did not reach significance (Overall $R^2 = .18$, $F(3, 20) = 1.47$, $p = .25$; **Table 14**). Initial HF-NF bias did not significantly moderate the relationship between marketing condition and intake of sugar (Change in $R^2 = .001$, $F(1, 20) = 0.02$, $p = .88$; **Table 15)** or salt (Change in $R^2 = .08$, $F(1, 20) = 1.80$, $p = .20$; **Table 16**).

Initial LF-NF did not significant moderate the relationship between marketing condition and intake of total Calories (Change in $R^2 = .07$, $F(1, 20) = 1.49$, $p = .24$; **Table**

[6] Pre-meal hunger was considered as a covariate for analyses examining fat, sugar, and salt, but adjusted models did not contribute to statistical interpretation or change results.

17). Marketing condition significantly predicted fat intake after controlling for initial LF-NF bias (Change in $R^2 = .17, F(1, 21) = 4.35, p = 05$), although the overall model did not reach significance (Overall $R^2 = .19, F(3, 20) = 1.56, p = .23$; **Table 18**). Initial HF-NF bias did not significantly moderate the relationship between marketing condition and intake of sugar (Change in $R^2 = .08, F(1, 20) = 1.88, p = .19$; **Table 19**) or salt (Change in $R^2 = .02, F(1, 20) = 0.51, p = .48$; **Table 20**).

Initial HF-LF bias did not significantly moderate the relationship between marketing condition and intake of total Calories (Change in $R^2 = .004, F(1, 20) = 0.07, p = .79$; **Table 21**). Marketing condition significantly predicted fat intake after controlling for initial HF-LF bias (Change in $R^2 = .19, F(1, 21) = 5.09, p = 04$), although the overall model did not reach significance (Overall $R^2 = .30, F(3, 20) = 2.78, p = .07$; **Table 22**). Initial HF-LF bias did not significantly moderate the relationship between marketing condition and intake of sugar (Change in $R^2 = .07, F(1, 20) = 1.77, p = .20$;) or salt (Change in $R^2 = .01, F(1, 20) = 0.15, p = .71$; **Table 24**).

Table 13. Marketing condition and initial HF-NF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-NF	2.03	1.61	.26	1.92	1.69	.25	0.86	2.12	.11
Marketing Condition				-38.63	140.22	-.06	-77.69	150.88	-.12
Marketing x HF-NF Bias§							2.61	3.47	.21
R^2		.07			.07			.10	
Change in R^2		.07			.003			.03	
F for change in R^2		1.59			0.08			0.57	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to initial HF-NF.

Table 14. Marketing condition and initial HF-NF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-NF	0.00	0.00	-.01	0.00	0.00	-.11	0.00	0.00	-.05
Marketing Condition				-0.05	0.02	-.43	-0.05	0.03	-.40
Marketing x HF-NF Bias§							0.00	0.001	-.09
R^2		.00			.18			.18	
Change in R^2		.00			.18			.01	
F for change in R^2		0.002			4.48*			0.13	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to initial HF-NF.

*$p < .05$

Table 15. Marketing condition and initial HF-NF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-NF	0.02	0.11	.05	0.05	0.11	.10	0.03	0.15	.07
Marketing Condition				8.60	9.15	.21	8.09	9.98	.19
Marketing x HF-NF Bias§							0.03	0.23	.04
R^2		.002			.04			.04	
Change in R^2		.002			.04			.001	
F for change in R^2		0.05			0.88			0.02	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to initial HF-NF.

Table 16. Marketing condition and initial HF-NF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-NF	-0.34	1.49	-.05	-0.37	1.57	-.05	1.35	2.00	.19
Marketing Condition				-9.31	130.71	-.02	53.64	136.61	.09
Marketing x HF-NF Bias§							-4.21	3.14	-.38
R^2		.002			.003			.09	
Change in R^2		.002			.00			.08	
F for change in R^2		0.05			0.01			1.80	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to initial HF-NF.

