January 2010

Effects of Attention Allocation on Habituation to Food Cues in Normal Weight and Overweight Children

Vandana Aspen
Washington University in St. Louis

WASHINGTON UNIVERSITY

Department of Psychology

Dissertation Committee:
Denise Wilfley, Chair
Mark McDaniel
John Newcomer
Thomas Oltmanns
Richard Stein
Desiree White

EFFECTS OF ATTENTION ALLOCATION ON HABITUATION TO FOOD CUES IN

NORMAL WEIGHT AND OVERWEIGHT CHILDREN

by

Vandana Passi Aspen

A dissertation presented to the
Graduate School of Arts and Sciences
of Washington University in
partial fulfillment of the
requirements for the degree
of Doctor of Philosophy

August, 2010

Saint Louis, Missouri

ABSTRACT OF THE DISSERTATION

Effects of Attention Allocation on Habituation to Food Cues

in Normal weight and Overweight Children

by

Vandana Passi Aspen

Doctor of Philosophy in Psychology

Washington University in St. Louis, 2010

Professor Denise Wilfley, Chairperson

Despite the rising prevalence of pediatric overweight, minimal research has been conducted to understand the basic biological processes underlying overweight in children. The present study assesses changes in physiological response (i.e., salivation) to food over time. The primary aims were to examine whether salivation patterns in children vary based on weight status and/or allocating attention to a distracter task. It was hypothesized that: 1) overweight children would not habituate (salivation at the final trial would not decrease back to baseline level), regardless of distracter task condition while the normal weight children would habituate (i.e., salivation at the final trial would decrease back to baseline level) and that 2) all children attending to the distracter task would take longer to habituate as compared to those not attending to the task. Participants were 31 normal weight and 26 overweight children ages 9 to12 years. All children were presented with nine one-minute trials of a food stimulus (French fries). During each intertrial interval, participants either listened to sequential one-minute presentations of an audio-book (distracter task) or listened to white noise (no-distracter-task control). Pattern and rate of salivation were measured using a validated procedure (the Strongin-Hinsie Peck method) and analyzed using repeated measures ANCOVA

and Kaplan-Meier survival analysis. The rate of change in salivation over trials differed significantly by weight status ($p = .01$) but not by distracter task condition ($p > .10$). Specifically, regardless of distracter task condition, at the final trial of the study, normal weight children habituated to food cues while overweight children did not. Results suggest that children's physiological response to food is related to weight status. Such atypical habituation patterns could potentially lead to overconsumption, thus serving as a possible causal or maintaining factor in childhood overweight. The lack of a distracter effect is in contrast to previous findings and may be due to differences in methodology across studies. Future directions, including, a) experiments to explore causal mechanisms, b) experiments testing habituation in more naturalistic settings and, c) prospective studies to determine the role of salivary response in OW, are discussed.

ACKNOWLEDGEMENTS

This dissertation would not have been possible without the numerous individuals who helped me along the way.

First, I would like to thank my advisor Denise Wilfley for her dedicated mentorship throughout my graduate career. Whenever I needed it, she was there to offer her wisdom, guidance and encouragement in my abilities. I am especially grateful for her support on this project, which began during my transition to motherhood. Over the past year and a half, she has been both flexible and understanding of my role as a new mother, and supportive of my continued academic success. She has genuinely cared for both my personal and professional goals and has taught me a great deal about the balance between work and family. I have the sincerest appreciation for Denise, as I could not have achieved any of my graduate accomplishments without her. In short, she has been an incredible advisor and role model. As I embark on my own career, I will aspire to reach her broad perspective, her vast knowledge, and the diligence and responsibility that she applies to every project she embarks on.

I could not have made it throughout graduate school without Richard Stein, mentor colleague and friend. I will forever remember his support of this project and all others I embarked on while in graduate school. He was always there to provide me with thoughtful comments, hands-on mentorship, and late night brainstorming sessions. I am truly indebted to him for all of his help and support.

Without a doubt, the selfless sacrifices they have made for me are the sole reason I am where I am today. *Thank you, mom and dad, from the bottom of my heart.*

I would also like to thank my nieces, Ashlyn and Kiran Brandt, for reminding me of the simple pleasures in life, and of course, my sister and best friend, Nidhia Brandt, for her love and unwavering support in my abilities. I must also thank my mother-in-law, Maria Jedynak, for her love and caring during these years. She has always made my accomplishments feel so special and it is an honor that she is taking time off from work and flying from afar to attend my graduation ceremony.

Of course, a very special thank you to my son Dylan for bringing meaning to my life, inspiring me to be the best that I can, and for requiring me to be an expert in time management. Finally, my acknowledgements would be sorely incomplete without a very special thank you to my husband, Matt Aspen. Throughout the past six years, he has been my anchor. He has sacrificed uncountable days, weekends and evenings so that I could complete my work. I am forever grateful for his foot massages, warm meals after a long night, flexibility, unconditional support and boundless love. Unquestionably, without my family none of this would have been possible.

I would like to dedicate this to my dog, Harley (1993-2007). He was my dearest friend and companion for a great portion of my academic journey, all the way from high school through most of graduate school. I miss him with all of my heart.

Table of Contents

LIST OF TABLES

List of Figures

Introduction

Overview

Over the past two decades, adult obesity has risen in the United States (Robinson & Killen, 2002) and globally at such an alarming rate that the World Health Organization has now classified it as a worldwide epidemic (WHO, 1998). This increase in obesity has been associated with exponential increases in health care costs at a national level, as well as significant mental and physical difficulties at the level of individuals with this condition. Unfortunately, obesity in adults is a refractory condition that is often highly resistant to dietary and behavioral treatment (Douketi, Macie, Thabane, & Williamson, 2005; Wilson, 1994).

Childhood overweight (OW), a robust risk factor for adult obesity (Parsons, Power, Logan, & Summerbell,1999) is also on the rise (see Figure 1) and is currently the most prevalent nutritional disease among children and adolescents in the United States (Dietz, 1998). However, unlike treatment for adult obesity, results from treatment trials with OW children have been more promising (Epstein, Myers, Raynor, & Saelens, 1997; Epstein, Valoski, Wing, & McCurley, 1994; Wilson, 1994). As a result, some researchers have shifted their focus towards treating OW children rather than obese adults. Although current childhood OW treatment programs are effective, they are not a panacea, and new research is needed to improve the immediate and long-term outcomes of such treatment programs.

Eating behaviors are central to understanding the development and maintenance of OW in children. However, the basic biological and environmental factors influencing eating behaviors in children are poorly understood. Most treatment studies on childhood OW have tended to discount biology and focus solely on modifying environmental factors (e.g., stimulus control). Consequently, biological factors in the treatment of

1

Figure 1. Trends in Child and Adolescent Overweight[1]

Males 12-19
Males 6-11
Females 12-19
Females 6-11
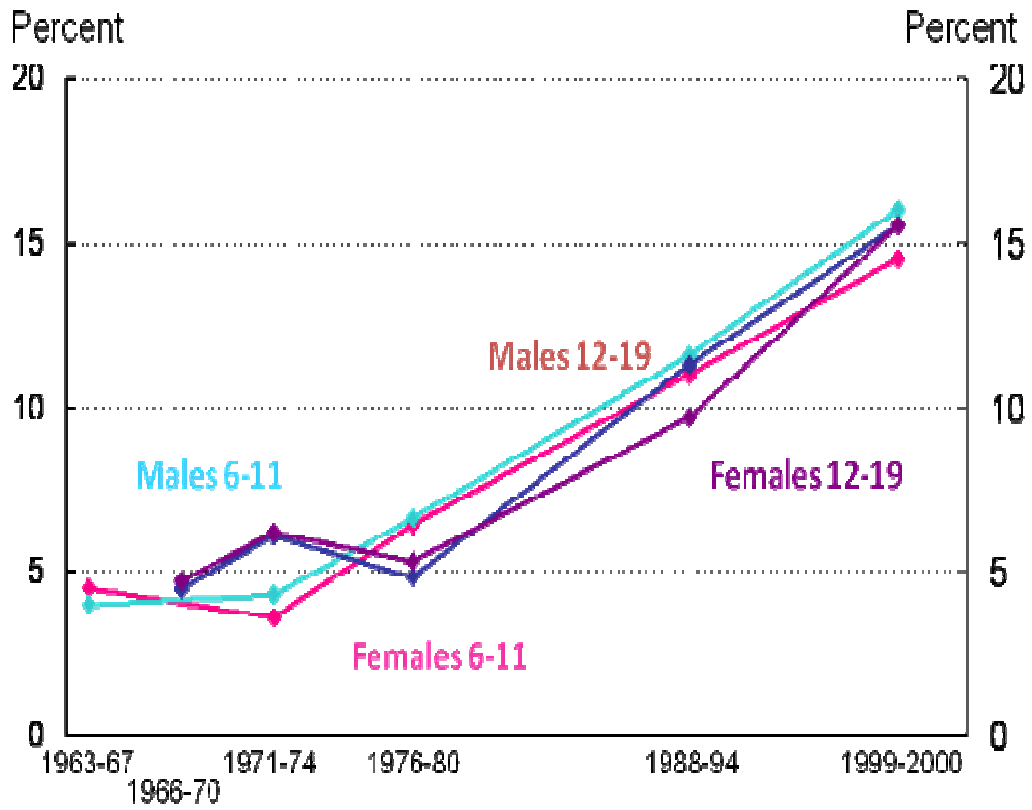
[1] Source: National Health Examination Surveys II (ages 6-11) and III (ages 12-17), National Health and Nutrition Examination Surveys I, II, III and 1999-2000, NCHS, CDC.

childhood OW have not received sufficient attention. The present study is designed to help bridge this gap by providing an examination of the effects of environmental factors on a basic physiological function related to the regulation of food intake.

This proposal's introduction will begin with key definitions and current data on the prevalence and associated features of childhood OW. This section is followed by a review of the treatment literature and a discussion of translational research in the context of environmental and biological factors that influence food ingestion. Lastly, the significance of the present research, specific hypotheses and clinical implications are discussed.

Defining Obesity and OW

For adults, the Centers for Disease Control and Prevention determine obesity and OW status using body mass index (BMI; weight in kilograms divided by the square of height in meters). In this adult classification system, a BMI greater than 25 indicates OW, and a BMI of 30 or greater indicates obesity. Among children, BMI is used as well; however, once calculated, it is plotted on the Centers for Disease Control growth charts, which take into account both age- and sex-specific characteristics to generate a percentile rank. This approach is used to adjust for expected changes in the amount of body fat that occur with age and expected differences that are related to sex during childhood.

Due to the stigma associated with the term 'obesity', this label has been avoided with children and has been replaced with the term OW. Specifically, based on the Centers for Disease Control growth curves (Kuczmarski et al., 2000), Children with a BMI between the 85th and 95th percentiles are categorized as "at risk of OW," whereas those with a BMI at or above the 95th percentile are considered OW.

Prevalence

Current estimates indicate that the prevalence of obesity in adults in the United States has increased from 20.9% in 2001 (Mokdad et al., 2003) to 32.2% in 2004 (Ogden et al., 2006). This general increase has been found across sex, race, educational level, and health status.

Based on findings from the National Health and Nutritional Examination Survey, approximately 31.9% of children and adolescents ages 2 to19 years are currently classified as OW or at risk for OW (Ogden, Carroll, & Flegal, 2008) with the highest percentages in non-Hispanic black children ages 12 to 19 years (38.1%) and Mexican American children ages 6 to 11 years (42.8%). The United States prevalence of OW in children has increased significantly from 13.9% in 1999 to 17.1% (over 12.5 million) in 2004 (Ogden et al., 2006).

Consequences of OW

Treating childhood OW is an urgent matter because in addition to the high prevalence rates and strong link with adult obesity, there are also significant medical, economic, and psychosocial consequences associated with this condition. These consequences will be discussed in detail in the sections below.

Medical consequences. Higher body weight increases the risk of all-cause mortality (NIH, 1998), and after OW status is reached, nearly all organ systems in the body are affected (Strauss, 1999). Childhood OW, independent of its relation to adult obesity, is associated with significant medical problems which have tripled among children 6 to17 years of age over the past 30 years (Dietz, 2006). The medical sequelae often associated with childhood OW include coronary artery disease, hypertension, dyslipidemia, orthopedic problems, endocrine problems (e.g., diabetes, insulin resistance), gastroenterological problems, respiratory (e.g., asthma, sleep apnea) and neurological difficulties (Strauss, 1999).

Most notably, as mentioned above, childhood OW is a significant risk factor for adult obesity (Parsons et al., 1999) and increases the risk of adult morbidity and mortality independent of adult OW status (Must, Jacques, Dallal, Bajema, & Dietz, 1992). Must and colleagues (1992) attempted to re-contact 508 lean (25th-50th percentile) and OW (>75th percentile) adolescents 13 to 18 years who participated in the Harvard Growth Study of 1922 to 1935. Medical history, weight, functional capacity, and other risk factors were assessed; cause of death was obtained from the death certificate for all deceased participants. Results revealed that OW in adolescence was associated with an increased risk of mortality (from all causes and disease specific), particularly among men. In terms of morbidity, coronary heart disease and atherosclerosis, among other medical conditions, were increased in both men and women who were OW during adolescence. In this study (Must et al.), OW in childhood was a stronger predictor of morbidity and mortality than obesity in adulthood; other studies have reported similar findings (Morgan, Tanofsky-Kraff, Wilfley, & Yanovski, 2002).

In addition, in the absence of any treatment, severely obese Children will gain weight at a continuous and substantial rate (Mockus, Epstein, & Wilfley, 2005). In a review of the literature from 1970 to 1992, Serdula et al. (1993) reported that approximately half of OW school-age children become obese as adults, and this risk was at least twice as high for OW children compared with normal weight children. These trends have been documented in numerous prospective studies (Freedman et al., 2004; Must et al., 1992; Whitaker, Wright, Pepe, Seidel, & Dietz, 1997) signifying the persistence of this condition.

Psychosocial consequences. OW during childhood and adolescence is also associated with significant social and emotional consequences. Several studies have shown that OW children have higher rates of depression (Erickson, Robinson, Haydel, & Killen, 2000; Everson, Maty, Lynch, & Kaplan, 2002; Sjoberg, Nilsson, & Leppert, 2006;

Swallen, Reither, Haas, & Meier, 2005) and an increased risk for a suicide attempt (Falkner et al., 2001) as compared with their normal weight peers. In addition, OW Children receive more weight- and appearance-related teasing by peers and family members (Eisenberg, Neumark-Sztainer, & Story, 2006; Hayden-Wade et al., 2005), have more negative peer interactions (Braet, 1997) and are more socially isolated (Strauss & Pollack, 2003) than their normal weight peers.

Discrimination and prejudice related to OW status is frequently reported (Goldfield & Chrisler, 1995; Gortmaker, Must, Perrin, Sobol, & Dietz, 1993) and can be especially troubling during the school years. Children as young as first grade used exclusively unfavorable adjectives to describe silhouettes of obese children (e.g., lazy, dirty, stupid, cheats, and lies), and ranked obese children as those they would least like to have as friends (Richardson, Goodman, Hastorf, & Dornbusch, 1961). Latner and Stunkard (2003) recently replicated this study and asked a sample of approximately four hundred fifth and sixth grade children to rank drawings in terms of likability of a same sex child with OW, disabilities or no disability (healthy child). Children in both studies ranked the healthy child as most likeable and the OW child as the least likable. Notably, in the most recent study (Latner & Stunkard, 2003) the difference between the healthy child and OW child were further polarized, suggesting that prejudice towards OW has increased over the past 40 years.

OW children have a more negative body image than their peers (Striegel-Moore et al., 2000), which is an identified risk factors for the development of eating disorders (Killen et al., 1994; Killen et al., 1996). Indeed, studies have found that OW children are significantly more likely than normal weight children to report binge eating (consumption of a large amount of food accompanied by a sense of loss of control), with higher rates of bingeing increasing with proportion of OW (Goldschmidt, Passi Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008; Neumark-Sztainer, Story, French, & Resnick, 1997;

6

Tanofsky-Kraff et al., 2004). OW children are also more likely to engage in unhealthy weight-control methods (Boutelle, Neumark-Sztainer, Story, & Resnick, 2002; Neumark-Sztainer, Wall, Eisenberg, Story, & Hannan, 2006). A recent review of this literature reported that up to 79% of OW children engage in unhealthy weight control behaviors (e.g., fasting, using cigarettes or diet pills to suppress appetite) while up to 17% engage in extreme weight control behaviors (e.g., self-induced vomiting, laxative or diuretic abuse) (Goldschmidt et al., 2008). This overlap in disordered eating among OW youth is particularly concerning as each condition has been found to perpetuate the other. Specifically, disordered eating is predictive of childhood OW (Field et al., 2003; Tanofsky-Kraff et al., 2004) and childhood OW is a risk factor for disordered eating and full syndrome eating disorders (Fairburn et al., 1999; Striegel-Moore et al., 2005).

Economic consequences. OW- and obesity-related conditions are among the most costly health-care expenditures, even exceeding direct costs for coronary heart disease, hypertension, and diabetes (Thompson & Wolf, 2001). Wang and Dietz (2002) recently published a study examining the economic costs associated specifically with OW in Children from 1979 to 1999. They found that these hospital costs have more than tripled over the past 20 years and were estimated at 127 million dollars per year. For Children, the greatest increase in OW-related health conditions over this time-period included, sleep apnea (436%) and gallbladder disease (228%), both of which require significant medical attention.

Treatment of Childhood OW

A detailed review of the behavioral weight-loss treatment literature for adult obesity is beyond the scope of this paper; however, a few key points are germane to the current discussion. There is extensive literature on this topic spanning over 40 years. The findings from these treatment outcome studies have been positive immediately following treatment (Perri & Fuller, 1995), yet very discouraging in terms of maintenance

(Anderson, Konz, Frederich, & Wood, 2001; Jeffery et al., 2000; Wilson, 1994). Indeed, Wilson (1994) reported that between 90 and 95% of obese adults receiving behavioral treatment returned to their baseline weight within five years. As a result of these and similar findings in the field, the efficacy of behavioral treatment with adults has been questioned (Garner & Wooley, 1991). As such, the research focus for reducing the obesity epidemic is shifting towards treatment and prevention in children and adolescents (Epstein et al., 1997).

In addition to the refractory nature of adult obesity, there are three key advantages to targeting OW children. First, parents can be used as an agent of change. Research has shown that parental involvement is critical to treatment success (Epstein et al., 1994), as parents can model appropriate behaviors (Wrotniak, Epstein, Paluch, & Roemmich, 2005), provide social support and food management (Wilfley, Passi, Cooperberg, & Stein, 2006; Wilson, 1994). In addition, when parents take charge of factors in the home environment (e.g., amount of high-fat foods in the home) they reduce the need for the child to rely on self-control (Wilson, 1994), a factor often associated with relapse (Foreyt & Goodrick, 1991). Finally, targeting younger populations shortens the time-period between onset of the condition and initiation of treatment, increasing the likelihood that behaviors associated with OW (e.g., regularly consuming high fat foods) will be malleable. For example, because eating habits become more ingrained with age, children can implement dietary changes more easily to facilitate better long-term outcome.

The broad goal of childhood OW interventions is to reduce the BMI percentile (amount the child is above the 95th percentile) to non-OW status (below the 95th percentile). To accomplish this goal, interventions usually target dietary intake (e.g., teaching healthier eating choices) and behavior change (e.g., reducing sedentary behavior and/or increasing physical activity). To date, over 70 randomized treatment

studies have been conducted on child and adolescent weight loss (Zametkin, Zoon, Klein, & Munson, 2004). Overall, behavioral modification studies have shown short-term efficacy with a 5 to 20% decrease in BMI percentile (percentage by which a child's weight on the CDC growth chart drops; e.g., child at 95th percentile drops 5% during study = 90th percentile) (Jelalian & Saelens, 1999). Most of the treatment programs that have been tested in randomized control trials for OW children are multi-component and include comprehensive behavioral and dietary modifications (e.g., self-monitoring of diet and activity, contingency management, stimulus control strategies) (e.g., Emes, Velde, Moreau, Murdoch, & Trussell, 1990; Floodmark, Ohlosson, Ryden, & Sveger, 1993; Israel, Guile, Baker, & Silverman, 1994). In a thorough review of the randomized control interventions for OW children and adolescents (n = 42), Jelalian and Saelens (1999) found that studies using these multi-component behavioral programs were more effective in short-term weight-loss in comparison to wait-list control and nutrition education. Although there are variations in- and modifications of- the components used in these studies (e.g. amount of exercise prescribed, parental role, number of sessions, group vs. individual), currently there is not enough data to determine which specific components are most efficacious for achieving weight-loss (Jelalian & Saelens, 1999).

Several recent literature reviews confirm that of the multi-component behavioral programs offered, family-based behavioral treatment is the most empirically supported treatment for children ages 7 to 12 years (Faith, Saelens, Wilfley, & Allison, 2001; Jelalian & Saelens, 1999; Morgan et al., 2002; Tanofsky-Kraff, Hayden-Wade, Cavazos, & Wilfley, 2003). In contrast to adult obesity interventions, family-based behavioral treatment has produced promising long-term weight loss outcomes (Epstein et al., 1994). Key components of this intervention include parental involvement, diet (e.g., reduce caloric intake, increase nutrient density) and activity modifications (e.g., daily exercise routine). Despite these promising findings for family-based behavioral

treatment, it is not a universal remedy. There are few large-scale randomized controlled trials in this area, and long-term maintenance is difficult to achieve (Epstein et al., 1997; Marcus, Kalarchian, & Levine, 2005; Wilfley et al., 2007). Thus, further research is needed to improve the efficacy of these clinical interventions (Summerbell et al., 2003; Wilfley et al., 2007).

Current treatments available for very severely OW children and adolescents (i.e., >99th BMI percentile) include two medications approved by the Food and Drug administration (sibutramine for adolescents $\geq$16 years and orlistat for adolescents $\geq$12 years), very low-calorie diet and weight control surgery (Barlow & the Expert Committee, 2009). Findings from these intensive treatments have been favorable; however, they are considered a last resort and should only be used when all other empirically supported weight-loss approaches fail (Barlow & the Expert Committee).

Translational Research

Translational T1 research ("bench to bedside") is well accepted as an approach for improving the efficacy of clinical interventions (Sussman, Valente, Rohrbach, Skara, & Pentz, 2006), but there are few clinical researchers who conduct basic research and translate that basic research into clinical interventions (Epstein, 1992). Translational T1 research is innovative and multidisciplinary in nature, as it applies paradigms and methods from relevant areas of basic science to advance clinical diagnosis, prevention and treatment development.

Existing research on the treatment of OW and obesity can be supplemented with laboratory studies exploring the basic processes that bring about dysregulated eating. Within the sphere of obesity, a great deal of molecular and genetic research has already been conducted and successfully used to develop pharmacological interventions (Epstein et al., 1998). In contrast, very little research has been conducted in the area of

10

childhood OW and is urgently needed to better understand the etiology and maintaining factors of this condition.

Childhood OW results from ingesting more calories than are expended. Although biology and environment are two key variables that influence ingestion (Brownell, 2002; Hill & Peters, 1998), little research has been conducted to understand how these variables interact to influence weight status. Information on the interplay between biology and environment may help to explain the refractory nature of OW and inform behavioral approaches to treat and prevent OW.

Biological Effects on Childhood OW

Eating, a biological necessity, is a motivated behavior influenced by numerous factors (Schwartz, 2001). One key factor influencing food intake is simply the presence of food. In particular, the sensory stimulation of food (i.e., color, flavor and aroma) elicits the first responses from the body in preparation for ingestion. These physiological responses, known as the cephalic phase response (CPR; Powley & Berthoud, 1985), include increases in saliva, heart rate, temperature, and gastric activity (Nederkoorn, Smulders, & Jansen, 2000). The CPR is significant because it is associated with the initiation and termination of an eating episode and is implicated in influencing the total amount of food that an individual consumes within a meal (Nederkoorn et al., 2000). Accordingly, understanding the role of CPR in food ingestion may help identify the factors leading to overconsumption in Children.

Salivary flow, a primary component of CPR, is considered a valid measure of hunger (Wooley & Wooley, 1973) as it is directly proportional to the duration of food deprivation (Wooley & Wooley, 1976). In addition, data in non-human primates and humans show that repeated presentations of the same food cues (e.g., during a given meal) lead to a decrease in salivation (Epstein, Saad, Giacomelli, & Roemmich, 2005), resulting in satiation for that food (Swithers & Hall, 1994) and the termination of an

11

eating episode (Wisniewski, Epstein, & Caggiula, 1992). This general process of decreasing responsiveness after repeated exposures to a stimulus is referred to as habituation and is a common biological process found across species.

Through habituation, humans and animals learn to ignore stimuli that have lost novelty or meaning, allowing them to attend to stimuli that are more important (Stephenson & Siddle, 1983). In applying this paradigm to OW, it can be hypothesized that disruptions in habituation lead to an extended eating episode and, overconsumption. Accordingly, some researchers have become interested in whether disrupting habituation may explain dysregulated eating in children and adults.

To date, approximately 20 studies have been published in humans examining the relationships between habituation and hedonics, smoking, food types, food variety, facial muscle response, and motivated responding. Populations studied include normal weight adults and children, obese adults, and women diagnosed with bulimia nervosa. To the author's knowledge, all such studies in humans have been conducted in the laboratory of Dr. Leonard Epstein at the University of Buffalo. Relevant findings from this body of research are described below.

Habituation trials. In habituation trials, participants sit quietly in a room with minimal distractions. As a starting point, a baseline level of salivation is recorded in the absence of food. The experimenter then repeatedly presents the same food to the participants across a number of trials, with the food presented for approximately one-minute on each trial (e.g., 10 hamburger presentations on 10 trials lasting one minute each). The food is either heated and placed directly under the participant's nose, or it is placed directly on their tongue. The participants are instructed to smell the food (if heated) and imagine eating it. The level of salivation on each trial is recorded. The typical pattern observed is a sharp increase in salivation when food is initially presented, followed by a progressive decline over subsequent trials until a baseline level of

12

salivation is reached. Habituation is defined as a salivation level at the final trial that returns to baseline (non-significant difference).

Conditions associated with distinctive eating patterns. To date, three studies have been conducted investigating the relation between conditions associated with distinctive eating patterns and habituation. In the first study (Wisniewski, Epstein, Marcus, & Kaye, 1997), the salivation levels of 18 women with bulimia nervosa and 18 demographically-matched controls were compared across 10 trials during which frozen yogurt was presented. Control subjects showed a typical decrease in salivation across trials. In contrast, the opposite pattern was observed in women with bulimia nervosa, as salivation increased across trials.

In the second study (Epstein, Paluch, & Coleman, 1996), salivary response was assessed in 10 obese and 10 non-obese college women. Participants were presented with 10 repeated trials of lemon yogurt. As shown in Figure 2, habituation was observed in the non-obese women but not in the obese women[2]. The third and most recent study (Temple, Giacomelli, Roemmich, & Epstein, 2007; published after the completion of this study) used a more indirect method to examine habituation which is entitled motivated responding (i.e., operant responding to food). Motivated responding was measured in children whose BMI was below the 85th percentile (normal weight) and above the 85th percentile (i.e., at-risk for OW and OW) using a computer generated task. In this task, participants could earn a 100-kcal portion of a Wendy's Jr. Cheeseburger per response. The specific task consisted of a variable interval reinforcement schedule during which

---

[2] There are multiple ways to describe habituation data. Some authors may state that one group took longer to habituate even though their data do not depict this scenario (the assumption is that everyone habituates eventually). However, for the purposes of this review and of the present study, the habituation data is described as what occurred during the trials of the experiment.

Figure 2. Salivary Responses in Obese and Nonobese Women Across Trial Blocks. [3]
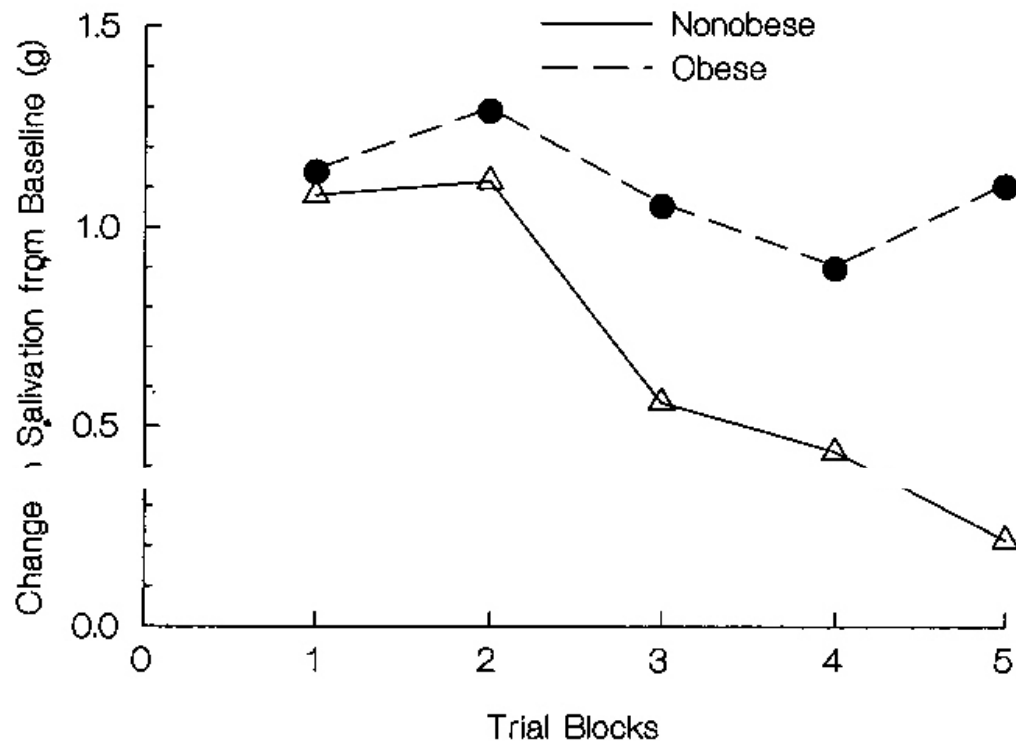
[3] From "Differences in salivation to repeated food cues in obese and nonobese women" by Epstein, L.H., Paluch, R., & Coleman, K.J., 1996. *Psychosomatic Medicine, 58*(2), *160-164.* Adapted with permission of the author.

participants worked for the food stimulus by pressing the mouse button until a green square appeared on the screen (a green square was equated with one point). The primary outcome measure was number of responses (i.e., clicking the mouse) made during each two-minute interval. Food was given immediately after the point was earned, and participants were instructed to work for the food stimulus as long as they desired it. Results indicated that children with a BMI above the 85th percentile took significantly longer to habituate (i.e., decrease their rate of mouse clicking across trials) via motivated responding as compared with the children below the 85th percentile.