Table 17. Marketing condition and initial LF-NF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial LF-NF	0.16	1.86	.02	0.31	1.91	.04	-1.62	2.46	-.19
Marketing Condition				-78.77	141.90	-.12	-59.81	141.13	-.09
Marketing x LF-NF Bias§							4.67	3.83	.34
R^2		.00			.02			.08	
Change in R^2		.00			.01			.07	
F for change in R^2		0.01			0.31			1.49	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to initial LF-NF.

Table 18. Marketing condition and initial LF-NF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial LF-NF	0.00	0.00	.03	0.00	0.00	.09	0.00	0.00	.20
Marketing Condition				-0.05	0.02	-.42	-0.05	0.02	-.43
Marketing x LF-NF Bias§							0.00	0.001	-.17
R^2		.001			.17			.19	
Change in R^2		.001			.17			.02	
F for change in R^2		0.02			4.35*			0.42	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to initial LF-NF.

*$p < .05$

Table 19. Marketing condition and initial LF-NF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial LF-NF	0.10	0.12	.18	0.09	0.12	.16	-0.05	0.15	-.09
Marketing Condition				6.77	8.92	.16	8.10	8.80	.19
Marketing x LF-NF Bias§							0.33	0.24	0.37
R^2		.03			.06			.14	
Change in R^2		.03			.03			.08	
F for change in R^2		0.72			0.58			1.88	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to initial LF-NF.

Table 20. Marketing condition and initial LF-NF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial LF-NF	-1.01	1.66	-.13	-1.03	1.71	-.13	0.01	2.26	.002
Marketing Condition				8.47	127.53	.02	-1.71	129.84	-.003
Marketing x LF-NF Bias§							-2.51	3.52	-.20
R^2		.02			.02			.04	
Change in R^2		.02			.00			.02	
F for change in R^2		0.37			0.004			0.51	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to initial LF-NF.

Table 21. Marketing condition and initial HF-LF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-LF	-1.03	1.41	-.15	-1.26	1.46	-.19	-1.67	2.12	-.25
Marketing Condition				-102.49	141.64	-.16	-94.37	147.90	.15
Marketing x HF-LF Bias§							0.81	2.98	.09
R^2		.02			.05			.05	
Change in R^2		.02			.02			.004	
F for change in R^2		0.53			0.52			0.07	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to initial HF-LF.

Table 22. Marketing condition and initial HF-LF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-LF	0.00	0.00	-.10	0.00	0.00	-.20	0.00	0.00	.11
Marketing Condition				-0.05	0.02	-.45	-0.06	0.02	-.51
Marketing x HF-LF Bias§							-0.001	0.00	-.45
R^2		.01			.20			.30	
Change in R^2		.01			.19			.09	
F for change in R^2		0.23			5.09*			2.58	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to initial HF-LF.

*$p < .05$.

Table 23. Marketing condition and initial HF-LF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-LF	0.09	0.09	.22	0.12	0.09	.27	-0.003	.13	-.01
Marketing Condition				10.19	8.83	.24	12.56	8.85	.30
Marketing x HF-LF Bias§							0.24	0.18	.40
R^2		.05			.11			.18	
Change in R^2		.05			.06			.07	
F for change in R^2		1.11			1.33			1.77	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to initial HF-LF.

Table 24. Marketing condition and initial HF-LF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Initial HF-LF	-1.46	1.24	-.24	-1.54	1.30	-.26	-1.03	1.89	-.17
Marketing Condition				-35.27	126.40	-.06	-45.40	131.76	-.08
Marketing x HF-LF Bias§							-1.01	2.65	-.12
R^2		.06			.06			.07	
Change in R^2		.06			.003			.01	
F for change in R^2		1.38			0.08			0.15	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to initial HF-LF.