Results from these three studies suggest that conditions characterized by certain eating patterns are associated with abnormal patterns of habituation in response to food.

Competing environmental stimuli. Epstein and colleagues (2005) conducted two experiments to determine whether children's responses to food stimuli are disrupted by allocating attention to non-food related tasks during an eating episode. In these studies, allocation of attention refers to focusing attention on a task during an eating episode (e.g., watching television while eating). In the first experiment, 42 normal weight children 8 to12 years of age were randomly assigned to one of three task conditions that varied in attentional demand: (1) controlled search task (high attention), (2) automatic search task (low attention) and (3) no task (control). Children were presented with eight one-minute presentations of a hamburger during which they were instructed to look at and smell the hamburger, but not eat it. During intertrial intervals the (one minute between each food presentation), children in the high and low attention task conditions completed their respective computer tasks, whereas children in the no-task condition sat quietly. The authors reported that children in the high-attention task condition did not habituate, as their salivation did not decline across trials. In contrast, children in the low-attention and no-task conditions did habituate, with children in the low attention condition taking

longer to habituate than children in the no-task condition. In sum, the findings from this study show that increasing attentional demands results in decreased habituation.

In the second experiment, 22 normal weight children 9 to 12 years of age were randomly assigned to an audio-book or white noise condition. The children were presented with 10 one-minute presentations of pizza. During the intertrial intervals (one minute between each food presentation), children either listened to one minute of an audio-book or sat quietly. The results from this experiment were similar to those of the first experiment, in that children in the attention-demanding audio-book condition took longer to habituate as compared to those children in the white noise condition (See Figure 3).

The findings from these two experiments suggest that attentional competition between non-food and food stimuli influences the rate of habituation. In particular, tasks requiring a great deal of attention appear to impede habituation, whereas tasks involving moderate attention protract the habituation process. See Table 1 for a comprehensive review of habituation studies investigating competing stimuli.

Summary of habituation findings. To summarize, this body of research suggests that termination of a particular eating episode is related to a decrease in salivation after repeated exposure to food cues (e.g., meal), and competing attention-demanding activities interfere with this process. In addition, individuals with distinctive eating patterns appear to display abnormal patterns of habituation. These findings may be able to help explain some of the environmental data (e.g., watching television/ playing video game) linked with OW. In the following section, applicable literature is reviewed.

Figure 3. Changes in Salivation in the Audio-book and no Audio-book Conditions[4]
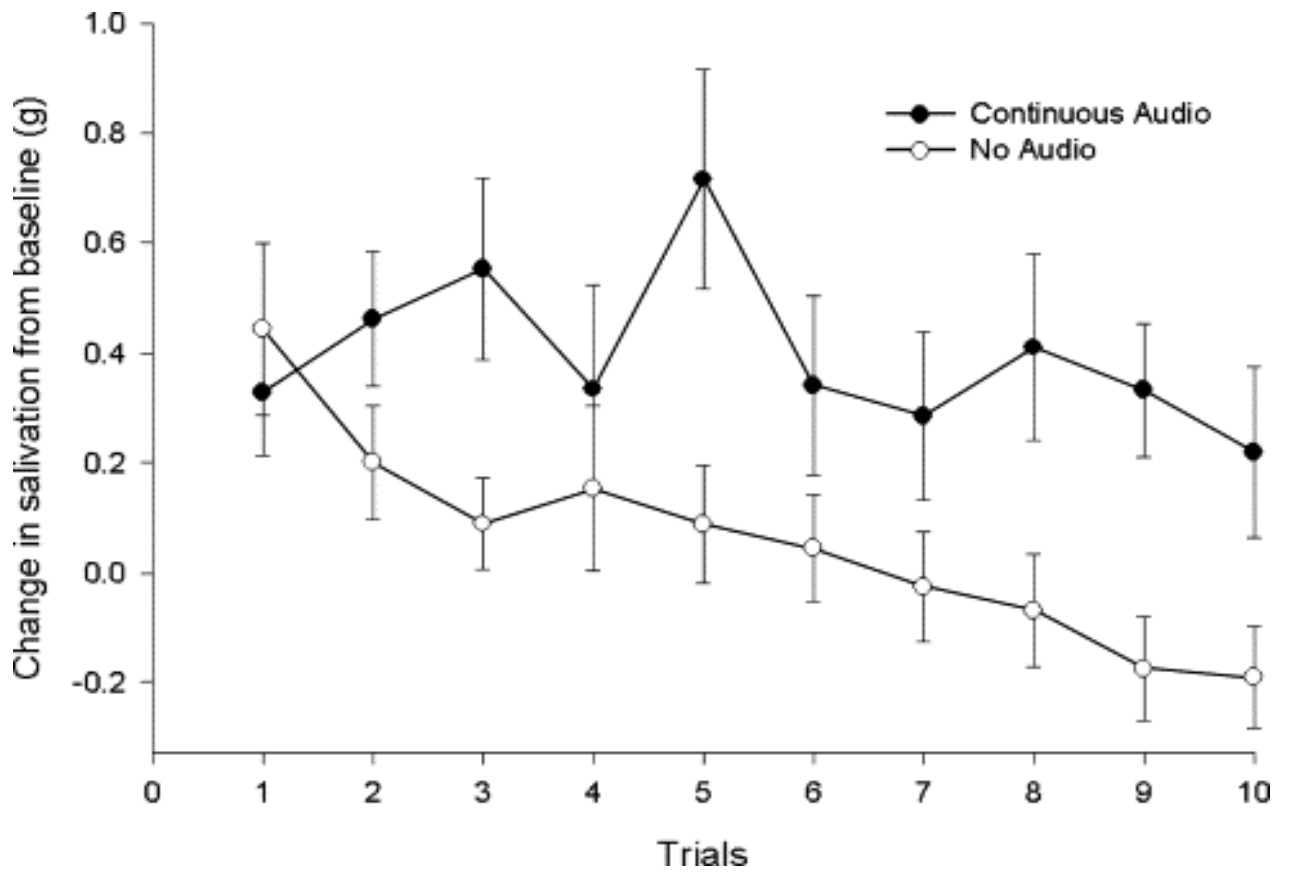
[4]From "Effects of allocation on habituation to olfactory and visual food stimuli in children" by Epstein, L.H., Saad, F.G., Giacomelli, A.M., & Roemmich, J.M., 2005. *Physiology & Behavior*, *84*, 313-319. Adapted with permission of the author.

Table 1
Review of habituation and competing environmental stimuli studies

Author	Sample	Design	Findings
Epstein, Rodefer, Wisniewski, & Caggiula (1992)	16 NW[1] adult women	Habituation measured using 10 salivation trials. Distracter condition subjects played Computer game during two minute intervals whereas control subjects sat quietly. *Test food: Lemon juice*	Control subjects habituated whereas distracter condition did not
Epstein, Mitchell, & Caggiula (1993)	30 NW adult women	Habituation measured using seven salivation trials. Participants assigned to three conditions: High attention (videogame plus mental arithmetic stressor), Low attention (video game only) or Control. Prior to the final habituation trial, participants in the distracter (High and low) conditions viewed video and/or completed arithmetic while control subjects sat quietly *Test food: Lemon juice*	Significant dishabituating effects (salivation decreased with attention task and increased when the juice was represented) of High verses low were found for salivation.

Author	Sample	Design	Findings
Mitchell & Epstein (1996)	NW adult (n = 16) restrained and (n = 16) nonrestrained women	Habituation measured using seven salivation trials. Distracter condition (half restrainers and nonrestrainers) performed Stroop task while control subjects sat quietly. *Test food: Flavored yogurt*	No significant differences between conditions
Epstein, Paluch, Smith, & Sayette (1997)	30 NW adult women	Habituation measured using 10 salivation trials. Participants assigned to three conditions: High attention, Low attention or Control. During the one minute intervals, distracter (High and low) conditions worked on a computer search task varied in attentional requirements while control subjects sat quietly *Test food: Lemon Yogurt*	High distracter condition did not habituate while the Low attention and control condition habituated over time

Author	Sample	Design	Findings
Epstein, Saad, Giacomelli, & Roemmich (2005)	42 NW children ages 8 to12 years	Habituation measured using seven salivation trials. Participants assigned to three conditions: High attention, Low attention or Control. During the one- minute intervals, distracter (High and low) conditions worked on a computer search task varied in attentional requirements while control subjects sat quietly. *Test food: Hamburger*	High distracter condition did not habituate while the Low attention and control condition habituated over time
Epstein, Saad, Giacomelli, & Roemmich (2005)	22 NW children ages 9 to 12 years	Habituation measured using 10 salivation trials. Distracter condition listened to one-minute segments of preferred audio-book while control subjects sat quietly. *Test food: Pizza*	Audio-book condition took longer to habituate

Author	Sample	Design	Findings
Temple, Giacomelli, Kent, Roemmich, & Epstein (2007)	26 NW children ages 9 to12 years	Total amount of food consumed measured. Participants assigned to three conditions: Continuous TV[2], repeated segment (1.5-minute TV clip repeated on a loop) or Control. In the distracter conditions (continuous TV and repeated segment), participants instructed to watch and eat as much as desired. Control subjects given identical consumption instructions but without any distractions. *Test food: Snack food of choice (popcorn, Doritos, chips or cheetos)*	Continuous TV condition consumed more energy and spent more time eating

[1]NW = normal weight
[2]TV = television

Environmental Effects on Childhood OW

Many researchers attribute the steep rise in obesity and OW to environmental causes (Brownell, 2002; Hill & Peters, 1998; Levitsky, 2005), and current data support a causal link between certain environmental variables and OW (Wilfley & Saelens, 2002). These variables extend from the child's immediate environment, such as parenting style (Agras & Mascola, 2005) and food portion size (Ello-Martin, Ledikwe, & Rolls, 2005) to the broader societal level, such as the influence of media (Kotz & Story, 1994; Strasburger, 2001) and built environment (man made surroundings; Sallis & Glanz, 2006). However, one area that has garnered a great deal of attention is the comparable growth of technology and rates of OW. Specifically, there is widespread speculation that the sharp increase in watching television/playing computer games is related to the corresponding rise in OW in children (CDC, 1999).

Children ages 2 to 17 years spend over six hours a day engaged in media-related sedentary behaviors--more than four of which are spent on media with a screen medium (e.g., watching television and playing video games) (Jordan, 2004). In one of the first seminal papers investigating the potential connection between time engaged in these behaviors and OW, Gortmaker and colleagues (1996) reported a strong dose-response relationship between hours of television watched and weight status. Specifically, compared with children watching 0 to 2 hours of television per day, children who watched more than five hours of television per day were found to have 4.6 times greater risk of being OW. Results published after this study have varied, with only some reporting a clear association or causal link (Epstein et al., 1995; Robinson, 1999) and others failing to find such a link (Durant, Baranowski, Johnson, & Thompson, 1994) or finding only a weak positive association (Robinson & Killen, 1995). However, in April 2006, an expert panel convened to address the relation between television viewing and weight status in children (Jordan & Robinson, 2008). Based on a review of several

cross-sectional, prospective and randomized control trials, the panel concluded that there is persuasive evidence for a relation between time spent viewing television and OW.

Marshall and colleagues (2004) recently conducted a meta-analysis on 52 studies investigating television viewing/video game use and their association with either body weight or physical activity. Out of these studies, 43 were cross sectional, 8 were longitudinal, and one was a randomized control trial. Most of the samples were from the United States ($k = 25$), and for the meta-analyses samples were divided by age (0 to 6; 7 to 12; 13 to 18 years). Analyses indicated that although time spent watching television/playing video games was consistently associated with OW, this relation was weak and likely not of clinical significance. However, few longitudinal and treatment studies were evaluated in this meta-analysis, making it difficult to examine a cause-effect relationship. The authors note that the only randomized control trial reviewed (Robinson, 1999) provided causal evidence that watching less television decreases weight in OW children.

More recently, Hancox and Poulton (2005) conducted a longitudinal study to assess the causal relationship between television viewing and BMI. They followed 1037 participants from 3 to 15 years of age and data were collected at two-year intervals (ages 3, 5, 7, 9, 11, 13 and 15). A higher amount of time spent watching television was found to be a significant predictor of OW in childhood. As with the Marshall et al. (2004) review, the effect size reported in this study was small. However, the authors note that effect sizes for dietary intake and physical activity, which are clear contributors to OW, are often lower than those for television viewing. Thus, they concluded that television viewing is an important contributor to childhood OW. In another recent longitudinal study, Jago and colleagues (2005) evaluated the same relation in children 3 to 7 years old followed for three years. Television viewing again significantly predicted BMI in children,

with predictive power strengthening with age. Finally, a recent cross-sectional study reported a significant independent association between time spent playing electronic games and OW in children in grades 1 to 3 (Stettler, Signer, & Suter, 2004).

Although the strength of the relation between television viewing and OW is still open for debate, taken together, these studies suggest that television viewing/playing video games is associated with and causally predictive of OW in Children. One possibility for this relationship is dietary intake. Indeed, several research studies have reported an association between watching television and increased dietary intake (Crespo et al., 2001; Matheson, Killen, Wang, Varady, & Robinson, 2004; Matheson et al., 2004). However, the specific mechanisms underlying this relation are not well understood. As previously discussed, one logical explanation is that television viewing/playing video games disrupts the habituation process, leading to increased consumption. To the author's knowledge, no studies have examined this hypothesis in OW children and this is one of the goals of the present study. Given the considerable amount of time children spend engaging in media-related sedentary behaviors daily, it is clearly an important area for research that may offer a new avenue of focus for treatment and prevention of OW.

Significance of Present Research & Specific Hypotheses

OW and obesity are defined by an energy imbalance in which intake is greater than the expenditure. Existing research on the treatment of OW can be supplemented with translational research—that is, basic laboratory studies exploring the interaction between the environment and biological processes that bring about dysregulated eating in OW Children. The present investigation was a translational study on habituation, a basic biological process known to influence ingestive behavior in children. The specific goal was to investigate the influence of attention-demanding activities on habituation in normal weight and OW Children. **Habituation was defined as: 1) a non-significant**

24

difference between mean baseline salivation (i.e., salivation level prior to food presentation) and the mean salivation at the final blocked trial and 2) any trial where the mean salivation level was equivalent or less than mean baseline salivation.

This study was a replication and extension of the audio-book study (experiment 2) from the Epstein et al. (2005) paper. Specifically, normal weight and OW Children were randomized to either an attention-demanding condition task or a white noise condition. Results from the Epstein et al. (2005) study clearly indicate that allocating attention interferes with the habituation process; a replication of this finding is expected. **Thus, it was hypothesized that, regardless of weight status, participants in the attention-demanding condition would not habituate to food, whereas those in the white noise condition would show a decrease in salivation across trials.**

It was currently unknown whether this finding would extend to, or possibly be more pronounced in, OW Children. However, previous research demonstrated that, in the absence of an attention task, obese adults take longer to habituate than normal weight adults (Epstein et al., 1996). In addition, a recent study (Temple et al., 2007) found that children with a BMI above the 85th percentile take longer to habituate using motivated responding than children below the 85th percentile. **Thus, the secondary hypothesis was that the difference between the attention-demanding condition and white noise condition would be more pronounced in OW participants as compared to normal weight participants. Specifically, it was expected that habituation would take longer (for both normal weight and OW children) in the attention-demanding condition compared with the white noise condition; however, the effect would be more pronounced in the OW group.**

Due to the similarities between the audio-book task and television viewing, potential implications of the present research may extend to offer insight into the basic

biological processes underlying the relation between television viewing and weight status. In particular, this research may shed light on specific mechanisms involved in increasing the amount of energy consumed within a meal—namely, attending to external stimuli while eating. This information can be translated to enhance and refine the eating-regulation component of existing treatment strategies. For example, one behavior change strategy may involve having children exclusively eat fruits and vegetables while engaging in attention demanding activities, the biological reasoning being that while engaging in those activities they will take longer to habituate and thus eat more and increase consumption of healthy foods. Further, when eating foods that are high in calories, another strategy may be to restrict access to attention-demanding activities such as watching television, playing video games or talking on the telephone. Finally, if a sub-set of children take longer to habituate, they may be at a higher risk for overeating. This information would be informative for prevention programs, as these programs could tailor their approach to treat these high-risk children accordingly. Overarching implications from this line of research include broadening our understanding of the basic processes underlying ingestion and providing innovative strategies to improve the efficacy of current treatment and prevention programs for OW.

Method

Participants

Participants were 57 children, ages 9 to 12 years. A total of 31 participants were within a normal weight range (BMI less than the 85th percentile for age and sex), and a total of 26 participants were OW (BMI greater than 95th percentile for age and sex).

Participants were recruited from the St. Louis metropolitan area using Washington University Volunteer for Health, flyers and posters. Flyers were posted at

local children's hospitals, YMCAs, elementary schools, local fast food restaurants and other local stores. Children were excluded if they: (1) were taking medications that could influence olfactory sensory responsiveness or appetite (methylphenidate); (2) had any conditions that could influence olfactory sensory responsiveness or appetite (e.g., upper respiratory illness, diabetes, ADHD); (3) were currently dieting, as this could alter responding to food cues; (4) met criteria for a current psychological disorder or developmental disability, or; (5) rated foods used in the experiment less than "moderately liked".

A total of 172 parents contacted the study with interest in their child's participation. Of this number, 103 children were excluded due failure to meet the eligibility criteria (n = 93) or failing to attend their appointment (n = 10). The remaining 69 participants were considered eligible for participation and randomized to either the audio-book (35 children) or white noise condition (34 children). Initial eligibility and randomization were determined from parent-reported weight and height obtained during the phone screen. Weight status was verified by the experimenter on the day of the experiment. However, as to avoid eliciting a reaction prior to the habituation trials, weight and height were assessed after the completion of the study. Thus, 10 participants who were randomized and completed the study were later excluded because their BMI percentile fell in the 'at-risk for OW' category. An additional two participants were excluded from the analyses [one participant was disruptive during the testing session (i.e., did not follow study protocol) and the other had eaten within three hours of the testing session]. Thus, in total, 12 participants were excluded from analyses, resulting in sample sizes of 26 children (11 OW; 15 normal weight) in the audio-book condition and 31 (15 OW; 16 normal weight) in the white noise condition. Eligible children were offered a $25 gift card to Target® in appreciation of their participation.

Measures

Height and Weight: Height (kg) was assessed using a portable stadiometer (Seca, Hanover, MD). Weight (lb) was assessed using a lithium electronic scale (Homedics, Commerce Township, MI). Height and weight were converted to BMI percentile (kg/m^2).

Hollingshead: The Hollingshead (Hollingshead, 1975) self-report questionnaire was used to gather information regarding parental education level and occupational status, with questions added regarding child ethnicity and race. Social economic status (SES) was calculated using the standard procedure detailed in the Hollingshead scoring manual. Computed scores can range from a low of 8 to a high of 66.

Modified Dutch Eating Behavior Questionnaire: The Modified Dutch Eating Behavior Questionnaire (Hill & Pallin, 1998) is a 16-item self-report questionnaire measuring dieting awareness and restraint. The Modified Dutch Eating Behavior Questionnaire is a child adaptation of the adult version of the Dutch Eating Behavior Questionnaire (Van Strien, Frijters, Bergers, & Defares, 1986). All questions have three possible responses (Never, Sometimes, Very Often) and focus around dieting awareness/restraint in (1) family members, (2) themselves, and (3) a fictitious character. The total score obtained from questions 1 to 6 was used to determine restraint, whereas the total score on items 7 to16 was used to determine dieting awareness (with higher scores indicating greater restraint and awareness); only restraint was used in the present study.

Hunger Scale Questionnaire: The Hunger Scale Questionnaire (self report; Epstein et al., 2005) was used to assess hunger immediately before and after the experiment. It

consists of a single, five-point Likert-type question ranging from "not hungry" to "very hungry".

Food Scale Questionnaire*:* The Food Scale Questionnaire (self-report; Epstein et al., 2005) was used to assess test-food liking. It consists of a single, five-point Likert-type question ranging from "do not like" to "like very much" [Note: Wording of the food scale questionnaire was adapted from the original version to refer to the different test food used (i.e., French fries) used in the present study].

Food Preferences Questionnaire: The Food Preferences Questionnaire (self-report; Epstein et al., 2005) was used to assess general food preferences. It is a 43-item questionnaire made up of five-point Likert-type questions ranging from "do not like" to "like very much".

Salivation *(objective measure of physiological response):* Whole mouth parotid salivary flow using the Strongin-Hinsie Peck method (Peck, 1959) was used to measure habituation via salivation. Each participant placed dental rolls (cylindrical, 7mm diameter, 38mm length, Richmond Dental, Charlotte, NC) under their tongue and on both the left and right sides of their mouth between the cheek and lower gum (three total rolls). Before the first trial, participants were instructed on behaviors that they should avoid while the dental rolls were in their mouth (e.g., swallowing saliva, chewing, moving the tongue).

Food was presented during one-minute intervals that were each followed by one-minute intertrial intervals. As detailed below, food was heated to enhance olfactory

stimulation and held three inches from each participant's nose. After each food presentation, dental rolls were placed by the participants into a plastic bag. The same procedure was repeated for a total of 11 trials. After the experiment, salivation was measured by pre- and post-weights of dental rolls to 0.001 grams on a Balance Adventurer™ Pro Scale AV213 (Ohaus, Pine Brook, NJ).

Olfactory Functions Test: The Olfactory Functions Test (Epstein et al., 2005) was used to determine if participants had adequate olfactory functioning. This task involved having participants close their eyes and report the distance from which he/she could smell an alcohol pad, starting at a distance of 29 centimeters.

Procedure

Interested participants were first contacted by phone. During this phone call, a brief phone screen was administered with the parent to determine the child's eligibility. If eligible, the family was scheduled for an experimental session and the arrangement for obtaining consent was made as follows: Consistent with IRB guidelines, written informed consent from both custodial parents or legal guardian(s), and written informed assent from the minor participating in the research was obtained. To secure consent from both parents, the consent document was sent by postal mail or by electronic mail (email), and parents were asked to bring this signed document with them on the day of the experiment. Alternatively, parents who could not be present on the day of testing were given the option of (1) faxing the signed consent or (2) sending the signed consent via postal mail prior to the scheduled appointment for the experiment (in this case, a self-addressed stamped envelope in which to return the signed consent was provided). The

assent form was collected immediately prior to initiating the experiment. Finally, participants were instructed to refrain from eating the test food (i.e., French fries) on the day of testing and to fast for three hours prior to the experimental session.

The study was conducted at the Psychological Service Center, located on the West Campus of Washington University. After obtaining the signed consent and assent forms, parents were asked to complete the Hollingshead questionnaire, and children were asked to complete two subjective ratings (i.e., Hunger Scale Questionnaire, Food Scale Questionnaire). Next, children were asked to name the specific foods, as well as the time (hour and minutes) and amounts of each food, that they had consumed on the day of testing. This list was used to confirm that the child had not eaten during the three hours prior to testing, and to confirm that the test food had not been eaten on the day of testing.

Participants were stratified by sex and randomly assigned to one of two conditions: Audio-book or White Noise. Randomization was determined using a random number generator (http://www.randomizer.org/form.htm), unsorted and ranging from 1 (Audio-book) to 2 (White Noise). Prior to beginning the study, a recruitment goal of 60 participants was set. Based on this goal, four lists (OW, Male; OW Female; Normal Weight, Male; Normal Weight, Female) of 15 random numbers each (ranging from 1 to 2 per list) were generated based on weight status (OW or normal weight) and sex (male or female).

As with the Epstein et al. (2005) study, the Audio-books for the present study were three selections from a series of Bunnicula children's audio-books and were used as the attention task during the study. Children in the Audio-book condition were given brief descriptions of each story and were asked to choose the story they most preferred.

The experiment consisted of 11 habituation trials (i.e., 2 habituation practice trials followed by 9 test trials). Each habituation trial consisted of the experimenter bringing a food stimulus (i.e., 37 grams of Burger King French fries served on a 9-inch plate and heated on high in the microwave oven for 25 seconds in a separate room) into the experimental room and having the participants smell and look at the food for one minute while it was held approximately three inches beneath the participants' nose. After each trial, salivation was measured using the Strongin-Hinsie Peck method (Peck, 1959) as described earlier. In addition, a high efficiency particle-arresting (HEPA) filter was used to reduce the likelihood that lingering odors from the test food would affect results between trials and between participants.

Total time for participants was approximately 1.5 hours. Between each habituation trial, a one-minute intertrial interval occurred. During this intertrial interval, children in both conditions listened to an auditory stimulus using headphones. Participants in the audio-book condition listened to a compact disc of their selected audio-book for sequential 1-minute presentations. These participants were asked to pay attention to the audio-book and were told that they would be asked questions about it at the conclusion of the experiment. In the white noise condition, participants did not attend to an audio-book and instead listened to a compact disc of white noise and sat quietly for one-minute.

After the experiment concluded, all participants filled out the Hunger Scale Questionnaire again and completed the Modified Dutch Eating Behavior Questionnaire and Food Preferences Questionnaire. Participants in the audio-book condition also answered multiple choice questions (2-3 questions depending on the audio-book selected) based on the story they heard to verify they had attended to the task; these questions were written at a third-grade level. Prior to the conclusion of the study, height

and weight were measured, and the Olfactory Functions Test was conducted. At the conclusion of the study, all children were given a $25 gift card to Target® and debriefed about the purpose of the study.

Analytical Plan

Data were double-entered by two individuals, and all discrepancies were checked and corrected by the author. Analyses were conducted using SPSS version 14.0 for Windows; all tests were two-tailed, and statistical significance was set at $p < .05$ except when otherwise noted below.

First, baseline differences were examined by weight status (OW vs. normal weight) and condition (audio-book vs. white noise). Group/condition differences were examined using independent sample t-tests for continuous variables (restraint score, BMI percentile, age, food consumption, SES, test food liking, subjective hunger before and after the testing session), and chi-square tests for categorical variables (child race, child ethnicity, sex, family income range, parent marital status). Additionally, independent samples t-tests were used to compare responses by weight status, sex, race/ethnicity, and age on the Food Preferences Questionnaire and the Modified Dutch Eating Behavior Questionnaire.

Studies investigating salivation patterns generally analyze this type of data using individual salivation trials or individual salivation trials blocked into sets (two or more sequential trials averaged together to create a composite) as the dependent variable in the analyses. Typically, habituation studies block the data into sets for ease of presentation and/or to reduce variance. In the present study, salivation data was analyzed both ways. Specifically, for the primary analyses blocked salivation trials was entered in as the within subject variable. Data for blocked trials were represented in sets

by calculating the mean of two consecutive trials[5] (e.g., mean of trial 1 and 2 = blocked trial 1; mean of trial 3 and 4 = blocked trial 2), resulting in a total of five blocked trials. As a secondary method of analyses, all statistical tests were rerun using individual salivation trials (i.e., 1-11) as the within subject variable.

As a first step, paired sample t-tests between the baseline and initial trials/blocks were conducted to determine if there was a reliable increase in response to the food stimulus. Next, to test explore the pattern of responding across groups and conditions, salivation levels were analyzed using ANCOVA and repeated measures ANCOVA. For the repeated measures ANCOVA, weight status and condition were entered as the between subject factors and blocked trials as the within-subject repeated measures factor. Variables that differed significantly by group/condition and/or were significantly related to outcome were entered as covariates. Planned simple contrasts were used to determine if habituation had occurred at the final trial. For the repeated measures ANCOVA, simple contrasts were used to check for habituation. Specifically, habituation was determined by comparing mean baseline salivation (i.e., salivation level prior to food presentation) with the mean salivation at the final blocked trial; means that did not significantly differ from one another indicated habituation. In contrast, 'not habituating' was defined as a significantly higher mean (i.e., increased salivation as compared with the baseline salivation mean) at the final blocked trial.

Lastly, survival analyses using the Kaplan–Meier estimate of survival were conducted to test for the hypothesized differences in the *rate of habituation* among groups and conditions. For these survival analyses, habituation was defined at the individual level as the specific trial on which salivation level was equal to or below baseline salivation. For each trial, habituation was dichotomized (i.e., habituated or not

---

[5] The final blocked trial was a composite of trials 9, 10 and 11.

habituated) and once an individual was determined to have habituated, all further trials were marked as such.

Results

Preliminary Analyses

Participants were 28 males and 29 females ages 9 to 12 years. The white noise condition consisted of 26 children (11 OW; 15 normal weight), 57.7% were female. The audio-book condition consisted of 31 children (15 OW; 16 normal weight), 45.2% were female. The average child was 10.5 $\pm$ 1.2 years of age. Average baseline salivation was 1.574 $\pm$ 1.149 g, and participants rated the test food (French fries) as moderately to highly liked on a Likert-type scale from 1 to 5 (4.5 $\pm$ 0.8). Normal weight participants ($n =$ 31) had an average BMI of 17.6 $\pm$ 1.4 kg/m^2 and BMI percentile of 52.3 $\pm$ 19.1. OW participants ($n = 26$) had an average BMI of 28.9 $\pm$ 4.2 kg/m^2 and BMI percentile of 98.0 $\pm$ 1.3. The sample was 28.1% Non-Hispanic African American, 68.4% Non-Hispanic Caucasian, and 3.5% Non-Hispanic mixed race. According to parent report of marital status, 56% were married, 22% were single, 13.6% were divorced, and 1.7% were widowed. In terms of parental education level, 45.8% of mothers and 33.9% of fathers had completed college or university. Parents' self-reported household income levels varied: 10.7% reported an annual income below $30,000; 57.1% reported an income between $30,000 and $100,000; and 32.1% reported an income level over $100,000 (Table 2).