Specific Aim 3b: Marketing, Sustained Attentional Biases, and Intake[7]

Sustained HF-NF bias did not significantly moderate the relationship between marketing condition and intake of total Calories (Change in $R^2 = .05$, $F(1, 20) = 1.08$, $p = .31$; **Table 25**), fat (Change in $R^2 = .001$, $F(1, 20) = 0.03$, $p = .87$; **Table 26**), sugar (Change in $R^2 = .01$, $F(1, 20) = 0.12$, $p = .73$; **Table 27**), or salt (Change in $R^2 = .00$, $F(1, 20) = 0.004$, $p = .95$; **Table 28**). Sustained LF-NF bias did not significantly moderate the relationship between marketing condition and predict intake of total Calories (Change in $R^2 = .03$, $F(1, 20) = 0.59$, $p = .45$; **Table 29**), fat (Change in $R^2 = .003$, $F(1, 20) = 0.09$, $p = .77$; **Table 30**), sugar (Change in $R^2 = .00$, $F(1, 20) = 0.004$, $p = .95$; **Table 31**), or salt (Change in $R^2 = .00$, $F(1, 20) = 0.002$, $p = .97$; **Table 32**).

Sustained HF-LF bias did not significantly moderate the relationship between marketing condition and intake of total Calories (Change in $R^2 = .001$, $F(1, 20) = 0.02$, $p = .89$; **Table 33**). Sustained HF-LF statistically predicted intake of fat ($R^2 = .33$, $F(1, 22) = 10.83$, $p = .003$), and the overall model was also significant (Overall $R^2 = .38$, $F(3, 20) = 4.16$, $p = .02$; **Table 34**). However, marketing condition did not moderate that effect (Change in $R^2 = .01$, $F(1, 20) = 0.41$, $p = .53$). Sustained HF-LF bias predicted the intake of sugar ($R^2 = .21$, $F(1, 22) = 5.67$, $p = .03$), although the overall model did not reach significance (Overall $R^2 = .21$, $F(3, 20) = 1.73$, $p = .19$; **Table 35**). Sustained HF-LF did not significantly moderate the relationship between marketing condition and salt intake (Change in $R^2 = .03$, $F(1, 20) = 0.67$, $p = .42$; **Table 36**).

[7] Pre-meal hunger was considered as a covariate for analyses examining fat, sugar, and salt, but adjusted models did not contribute to statistical interpretation or change results.

Table 25. Marketing condition and sustained HF-NF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-NF	-1.38	1.54	-.19	-1.37	1.57	-.19	-3.06	2.25	-.42
Marketing Condition				-74.90	138.08	-.12	-94.14	139.04	-.15
Marketing x HF-NF Bias§							3.26	3.13	.32
R^2		.04			.05			.10	
Change in R^2		.04			.01			.05	
F for change in R^2		0.80			0.29			1.08	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to sustained HF-NF.

Table 26. Marketing condition and sustained HF-NF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-NF	0.00	0.00	.09	0.00	0.00	.09	0.00	0.00	.06
Marketing Condition				-0.05	0.02	-.41	-0.05	0.02	-.41
Marketing x HF-NF Bias§							0.00	0.001	.05
R^2		.01			.17			.18	
Change in R^2		.01			.17			.001	
F for change in R^2		0.18			4.20¥			0.03	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to sustained HF-NF.

¥ p = .053.

Table 27. Marketing condition and sustained HF-NF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-NF	-0.02	0.10	-.05	-0.02	0.10	-.05	-0.06	0.15	-.13
Marketing Condition				7.69	8.93	.18	7.26	9.21	.17
Marketing x HF-NF Bias§							0.07	0.21	.11
R^2		.002			.04			.04	
Change in R^2		.002			.03			.01	
F for change in R^2		0.05			0.74			0.12	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to sustained HF-NF.

Table 28. Marketing condition and sustained HF-NF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-NF	-0.46	1.41	-.07	-0.46	1.44	-.07	-0.37	2.13	-.06
Marketing Condition				-2.05	127.03	-.004	-0.95	131.32	-.002
Marketing x HF-NF Bias§							-0.19	2.96	-.02
R^2		.01			.01			.01	
Change in R^2		.01			.00			.00	
F for change in R^2		0.11			0.00			0.004	

Note: HF-NF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to sustained HF-NF.