A total of 31 participants were assigned to the audio-book condition; they completed a quiz at the end of the study to verify that they had attended to their chosen story. Of the 31 participants, 27 (87%) answered all questions correctly; 3 (9.6%) missed

Table 2

Baseline Demographics in Normal weight and OW Participants

Descriptive Variables	Group			Statistic		
	Total (N=57) $M(SD)$	NW[1] (n=27) $M(SD)$	OW[2] (n=30) $M(SD)$	t	p	ES[3]
Family Socioeconomic Score (Hollingshead)	51.7(11.3)	53.7(11.1)	49.2(11.4)	1.39	.170	.39
	%(n)	% (n)	%(n)	X^2	p	
Ethnicity						
Non-Hispanic African American	28.1(16)	7.4(2)	46.7(14)	13.98	.001	.49
Non-Hispanic Caucasian	68.4(39)	92.6(25)	46.7(14)			
Non-Hispanic Mixed Race	3.5(2)	0(0)	6.7 (2)			
Parental Marital Status						
Married	58.5(31)	76.0(19)	42.9(12)	12.26	.007	.48
Single	24.5(13)	4.0(1)	42.9(12)			
Divorced	15.1(8)	20.0(5)	10.7(3)			
Widowed	1.9(1)	0(0)	3.6(1)			
Parental Income Range						
<$30,000	10.7(6)	3.7(1)	17.2(5)	7.28	.026	.36
$30,000-$100,000	57.1(32)	48.1(13)	65.5(19)			
>$100,000	32.1(18)	48.1(13)	17.2(5)			

[1] Normal weight [2] Overweight [3] Effect Size

one question and 1 (3.2%) missed two questions. Participants that missed questions were retained in the analyses as excluding them did not change the outcome.

When comparing the sample by weight status, by definition, the OW group had a significantly higher BMI percentile, $t = -13.10$, $p < .0001$. There were no group differences in test food liking, age, or total amount of food consumed, however the OW group reported a significantly higher restraint score, $t = -3.74$, $p = .0004$, significantly lower sense of smell, $t = 2.39$, $p = .02$, and lower hunger ratings before, $t = 2.41$, $p = .014$, and after, $t = 1.86$, $p = .033$, the study (Table 3). In addition, normal weight children were significantly more likely to be Caucasian, χ^2 (2, N = 56) = 13.98, $p < .001$, and have parents who were married, χ^2 (2, N = 57) = 5.28, $p = .022$, with a higher yearly income range, χ^2 (2, N = 57) = 7.28, $p = .026$, as compared with the OW children. When comparing the sample by condition, the white noise condition had a significantly lower sense of smell, $t = 2.06$, $p = .044$ and a higher preference for the test food, $t = -2.47$, $p = .017$; no other significant differences were found on any baseline or demographic variables (Table 4).

Although level of restraint, sense of smell (Olfactory Functions Test), hunger ratings before and after the study, parental marital status, child's race and family income were significantly different between condition and group at baseline (see above), exploratory analyses were conducted (Grouin, Day, & Lewis, 2004; Raab, Day, & Sales, 2000) to determine which variables were significantly related to the outcome variable. Only baseline salivation ($p = .001$), Hunger Rating 2 ($p = .042$) and the Olfactory Functions Test ($p = .002$) were significantly associated with outcome and were thus included as covariates in all further analyses in the present study.

In terms of the salivation data, paired t-tests confirmed that there was a significant increase in responding to the food stimulus from the baseline blocked trial to

Table 3.

Baseline differences by weight status

Variable	Group				Statistic		
	Normal weight		Overweight		t	p	ES[1]
	M	*SD*	*M*	*SD*			
Restraint	7.6	4.5	11.8	3.3	-3.74	*.001*	*.90*
Hunger rating1	4.3	0.6	3.7	1.1	2.41	*.019*	*.90*
Hunger rating2	4.6	0.6	4.1	1.0	1.86	.068	.74
Test food liking	4.3	.8	3.9	.8	1.70	.094	.48
BMI Percentile	52.3	19.4	98.0	1.3	-13.10	*.001*	2.30
Age (Years)	10.4	1.1	10.7	1.2	-.836	.407	.22
Food consumed (g)	148.8	61.0	152.4	56.2	-.202	.841	.05
Olfactory Test (cm)	26.4	3.5	23.1	7.0	2.39	.020	1.00

[1] Effect Size

Note: Hunger ratings (1 and 2) and Test food liking scores can range from 1 to 5;
Restraint score can range from 6 to18

Table 4.

Baseline Differences by Condition

Variable	Condition				Statistic		
	Audio-book		White Noise		*t*	*p*	ES[1]
	M	*SD*	*M*	*SD*			
Restraint	9.6	4.2	10.1	4.7	-.546	.587	.14
Hunger rating 1	3.9	1.0	4.1	0.9	-.395	.694	.10
Hunger rating 2	4.3	0.8	4.4	0.9	-.101	.920	.03
Test food liking	3.9	.7	4.4	0.8	-2.470	*.017*	*.60*
BMI Percentile	77.0	24.7	76.0	28.0	.184	.855	.04
Age in Years	10.6	1.2	10.5	1.2	.032	.975	.00
Food consumed (g)	162.2	56.4	135.4	58.0	1.677	.100	.48
Olfactory Test	26.2	4.2	23.2	6.9	2.024	*.048*	.43

[1] Effect Size

Note: Hunger ratings (1 and 2) and Test food liking scores can range from 1 to 5;
Restraint score can range from 6 to 18

the first block, t = -2.4, p =.02, and from the baseline blocked trial to the first individual

trial, t = -2.03, p = .05.

Primary Analyses: Summary of hypotheses and findings

<u>*Hypothesis I:*</u> It was hypothesized that, regardless of weight status, participants in the audio-book condition would not habituate to food, whereas those in the white noise condition would habituate across trials. In analytic terms, a two-way interaction between trials and condition (audio-book vs. white noise) was predicted. Repeated measures ANCOVA was used to evaluate the pattern of responding between the audio-book and white noise conditions whereas, survival analysis was conducted to test for differences between conditions in the rate of habituation over blocked trials.

<u>Finding:</u> The expected two-way interaction between condition and blocked trials (Figure 4) was non-significant, $F(4,46)$ = 1.25, p = .31, η^2 = .08. Results using survival analysis were also non-significant (χ^2 = .418, p = 0.52, φ = .09). Thus, salivation patterns and rate of habituating (regardless of weight status) between the audio-book and white noise conditions were not significantly different from one another.

<u>*Hypothesis II:*</u> The secondary hypothesis was that the difference between conditions (audio-book vs. white noise) would be more pronounced in OW participants as compared with normal weight participants. In analytic terms, a 3-way interaction was predicted among trials, condition (audio-book, white noise) and group (OW, normal weight). Repeated measures ANCOVA was used to evaluate the pattern of responding between conditions and groups whereas, survival analysis was conducted to test for differences among conditions and groups in the rate of habituation over blocked trials.

<u>Finding:</u> The expected three-way interaction among condition, weight status, and blocked trials (Figure 5) was non-significant, $F(4,46)$ = 0.04, p = .77, η^2 = .02. Results

Figure 4. Data Plotted as a Function of a Two-Way Interaction between Condition and

Blocked Trials (Hypothesis I)
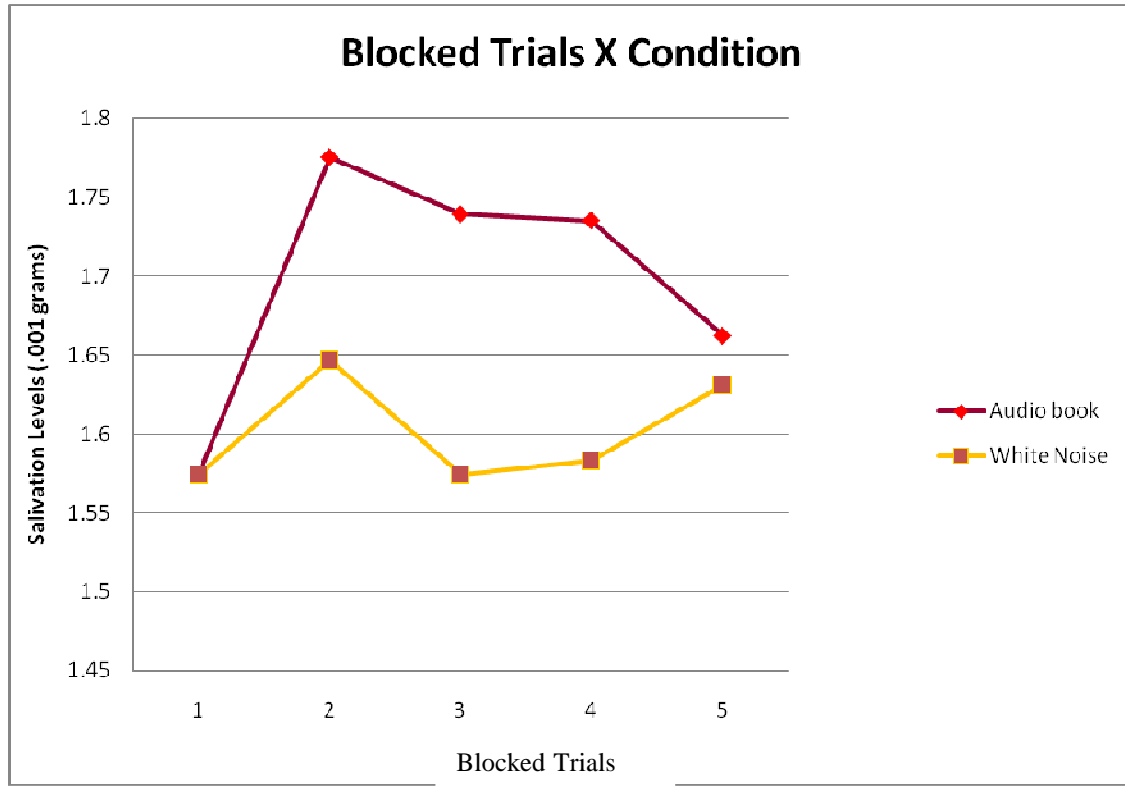
Figure 5. *Data Plotted as a Function of a Three-Way Interaction* among Condition,

Weight Status and Blocked Trials
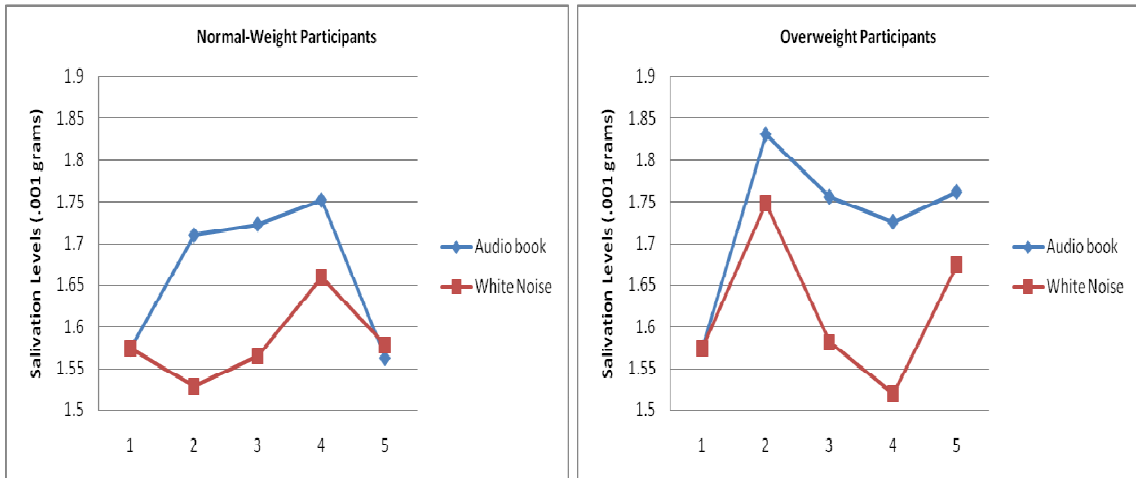
using survival analysis was also non-significant (χ^2 = 1.002, p = 0.32, φ = .17). Thus, salivation patterns and rate of habituating for the audio-book and white noise conditions did not differ by weight status.

Exploratory Analyses

To further evaluate the aims of the study (hypothesis I and II), an ANOVA was run with condition and condition by weight status as the respective fixed factor and trial of habituation as the dependent variable. These tests confirmed the above findings with non-significant results for both hypotheses (p's > .1). In addition, to better understand hypothesis II, separate ANCOVA analyses with blocked trial as the dependent variable, and all previous blocked trials as covariates were run for normal weight and OW participants. Findings revealed significant differences between conditions for each blocked trial (p's < .001).

There was a significant main effect of blocked trials, $F(4,46)$ = 3.71, p < .011, and a significant interaction between weight status (OW vs. normal weight) and blocked trials, $F(4,46)$ = 2.83, p = .035, η^2 = .19. Planned simple contrasts revealed that regardless of condition (audio-book vs. white noise), at the final trial, the normal weight group habituated, $F(1,22)$ = 2.58, p = .122, whereas the OW group did not, $F(1,25)$ = 4.71, p =.039 (See Figure 6).

Secondary Method of Analyses

The same overall pattern of results (Hypothesis I & II and Exploratory analyses) was found when using individual trials in the survival analyses and as the repeated measure. Specifically, non-significant results (ANCOVA and Kaplan-Meier survival analyses, respectively) were found for the above mentioned hypothesis I, $F(10,41)$ = 0.68, p = .74, χ^2 = .427, p = 0.51, and II, $F(10,41)$ = 0.37, p = .95, χ^2 = 1.130, p = 0.29. A

Figure 6. Two-Way Interaction between Weight Status and Blocked Trials
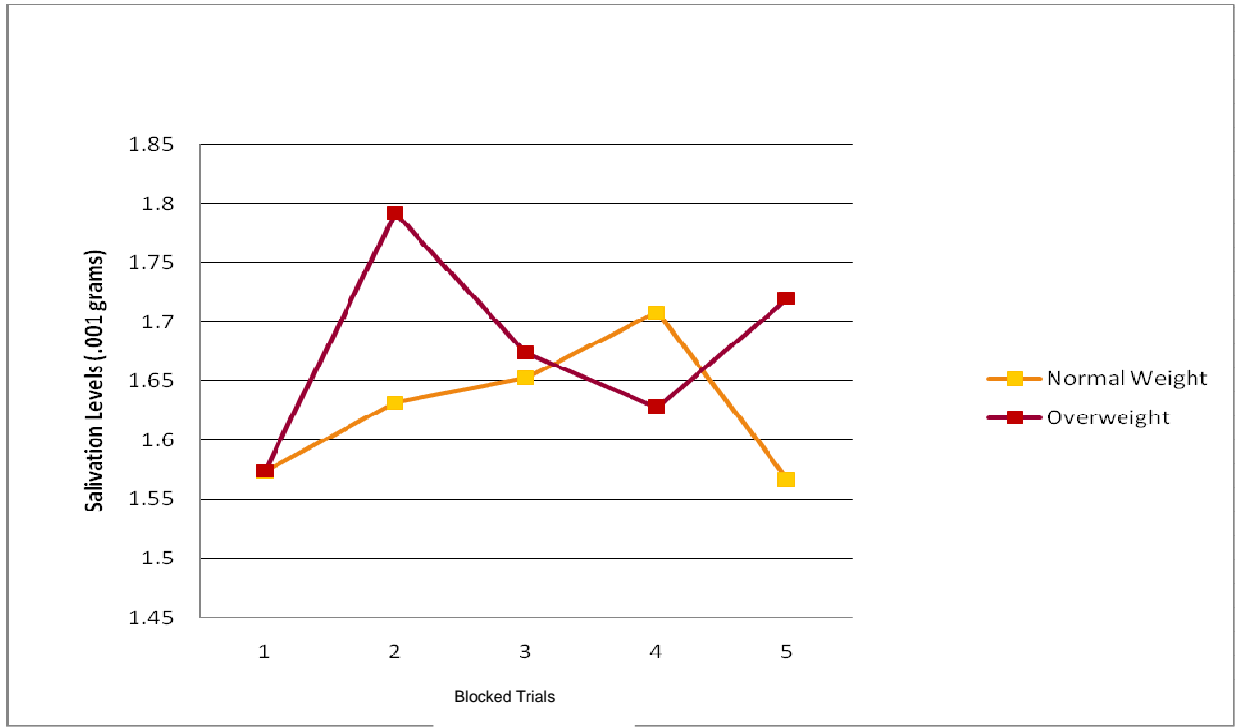
significant main effect of trials was found, $F(10,41) = 3.66$, $p = .001$, and a significant interaction between weight status and trials, $F(10,41) = 2.08$, p = .048. Based on simple contrasts, at the final trial the normal weight group habituated, $F(1,23) = 0.01$, $p = .93$, whereas the OW group did not, $F(1,26) = 3.32$, $p = .07$, (trend level; finding approached significance).

Additional Findings

Dietary Restraint (Modified Dutch Eating Behavior Questionnaire: OW children were significantly more restrained, $t = -3.74$, p < .0001, and aware of dieting, $t = -2.31$, p = .025, as compared to normal weight children. No sex, age or race differences were found.

Food Preferences (Food Preferences Questionnaire): Food items were coded into unhealthy (e.g., ice cream, bacon, Doritos) or healthy (e.g., English muffin, peas, baked potato) categories based on nutritional content. No race differences were found for preference for unhealthy foods; however, African American children reported a significantly higher preference for healthy foods as compared to Caucasian children, $t = 2.09$, $p = .04$. No significant differences were found between normal weight and OW children on healthy, $t = 0.75$, $p = 0.46$, or unhealthy, $t = 1.73$, $p = 0.09$, food preferences. Similarly, no sex or age differences were found.

Discussion

Summary of Findings

This study sought to explore one mechanism of the interaction between the environmental (media influence on attentional processes) and biological (habituation measured via salivation) effect on OW in children. The primary objectives of the study were to determine if salivation patterns varied based on weight status and/or allocating

attention to a task. Results demonstrated that salivation patterns varied by weight status but not by task condition. More specifically, it was found that across conditions, normal weight children habituated to food cues whereas OW children did not. These findings suggest that normal weight and OW children have different biological responses to food.

Hypothesis I

To determine whether salivation patterns vary when attending to a distracter task (regardless of weight status; Hypothesis I), participants were randomly assigned to either listen to an audio-book or sit quietly (listening to white noise) during each intertrial interval of the experiment (i.e., the one-minute period between each of the nine salivation trials following the two baseline trials). Based on the Epstein et al. (2005) findings, it was expected that children listening to white noise would habituate to the food cues (i.e., salivation would return to baseline level) whereas those attending to the audio-book would not. However, the results from the present study did not replicate this previously reported finding and, instead, showed a non-significant difference in salivation patterns between the white noise and audio-book conditions [ES (η^2) =.08]; these findings suggest that in this sample, paying attention to a task while eating did not impede the habituation process.

The lack of effect of the attention condition is especially interesting, given that six previous studies have demonstrated the effect (Epstein, Mitchell, & Caggiula, 1993; Epstein et al., 1997; Epstein, Rodefer, Wisniewski, & Caggiula, 1992; Epstein et al., 2005; Mitchell & Epstein, 1996; Temple et al., 2007). The discrepancy in findings may be due to several factors. First, the testing environments were different. All of Epstein and colleagues' studies were conducted in a controlled feeding laboratory using HEPA air purifiers with an air delivery system that circulates new air through the room 10 times per

hour. In contrast, the present study was conducted in a conventional therapy room converted for a lab experiment, which may have introduced additional variance.

However, several measures were taken to ensure that the testing environment was as controlled as possible. For example, the experimenter was trained in the specifics of the habituation methodology by Dr. Jennifer Temple in the laboratory of Dr. Leonard Epstein at the University at Buffalo. In addition, as with the Epstein laboratory, a HEPA air purifying system (containing a carbon, permanganate and zeolite filter) was used in all trials to remove airborne odors. Further, the food stimulus was promptly removed from the testing room after each trial, to isolate smell and eliminate the possibility that the aroma or presence of the test food could influence additional trials. Lastly, the testing room was selected as it was directly next to a kitchen facility, which ensured the timely delivery of the food stimulus. Other precautionary measures included prerecording four compact discs (CDs; three Audio-books—one for each story selection; one white noise) to ensure perfect timing of habituation and intertrial intervals trials. White noise and audio-book segments were divided into timed one-minute interval segments and each habituation trial began and ended with pre-recorded instruction to place and remove the dental rolls from the mouth. Given these preventative measures, it seems reasonable to assume that the testing environment controlled for enough variance to detect an effect.

Another possibility is that the audio-book condition did not attend to their respective stories, cancelling out the attention component of the experiment. However, this explanation is also unlikely as all participants in the audio-book condition completed a quiz at the end of the study to verify that they paid attention and 87% answered all questions correctly (i.e., of the 31 participants, 87% answered all questions correctly; 9.6% missed one question and 3.2% missed two questions). Furthermore, the audio-book condition did not habituate which is consistent with attending to a task.

47

One clear advantage of the current study's testing environment over the traditional laboratory settings is that it offers novel information on the external validity of the habituation effect. Specifically, whether the effect of attention on habituation translates to the home environment and whether it can explain the relation between television viewing and weight status. Based on the findings of this study, allocating attention to a task in relatively uncontrolled conditions (i.e., a more naturalistic setting) does not disrupt the habituation process. Given this information, it is worth exploring other valid explanations for the association between television viewing and weight status.

Television viewing is typically a sedentary behavior that replaces vigorous activity (Caroli, Argentieri, Cardone, & Masi, 2004) and sedentary behavior predicts obesity (Hu, Li, Colditz, Willett, & Manson, 2003; Tucker & Bagwell, 1991). Further, many researchers have documented the rapid increase in the number of junk food commercials (advertising foods high in fat, salt, sugar; soda and sugared beverages; fast food and frozen foods) targeting both parents and children by using popular cartoon characters and by emphasizing the "nutritional value" of their products (Caroli et al., 2004). A recent study evaluating the effect of television advertisements for food on children found that OW children have a heightened awareness of food related commercials as compared to normal weight children and that exposure to food related advertisements increases food intake among normal weight and OW children (Halford, Gillespie, Brown, Pontin, & Dovey, 2004). Based on these findings, it is not surprising that many studies have reported an association between watching television and dietary intake (Crespo et al., 2001; Matheson et al., 2004a; Matheson et al., 2004b). In this way, the link between OW and television viewing may simply be related to an increase in sedentary behavior (decrease in physical activity) coupled with the persuasive junk food

advertisements encouraging poor food choices as opposed to a disruption in the habituation process.

Finally, in our current technology-based environment, children and adolescents grow up attending to multiple stimuli. In fact, approximately 30% of children live in households in which television or other screen media are present all or most of the time, an additional 30% regularly view television during meals and approximately 75% have a television in their bedroom (Jordan & Robinson, 2008). Thus, another possibility is that the audio-book task, in a more natural testing environment, did not "distract" the children sufficiently to interfere with the habituation process.

Hypothesis II & Exploratory Analyses

The second aim of the study was to explore whether salivation patterns, when allocating attention to a task, varied by weight status (three-way interaction; Hypothesis II). Although this three-way interaction among weight status, attention and blocked trials was found to be non-significant, results did show a significant weight effect. In particular, regardless of attention condition, normal weight children habituated to food cues at the final trial whereas OW children did not. Closer inspection reveals that the trajectory for the OW and normal weight children at the final two trials were opposite to one another, with the OW group shifting dramatically upwards (increased salivation) and the normal weight children shifting dramatically downwards (decreased salivation) (See Figure 6 in the Results Section). This pattern appears to suggest that if the trials had continued, the normal weight group would continue to decrease and the OW group would continue to increase.

The salivation pattern (increasing salivation over trials) observed in the OW group is similar to patterns previously found for obese women and women with bulimia nervosa (Epstein et al., 1997; Wisniewski et al., 1997). In all three cases, salivation

patterns generally increase over time for the target group (obese women; OW children; women with bulimia nervosa) with no habituation and decrease, indicating habituation, for the control group (normal weight, no eating disorder symptoms), suggesting that conditions associated with certain eating patterns may display irregular patterns of habituating to food. However, this trend of findings is inconsistent with a recent study (Temple et al., 2007) in which OW children were reported to have habituated. In the Temple et al. (2007) study, habituation was measured using a motivated responding computer task in children above (at-risk for OW and OW) and below (normal weight) the 85th BMI percentile. All children habituated, but the at-risk for OW children took longer to do so, which differs with the present study, in which the OW children did not habituate.

The conflicting results between the present study and the Temple et al. (2007) study are concerning given that both attempted to measure habituation in OW children. However, three key differences may help elucidate the discrepancies. First, in terms of the study design, the Temple et al. study ran for 10 habituation trials whereas the current study ran for 9 (i.e., 2 baseline trials followed by 9 habituation trials). It is possible that if the current study had increased the number of trials, that the OW group would have habituated as well.

Second, in terms of the population, the Temple et al. (2007) study compared children above the 85th percentile (at-risk for OW and OW) with those below the 85th percentile (normal weight), whereas the present study compared children below the 85th (normal weight) and above the 95th (OW). Stated differently, the Temple et al. study included children at-risk for OW (85th to 95th), a less severe population. Thus, the slowed habituation patterns may be due to the influence of the at-risk participants. Although there is no participant breakdown on the number of OW versus at-risk for OW participants in Temple's study to verify this theory, merging the literature together

50

reveals a potential pattern wherein as weight increases, a more irregular response to food may develop , as follows:

NW habituate → At-risk for OW habituate slower than NW→ OW do not habituate

Another difference between these studies relates to the mode of measuring habituation (saliva collection vs. motivated responding). Salivation production is defined as a reflexive physiological response, whereas motivated responding is a complex behavioral response. Based on previous findings in animal and human data, Temple and colleagues argue that habituation principles can be extended to explain motivated responding (Melville, Rue, Rybiski, & Weatherly, 1997; Temple et al., 2006). Specifically, they report that when a food is repeatedly presented, salivation patterns (i.e., increases and decreases, response to novel food) and motivated behavior patterns (i.e., working for food reinforcement) respond similarly (e.g., work is the hardest initially, followed by a decrease over time; work level returns to the highest point when a new food is introduced). They further argue that this parallel response to food suggests that the same habituation process underlies both processes (Temple et al., 2006). Despite the similarities between motivated responding and salivation, distinct differences remain in terms of how the data are obtained (saliva weighed on cotton rolls vs. responses entered onto a computer) and the resulting interpretations that can be made from each. Specifically, given that motivated responding is a proxy for salivation, results using this process are likely less informative of the physiological processes involved in habituation to food cues.

Taken together, there appears to be mounting evidence that increased weight and other eating-related disturbances are associated with altered salivary responses to food. To the author's knowledge, three habituation studies using OW child and adult

samples (including the present investigation) have been conducted to date. In all of them, irregular habituation patterns in the OW samples were found compared to their normal weight counterparts suggesting that there is something distinct occurring in the biological response OW individuals have to food cues.

Interpretation of Findings

Salivary flow is a valid measure of hunger (Wooley & Wooley, 1973). Increased salivation is associated with hunger and the beginning of a meal[6] (Nederkoorn et al., 2000), whereas decreased salivation is linked with satiation[7] (Swithers & Hall, 1994) and the termination of a meal (Wisniewski et al., 1992). In other words, this literature suggests that an average individual will salivate when hungry, which often coincides with the beginning of a meal. Once the individual is no longer hungry, salivation levels tend to decrease and the meal typically ends.

Applying the above salivation literature to the findings in the present study suggests that over the course of a typical meal, normal weight children become satiated and discontinue eating (habituation), whereas OW children continue eating (lack of habituation), likely leading to a surplus of energy intake. If this lack of habituation response among OW children occurs with all or even most eating episodes, it may serve as a possible causal or maintaining factor in their OW status and provide us with valuable physiological information about why these individuals maintain a positive energy balance. This application of the literature can also be reasonably applied to previous habituation findings (Epstein et al., 1996; Wisniewski et al., 1997) and is consistent with each respective diagnosis (obesity, OW, bulimia nervosa). Specifically,

---

[6]The repeated trials over the course of this experiment are best interpreted as measurements taken throughout the course of a meal or eating episode (e.g., dinner, snack).

[7] The quality or state of being fed to or beyond capacity

greater energy intake is typically associated with higher weight status and/or bingeing episodes.

Strengths and Limitations

Strengths of the current study include a large and racially diverse sample of children. Other strengths include stratifying the sample by sex, the relative care taken to have controlled conditions, and the novel application of a habituation paradigm in a sample of OW children. In addition, the experimenter was trained in the specifics of the habituation methodology by Dr. Jennifer Temple in the laboratory of Dr. Leonard Epstein at the University at Buffalo.

Limitations include a testing room lacking the sophistication of a true feeding laboratory. As previously noted, the salivation patterns reported in the present study were more variable than those reported by Epstein et al. (1992, 1997, 1993, 2005). It is likely that this variability is due to the difference between testing conditions in a controlled setting versus a more real-life setting. However, this limitation is also a strength of the study, as finding a significant weight effect in this uncontrolled environment testifies to the likelihood that it is indeed a real phenomenon.

Summary & Implications

OW in children and obesity in adults has increased dramatically over the past two decades. There are numerous medical, psychosocial and economic consequences associated with OW. In addition, child OW is a known risk factor for the development of adult obesity and increases the risk of adult morbidity and mortality independent of adult obesity status. Given these consequences, risk factors, and the potential health benefits resulting from successful treatment, the WHO (2003) recently declared successful long-term treatment of childhood OW as the most promising direction to address this

epidemic. Although there are effective short-term treatments available for OW children and adolescents (FBT; reviewed in detail in introduction), relapse rates remain high. To best improve the current treatment options, translational research exploring the underlying causal and maintaining factors of OW are needed. Only then can we apply the knowledge from these basic studies to design optimal treatments to both prevent and cure OW.