Table 29. Marketing condition and sustained LF-NF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained LF-NF	-0.09	1.97	-.01	-0.41	2.07	-.04	0.90	2.69	.10
Marketing Condition				-83.12	145.71	-.13	-97.34	148.32	-.15
Marketing x LF-NF Bias§							-3.28	4.27	-.22
R^2		.00			.02			.04	
Change in R^2		.00			.02			.03	
F for change in R^2		0.002			0.33			0.59	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to sustained LF-NF.

Table 30. Marketing condition and sustained LF-NF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained LF-NF	0.001	0.00	.30	0.00	0.00	.21	0.00	0.00	.16
Marketing Condition				-0.04	0.02	-35	-0.04	0.02	-.34
Marketing x LF-NF Bias§							0.00	0.001	.08
R^2		.09			.21			.21	
Change in R^2		.09			.11			.003	
F for change in R^2		2.23			3.01			0.09	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to sustained LF-NF.

Table 31. Marketing condition and sustained LF-NF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained LF-NF	-0.17	0.12	-.29	-0.16	0.13	-.26	-0.15	0.17	-.25
Marketing Condition				4.76	8.96	.11	4.68	9.26	.11
Marketing x LF-NF Bias§							-0.02	0.27	-.02
R^2		.09			.10			.10	
Change in R^2		.09			.01			.00	
F for change in R^2		2.08			0.28			0.004	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to sustained LF-NF.

Table 32. Marketing condition and sustained LF-NF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained LF-NF	0.83	1.76	.10	0.88	1.87	.11	0.94	2.46	.11
Marketing Condition				14.27	131.41	.02	13.60	135.71	.02
Marketing x LF-NF Bias§							-0.16	3.91	-.01
R^2		.01			.01			.01	
Change in R^2		.01			.001			.00	
F for change in R^2		0.22			0.01			0.002	

Note: LF-NF bias = Bias when comparing low palatable foods to neutral/nonfood items.

§ Refers to sustained LF-NF.

Table 33. Marketing condition and sustained HF-LF bias on total Calories

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-LF	0.18	1.48	.03	0.56	1.62	.08	0.87	2.71	.13
Marketing Condition				-95.31	151.37	-.15	-94.78	155.06	-.15
Marketing x HF-LF Bias§							-0.50	3.43	-.06
R^2		.001			.02			.02	
Change in R^2		.001			.02			.001	
F for change in R^2		0.01			0.40			0.02	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to sustained HF-LF.

Table 34. Marketing condition and sustained HF-LF bias on arcsin fat

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-LF	-0.001	0.00	-.57	-0.001	0.00	-.49	0.00	0.00	-.34
Marketing Condition				-0.03	0.02	-.22	-0.03	0.02	-.22
Marketing x HF-LF Bias§							0.00	0.00	-.19
R^2		.33			.37			.38	
Change in R^2		.33			.04			.01	
F for change in R^2		10.83*			1.40			0.41	

Note: HF-LF bias = Bias when comparing high palatable foods to neutral/nonfood items.

§ Refers to sustained HF-LF.

*$p < .01$.

Table 35. Marketing condition and sustained HF-LF bias on sugar

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-LF	0.20	0.09	.45	0.20	0.09	.45	0.22	0.16	.49
Marketing Condition				0.65	8.76	.02	0.68	8.97	.02
Marketing x HF-LF Bias§							-0.03	0.20	-.05
R^2		.21			.21			.21	
Change in R^2		.21			.00			.001	
F for change in R^2		5.67*			0.01			0.02	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to sustained HF-LF

*$p < .05$.

Table 36. Marketing condition and sustained HF-LF bias on salt

	Model 1			Model 2			Model 3		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Sustained HF-LF	-1.17	1.31	-.19	-1.35	1.44	-.22	0.18	2.37	.03
Marketing Condition				45.36	134.71	.08	47.95	135.83	.08
Marketing x HF-LF Bias§							-2.45	3.00	-.30
R^2		.04			.04			.07	
Change in R^2		.04			.01			.03	
F for change in R^2		0.80			0.11			0.67	

Note: HF-LF bias = Bias when comparing high palatable foods to low palatable foods.

§ Refers to sustained HF-LF