OW children are consuming energy well beyond their physical needs. Results from the present study suggest that this overconsumption may be related to their biological response to food. In particular, the salivation patterns found in this study indicate that OW children eat past the point of satiety whereas normal weight children terminate eating when sated. This salivation pattern may be an early marker of individuals who go on to develop OW and/or other eating disturbances. In addition, these findings raise the possibility that OW children may not be able to rely on their internal signals of hunger and satiety to regulate their food intake which, in turn, could help to explain the refractory nature of this condition.

Future Directions

Habituation studies. Future habituation studies using in vivo tasks or settings are needed to further evaluate the generalizability and applicability of these results to naturalistic settings. For example, simulating an actual meal and testing salivation levels in normal weight and OW samples pre and post feeding would be an informative test of the validity of a weight effect in a more natural environment. It would also be useful to identify any other potential mediating/moderating factors that may impede the habituation process. Variables identified by existing studies include types of foods (Myers & Epstein, 2007), the variety of foods available (Myers & Epstein, 2002), and weight status (Epstein et al., 1996; Temple et al., 2007; current study). Other factors to

possibly consider include stress level (cortisol) and mood immediately prior to food consumption.

To determine if the lack of habituation observed in OW children is indicative of an inability to biologically regulate food intake, more replication of habituation studies with OW samples is required. In addition, assessing the strength of the relation between objective (blood glucose, stomach distension) and subjective measures of hunger and satiety (self-report questionnaires) are needed. Lastly, a more extensive habituation study that would allow an objective exploration of treatment response and differential outcome is warranted. For example, an ideal study would randomize OW children to a standard weight loss treatment (e.g., FBT) or control group. Habituation patterns assessed at baseline, post treatment and follow-up could test whether baseline patterns predict weight status at post treatment (weight loss) or follow-up (weight-loss maintenance). If OW children (or a subset of these children) are indeed unable to regulate their hunger and satiety cues, a stronger focus on portion control may be a useful strategy to improve prevention and treatment programs.

Causal or maintaining mechanisms. A major gap in the literature is in understanding the causes of the habituation patterns observed in OW children and disordered eating populations. Salivation is just one of many responses (physiological, endocrine and autonomic) of the digestive system initiated during the cephalic phase response (CPR). Several hunger and appetite regulating hormones (i.e., insulin, leptin, cholecystokinin [CCK] and grehlin) and central neurotransmitters are also involved. Interestingly, these hormones are associated with some of the key habituation associations: -- weight status, satiation and meal termination (Power & Schulkin, 2008). Given this overlap in associations, it seems reasonable to conjecture that one or all of these hormones may be causing or linked with salivation rates.

Although there is minimal research on the pathophysiology of salivation patterns to date, the role of CCK seems particularly promising as it is known to affect meal size (Smith & Gibbs, 2002) and may mediate the action of insulin and leptin (Moran, 2000). Further, after food ingestion, particularly with larger meals, individuals with bulimia nervosa have a blunted release of CCK as compared to control subjects (Devlin et al., 1997; Geracioti & Liddle, 1988; Pirke, Kellner, Friess, Krieg, & Fichter, 1994), likely leading to greater food consumption (i.e., binge episodes). In terms of weight status, rats with a null mutation of the CCK_A receptor have higher rates of overeating and obesity (Moran, 2000), and administering CCK (via intravenous infusion) significantly decreases meal size in obese human subjects as compared to a saline infusion (Pi-Sunyer, Kissileff, Thornton, & Smith, 1982). Taken together, these findings suggest that the abnormal salivation patterns observed in obese, OW and bulimic samples may be due to corresponding abnormalities in the CPR—deficiency of CCK or CCK_A receptors.

The specific brain regions involved in the CPR are not well understood, however, the hypothalamus, a key structure involved in appetite regulation, has been implicated (Klajner, Herman, Polivy, & Chhabra, 1981; Lechan & Fekete, 2006). A recent study conducting genome-wide screens, in order to identify genetic variants associated with obesity, reported that several of the identified genes are expressed in the hypothalamus (Hotta, 2009), giving rise to the possibility of genetic susceptibility as a causal agent of abnormalities in the CPR.

Another possibility may relate to differences in taste perception between normal weight and OW individuals. Eating is often driven by the hedonic value of food (Tataranni & DelParigi, 2003). Studies have found that individuals with OW display a higher taste sensitivity to foods (Drewnowski, 1985; Pasquet, Frelut, Simmen, Hladik, & Monneuse, 2007), display an increased anticipation to eat (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008), and find foods more reinforcing (Saelens & Epstein, 1996)

as compared to their normal weight counterparts. A recent fMRI study confirmed that during exposure to a food stimulus (anticipatory and consummatory), OW adolescents displayed greater increases in activation of brain regions associated with the hedonic aspects of food and reward brain circuitry as compared to normal weight adolescents (Stice et al., 2008). Thus, another possible causal/maintaining theory is that among OW youth, the increased reward/hedonic circuitry activated during exposure to food overrides the hypothalamus regulatory function, resulting in abnormalities in the CPR and larger/more frequent eating episodes.

From a learning perspective, memory and classical conditioning may also offer information on causality. By definition, habituation is a simple form of learning in which animals filter out *unimportant* information so that they can focus attention on the most central features of the environment. Research has shown that obese adults (King, Polivy, & Herman, 1991), OW children (Halford et al., 2004; Soetens & Braet, 2007) and individuals with eating disorders (Dobson & Dozois, 2002; Johansson, Ghaderi, & Andersson, 2005; Lee & Shafran, 2004) have an explicit bias towards remembering food-related information. These findings suggest that individuals with weight-related disorders are more focused on food-related cues and hence may not habituate because food related information remains of high importance as compared to other aspects of the environment.

The classical conditioning model of overeating (i.e., cue reactivity) posited by Jansen (1998) states that after repeated pairings between food and a particular cue (e.g., site, smell or taste of food), the cue will elicit the same food effects (i.e., initiation of the CPR) which are interpreted by the individual as cravings. Interestingly, in a laboratory study, larger meals were associated with higher rates of salivation immediately following food exposure, particularly so among OW children (Jansen et al., 2003). If the positive energy balance that defines OW and obesity results from repeated

episodes of overeating, perhaps over time these pairings (between food cues, large meals and salivation) result in food cues (conditioned stimulus) eliciting an extended salivary response (conditioned response).

The present study detected weight-related differences in children's responses to food. It is currently unknown whether a biological vulnerability causes the disruption of the habituation process (e.g., genetic susceptibility leading to CPR abnormalities) or whether dysregulated eating disrupts an individual's biological response to food (e.g., cue sensitivity), or both. Thus, prospective research in this area, following children before the development of OW or any other disturbed eating patterns, is warranted. Based on the current literature, it appears that there may be an incremental relationship between weight status and biological response to food. However, the causal direction of this potential pattern is unclear, and more habituation studies are needed with OW children and those at-risk for OW to evaluate this hypothesis and to clarify the discrepancies between the present study and those of the Temple et al. (2007). Furthermore, very little research exists on the causal and/or maintaining agents underlying the habituation patterns among OW children. As described above, areas of particular promise include: (1) hormonal irregularities, (2) genetic susceptibility, (3) hedonic/reward brain circuitry, (4) explicit memory bias and (5) classical conditioning (see Figure 7 for a proposed causality/maintaining model with possible pathways indicated).

Conclusions

In sum, it is well documented that approximately half of OW children become obese adults (Serdula et al., 1993; Freedman et al., 2004; Must et al., 1992; Whitaker et al., 1997). Given the poor long-term success of weight loss treatments for adults with obesity, it is imperative that we intervene with Children before their OW tracks into

adulthood. The findings from the present study suggest that biological factors play an important role in explaining the positive energy balance that characterizes this condition. Future research is needed to explore potential causal and/or maintaining mechanisms and to evaluate whether the lack of habitation among OW children is in any way facilitating the maintenance of their OW status. If confirmed, these findings could provide a new understanding of how OW develops and is maintained, and could potentially offer novel strategies to improve long-term success in the treatment of OW.

Figure 7. Proposed Causality/Maintaining Model

Cognitive Stimulus (e.g.. Presence of food)

(2) Genetic Susceptibility

(3) Reward/hedonic brain circuitry

Elicited Response

Hypothalamus (Negative Feedback Loop)

(5) Learned Behaviors

(4) Explicit Memory Bias

Initiates CPR

Extended Salivary Response

(1) Decreased CCK Response

Increased Energy Consumption Meal Size

Decreased Release of Interocepters (e.g.. Leptin. Grehlin & Insulin)

Increased Fat Stores

References

Ackard, D.M., Neumark-Sztainer, D., Story, M., & Perry, C. (2003). Overeating among adolescents: Prevalence and associations with weight-related characteristics and psychological health. *Pediatrics*, *111*(1), 67-74.

Agras, W.S. & Mascola, A.J. (2005). Risk factors for childhood OW. *Current Opinion in Pediatrics*, *17*, 648-652.

Anderson J.W., Konz, E.C., Frederich, R.C., & Wood C.L. (2001) Long-term weight loss maintenance: A meta-analysis of US studies. *American Journal of Clinical Nutrition*, *74*, 579 –84.

Barlow, S.E. & the Expert Committee (2007). Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: Summary report. *Pediatrics, 120,* S164-S192.

Birch, L.L. & Fisher, J.O. (1998). Development of eating behaviors among children and adolescents. *Pediatrics*, *101*, 539-549.

Boutelle, K., Neumark-Sztainer, D., Story, M., & Resnick, M. (2002). Weight control behaviors among obese, overweight, and nonoverweight adolescents. *Journal of Pediatric Psychology*, *27*, 531-540.

Braet, C., Mervielde, I., & Vandereycken, W. (1997). Psychological aspects of childhood obesity: A controlled study in a clinical and nonclinical sample. *Journal of Pediatric Psychology*, *22*, 59-71.

Brownell, K.D. (2002). The environment and obesity. In C.G. Fairburn & K.D. Brownell (Eds.), *Eating disorders and obesity* (2nd ed., pp. 162-170). New York: Guilford Press.

Caroli, M., Argentieri, L., Cardone, M., & Masi, A. (2004). Role of television in childhood obesity prevention. *International Journal of Obesity*, *28*, S104-S108.

Centers for Disease Control and Prevention, National Center for Health Statistics.

> Prevalence of OW in children and adolescents: United States, 1999. Available at
> http://www.cdc.gov/nchs/products.

CPMP. Points to Consider on Adjustment for Baseline Covariates. EMEA: London, 2001.

> Available at www.emea.eu.int/htms/human/ewp/ewpptc.htm.

Crespo, C., Smit, E., Troiano, R., Bartlett, S., Macera, C., & Anderson, R. (2001).

> Television watching, energy intake and obesity in US children. *Archives of
> Pediatric Adolescent Medicine, 155*, 360-365.

Devlin, M.J., Walsh, T., Guss, J.L., Kissileff, H.R., Liddle, R.A., & Petkova, E. (1997).

> Postprandial cholecystokinin release and gastric emptying in patients with
> bulimia nervosa. *American Journal of Nutrition, 65*, 114-120.

Dietz, W.H. (1998). Health consequences of obesity in youth: Childhood predictors of

> adult disease. *Pediatrics, 101*, 518-525.

Dietz, W.H. (2006). OW in childhood adolescence. *New England Journal of Medicine*,

> 350 (9), 855-857.

Dobson, K.S. & Dozois, D.J.A. (2002). Attentional biases in eating disorders: A meta-

> analytic review of Stroop performance. *Clinical Psychology Review, 23*, 1001-
> 1022.

Douketis J., Macie, C., Thabane, L., & Williamson, D. (2005). Systematic review of long-

> term weight loss studies in obese adults: Clinical significance and applicability to
> clinical practice. *International Journal of Obesity, 29,* 1153-1167.

Drewnowski, A. (1985). Sweet tooth reconsidered: Taste responsiveness in human

> obesity. *Physiology & Behavior, 35*, 617-622.

Durant, R.H., Baranowski, T., Johnson, M., & Thompson, W.O. (1994). The relationship

> among television watching, physical activity, and body composition of young
> children. *Pediatrics, 94*, 449-455.

Eisenberg, M.E., Neumark-Sztainer, D. & Story, M. (2006). Associations of weight-based teasing and emotional well-being among adolescents. *Archives of Pediatric Adolescent Medicine*, *157*, 733-738.

Ello-Martin, J.A., Ledikwe, J.H., & Rolls, B.J. (2005). The influence of food portion size and energy density on energy intake: Implications for weight management. *American Journal of Clinical Nutrition*, *82*, 236S-241S.

Emes, C., Velde, B., Moreau, M., Murdoch, D.D., & Trussell, R. (1990). An activity based weight control program. *Adapted Physical Activity Quarterly, 7*, 314-324.

Epstein, L.H. (1992). Role of behavior theory in behavioral medicine. *Journal of Consulting and Clinical Psychology*, *60*, 493-498.

Epstein, L.H., Mitchell, S.L., & Caggiula, A.R. (1993). The effect of subjective and physiological arousal on dishabituation of salivation. *Physiology and Behavior, 53*, 593-597.

Epstein, L.H., Myers, M.D., Raynor, H.A., & Saelens, B.E. (1998). Treatment of pediatric obesity. *Pediatrics*, *101*, 554-570.

Epstein, L.H., Paluch, R., & Coleman, K.J. (1996). Differences in salivation to repeated food cues in obese and nonobese women. *Psychosomatic Medicine*, *58*, 160-164.

Epstein, L.H., Rodefer, J.S., Wisniewski, L., & Caggiula, A.R. (1992). Habituation and dishabituation of human salivary response. *Physiology and Behavior*, *51*, 945-950.

Epstein, L.H., Saad, F.G., Giacomelli, A.M., & Roemmich, J.M. (2005). Effects of allocation on habituation to olfactory and visual food stimuli in children. *Physiology & Behavior*, *84*, 313-319.

Epstein, L.H., Saad, F.G., Handley, E.A., Roemmich, J.N., Hawk, L.W. & McSweeney, F.K. (2003). Habituation and salivation and motivated responding for food in children. *Appetite*, *41*, 283-289.

Epstein, L.H., Valoski, A., Wing, R.R., & McCurley, J. (1994). Ten-year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychology*, *13*, 373-383.

Epstein, L., Valoski, A., Vara, L., McCurley, J., Wisniewski, L., Kalarchian, M., et al. (1995). Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. *Health Psychology*, *14*, 109-108.

Erickson, S., Robinson, T., Haydel, K., & Killen, J. (2000). Are overweight children unhappy? Body mass index, depressive symptoms, and OW concerns in elementary school children. *Archives of Pediatric Adolescent Medicine*, *154*, 931-935.

Erickson, M., Poston, C., & Foreyt, J.P. (1996). Common biological pathways in eating disorders and obesity. *Addictive Behaviors*, *21*, 733-743.

Everson, S. A., Maty, S. C., Lynch, J. W., & Kaplan, G. A. (2002). Epidemiologic evidence for the relation between socioeconomic status and depression, obesity, and diabetes. *Journal of Psychosomatic Research, 53*, 891-895.

Fairburn, C.G., Doll, H.A., Welch, S.L., Hay, P.J., Davies, B.A. & O'Connor, M.E. (1999). Risk factors for binge eating disorder: a community-based, case-control study. *Archives of General Psychiatry*, *55*, 425-432.

Faith, M.S., B.E. Saelens, D.E. Wilfley, & D.B. Allison, (2001). Behavioral treatment of childhood and adolescent obesity: Current status, challenges, and future directions. In J.K. Thompson & L. Smolak, *Body image, eating disorders, and obesity in youth: Assessment, prevention, and treatment (*pp. 313-340). American Psychological Association: Washington, DC.

Falkner, N.H., Neumark-Sztainer, D., Story, M., Jeffery, R.W., Beuhring, T. & Resnick, M.D. (2001). Social, educational, and psychological correlates of weight status in adolescents. *Obesity Research*, *9*, 32-42.

Field, A.E., Austin, S.B., Taylor, C.B. et al. (2003). Relation between dieting and weight change among preadolescents and adolescents, *Pediatrics, 112*, 900-906.

Floodmark, C., Ohlosson, T., Ryden, O., & Sveger, T. (1993). Prevention of progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics, 91*, 880-884.

Foreyt, J.T. & Goodrick, G.K. (1991). Factors common to successful therapy for the obese patient. *Medicine and Science in Sports and Exercise*, *23*, 292-297.

Freedman, D. S., Khan, L. K., Serdula, M. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2004). Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *International Journal of Obesity Related Metabolic Disorders, 28*, 10-16.

Garner, D.M. & Wooley, S.C. (1991). Confronting the failure of behavioral and dietary treatments for obesity. *Clinical Psychology Review*, *11*, 729-780.

Geracioti, T.D. & Liddle, R.A. (1988) Impaired cholecystokinin secretion in bulimia nervosa. *New England Journal of Medicine, 319,*683-688.

Goldfield, A. & Chrisler, J.C. (1995). Body stereotyping and stigmatization of obese persons by first graders. *Perceptual Motor Skills*, *81*, 909-910.

Goldschmidt, A.B., Passi Aspen, V.A., Sinton, M.M.**,** Tanofsky-Kraff, M., & Wilfley, D.E. (2008). Disordered eating attitudes and behaviors in overweight youth. *Obesity, 16*, 257-264.

Gortmaker, S.L., Must, A., Perrin, J.M., Sobol, A.M., & Dietz, W.H. (1993). Social and
economic consequences of OW in adolescence and young adulthood. *The New
England Journal of Medicine*, *329*, 1008-1012.

Gortmaker, S.L., Must, A., Sobol, A.M., Peterson, K., Colditz, G.A., & Dietz, W.H. (1996).
Television watching as a cause of increasing obesity among children in the
United States, 1986-1990. *Archives of Pediatric Adolescent Medicine*, *150*, 356-
362.

Grouin, J.M., Day, S., Lewis, J. (2004). Adjustment for baseline covariates: An
introductory note. *Statistics in Medicine*, *23*, 697–699.

Hancox, R.J. & Poulton, R. (2005). Watching television is associated with childhood
obesity: but is it clinically important? *International Journal of Obesity*, *30*, 171-
175.

Halford, J.C.G., Gillespie, J., Brown, V., Pontin, E.E., & Dovey, T.M. (2004). Effect of
television advertisements for food consumption in children. *Appetite, 42*, 221-
225.

Hayden-Wade, H. A., Stein, R. I., Ghaderi, A., Saelens, B. E., Zabinski, M. F., & Wilfley,
D. E. (2005). Prevalence, characteristics, and correlates of teasing experiences
among overweight children vs. non-overweight peers. *Obesity Research, 13*,
1381-1392.

Hill, A.J., & Pallin, V. (1998). Dieting awareness and low self-worth: Related issues in 8-
year-old girls. *International Journal of Eating Disorders*, *24*, 405-413.

Hill, J.O. & Peters, J.C. (1998). Environmental contributions to the obesity epidemic.
Science, *280*, 1371-1374.

Hollingshead, A. B. (1975). Four factor index of social status. Yale University.

Hotta, J. (2009). New insights about obesity related genes. *Japanese Journal of Clinical
Medicine*, *67*, 253-256.

Hu, F. B., Li, T. Y., Colditz, G. A., Willett, W. C., & Manson, J. E. (2003). Television

    watching and other sedentary behaviors in relation to risk of obesity and type 2

    diabetes mellitus in women. *Journal of the American Medical Association, 289*,

    1785-1791.

Israel, A.C., Guile, C.A., Baker, J.E., & Silverman, W.K. (1994). An evaluation of

    enhanced self-regulation training in the treatment of childhood obesity. *Journal of

    Pediatric Psychology, 19,* 737-749.

Jago, R., Baranowski, T., Baranowski, J.C., Thompson, D., & Greaves, K.A. (2005). BMI

    from 3-6y of age is predicted by TV viewing and physical activity, not diet.

    International Journal of Obesity, *29*, 557-564.

Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure.

    Behaviour Research and Therapy, *36*, 257-272.

Jansen, A., Theunissen, N., Slechten, K., Nederkoorn, C., Boon, B., Mulkens, S., et al.

    (2003). Overweight children overeat after exposure to food cues. *Eating

    Behaviors*, *4*, 197-209.

Jeffery, R.W., Epstein, L.H., Wilson, T.G., Drenowski, A., Stunkard, A.J., & Wing, R.R.

    (2000). Long-term maintenance of weight loss: Current status. *Health

    Psychology*, *19*, 5-16.

Jelalian, E. & Saelens, B.E. (1999). Empirically supported treatments on pediatric

    psychology: Pediatric obesity. *Journal of Pediatric Psychology*, *24*, 223-248.

Johansson, L., Ghaderi, A., & Andersson, G. (2005). Stroop interference for food- and

    body-related words: A meta-analysis. *Eating Behaviors*, *6*, 271-281.

Jordan, A. (2004). The role of media in children's development: An ecological

    perspective. *Developmental and Behavioral Pediatrics*, *25*, 196-206.

Jordan, A., & Robinson, T. (2008). Children's television viewing, and weight status: Summary and recommendations from an expert panel meeting. *Annals of the American Academy of Political and Social Science*, *615*, 119-132.

Killen, J.D., Taylor, C.B., Hayward, C.H., Haydel, F., Wilson, D., Hammer, L., Kraemer, H., Blair-Greiner, A., & Strachowski, D. (1996). Weight concerns influence the development of eating disorders: A four-year prospective study. *Journal of Consulting and Clinical Psychology*, *64*, 936-940.

Killen, J.D., Taylor, C.B., Hayward, C., Wilson, D.M., Haydel, K.F., Hammer, L.D., et al. (1994). Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: A three-year prospective analysis. *International Journal of Eating Disorders*, 16, 227-238.

King, G. A., Polivy, J., & Herman, C. P. (1991). Cognitive aspects of dietary restraint: Effects on person memory. *International Journal of Eating Disorders, 10*, 313-321.

Klajner, C.P., Herman, J., Polivy, & Chhabra, R. (1981). Human obesity, dieting, and anticipatory salivation of food. *Physiology and Behavior, 27*, 195-198.

Kotz, K. & Story, M. (1994). Food advertisements during children's Saturday morning television programming: Are they consistent with dietary recommendations? *Journal of American Dietetic Association*, *94*, 1296.

Kuczmarski, R.J., Ogden, C.L., Grummer-Strawn, L.M., Flegal, K.M., Guo, S.S., Wei, R., et al. (2000) CDC growth charts: United States. *Advanced Data, 314*,1-27.

Latner, J.D. & Stunkard, A.J. (2003). Getting worse: The stigmatization of obese children. *Obesity Research*, *11*, 452-456.

Lechan, R.M. & Fekete, C. (2006). The TRH neuron: A hypothalamic integrator of

    energy metabolism. *Progressive Brain Research, 153*, 209-235.

Lee, M., & Shafran, R. (2004). Information processing biases in eating disorders. *Clinical*

    Psychology Review, 24, 215-238.

Levitsky, D.A. (2005). The non-regulation of food intake in humans: Hope for reversing

    the epidemic of obesity. *Physiology & Behavior*, 86, 623-632.

Louis-Sylvestre, J. (1987). The cephalic phase of insulin secretion. *Diabetes &*

    Metabolism, 13, 63-73.

Marcus, M. D., Kalarchian, M. A., & Levine, M. D. (2005). Treatment of severe pediatric

    OW. in *Eleventh Annual Meeting for the Eating Disorder Research Society*. 2005:

    Toronto, Ontario Canada.

Marshall, S.J., Biddle, S.J.H., Gorely, T., Cameron, N., & Murdey, I. (2004). Relationship

    between media use, body fatness and physical activity in children and youth: A

    meta-analysis. *International Journal of Obesity, 28*, 1238-1246.

Matheson, D.M., Killen, J.D., Wang, Y., Varady, A., & Robinson, T.N. (2004a). Children's

    food consumption during television viewing. *American Journal of Clinical*

    Nutrition, *79*, 1088-1094.

Matheson, D.M., Wang, Y., Klesges, L.M., Beech, B.M., Kraemer, H.C., & Robinson,

    T.N. (2004b). African-American girls' dietary intake while watching television.

    Obesity Research, *12*, 32S-37S.

Melville, C.L., Rue, H.C., Rybiski, L.R., & Weatherly, J.N. (1997). Altering reinforcer

    variety or intensity changes the within-session decreases in responding. *Learning*

    and Motivation, *28*, 609-21.

Mockus, D. S., Epstein, L. E., & Wilfley, D. E. (2005). Self-monitoring predicts weight loss in OW children. Paper presented at the Eleventh Annual Meeting for the Eating Disorders Research Society, Toronto, Canada, September, 2005.

Mokdad, A.H., Ford, E.S., Bowman, B.A., Dietz, W.H., Vinicor, F., Bales, V.S. & Marks, J.S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, *Journal of the American Medical Association*, *289*, 76-79.

Moran, T.H. (2000). Cholecystokinin and satiety: Current perspectives. *Nutrition (16)*, 858-865.

Morgan, C. M., Tanofsky-Kraff, M., Wilfley, D.E., & Yanovski, J.A. (2002). Childhood obesity. *Child and Adolescent Psychiatric Clinics of North America, 11*, 257-278.

Myers, E.M. & Epstein, L.H. (2002). Habituation of responding for food in humans. *Appetite, 38*, 224-234.

Myers, E.M. & Epstein, L.H. (2007). The effect of dietary fat on salivary habituation and satiation. *Physiology and Behavior, 62*, 155-161.

Must A., Jacques P.F., Dallal G.E., Bajema C.J., & Dietz, W.H. (1992). Long-term morbidity and mortality of OW adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. *New England Journal of Medicine*, *327*, 1350-1355.

National Institute of Health (1998). Clinical Guidelines on the Identification, Evaluation, and Treatment of OW and Obesity in Adults. 228 pages. NIH Publication No. 98-4083.

Nederkoorn, C., Smulders, F.T.Y., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. *Appetite*, *35*, 45-55.

Neumark-Sztainer, D., Wall, M., Eisenberg, M.E., Story, M. & Hannan, P.J. (2006). Overweight status and weight control behaviors in adolescents: Longitudinal and secular trends from 1999 to 2004. *Preventative Medicine: An International Journal Devoted to Practice and Theory, 43*, 52-59.

Neumark-Sztainer, D., Story, M., French, S.A., & Resnick, M.D. (1997). Psychosocial

    correlates of health compromising behaviors among adolescents, *Health*

    Education Resource, 12, 37-52.

Ogden, C.L., Carroll, M.D., Curtin, L.R., Mcdowell, M.A., Tabak, C.J., & Flegal, K.M.

    (2006). Prevalence of OW and obesity in the United States, 1999-2004, *Journal*

    of the American Medical Association, *295* (13), 1549-1555.

Ogden, C., Carroll, M., & Flegal, K. (2008). High body mass index for age among

    US children and adolescents, 2003-2006. *Journal of the American Medical*

    Association, *299*, 2401-2405.

Pasquet, P., Frelut, M.L., Simmen, B., Hladik, C.M., & Monneuse, M.O. (2007). Taste

    perception in massively obese and non-obese adolescents. *International Journal*

    of Pediatric Obesity, 2, 242-248.

Perri, M.G. & Fuller, P.R. (1995). Success and failure in the treatment of obesity: Where

    do we go from here? *Medicine, Exercise, Nutrition, and Health*, *4*, 255-272.

Parsons, T. J., Power, C., Logan, S., & Summerbell, C. D. (1999). Childhood predictors

    of adult obesity: A systematic review. *International Journal of Obesity, 23*, S1-

    S107.

Peck, R.E. (1959). The SHP test-an aid in the detection and measurement of

    depression. *Archives of General Psychiatry*, *1*, 35-40.

Pirke, K.M., Kellner, M.B., Friess E, Krieg I, & Fichter, M.M.(1994). Satiety and

    cholecystokinin. International Journal of Eating Disorders,*15*, 63-69.

Pi-Sunver, X., Kissileff, H.R., Thornton, J., & Smith, G.P. (1982). C-terminal octapeptide

    of cholecystokinin decreases food intake in obese men. *Physiology and Behavior*

    29, 627-630.

Power, M. & Schulkin, J. (2008). Anticipatory physiological regulation in feeding

    biology: Cephalic phase responses. *Appetite*, *50*, 194-206.

Powley, T.L. & Berthoud, H. (1985). Diet and cephalic phase insulin responses. *The*

    American Journal of Clinical Nutrition, *42*, 991-1002.

Raab G.M., Day S., & Sales J. (2000). How to select covariates to include in the analysis

    of a clinical trial. *Controlled Clinical Trials*, *21*, 330-342.

Richardson S.A., Goodman N., Hastorf A.H., & Dornbusch S.M. (1961). Cultural

    uniformity in reaction to physical disabilities. *American Sociological Review*, *26*,

    241–247.

Robinson, T.N. (1999). Reducing children's television viewing to prevent obesity. *Journal*

    of the American Medical Association, *282*, 1561-1567.

Robinson, T.N. (2001). Television watching and childhood obesity. *Pediatric Clinics of*

    North America, 48, 1017-1025.

Robinson, T.N. & Killen, J.D. (1995). Ethnic and gender differences in the relationship

    between television viewing and obesity, physical activity and dietary fat intake.

    Journal of Health and Education, *26*, S91-S98.

Robinson, T.N. & Killen, J. D. (2002). Obesity prevention for children and adolescents. In

    J.K. Thompson & L. Smolak (Eds.), *Body image, eating disorders, and obesity in*

    youth (pp. 261-292). Washington D.C.: American Psychological Association.

Saelens, B.E. & Epstein, L.H. (1996). The reinforcing value of food in obese and non-

    obese women. Appetite, 27, 41-50.

Salvy, S., Kieffer, E., & Epstein, L. (2008). Effects of social context on overweight and

    normal weight children's food selection. *Eating Behaviors*, *9*, 190-196.

Sallis, J.F. & Glanz, K. (2006). The role of built environments in physical activity, eating,

    and obesity in childhood. *Future Child*, *16*, 89-108.

Schwartz, M.W. (2001). Brain pathways controlling food intake and body weight. *Experimental Biology and Medicine*, 226, 978-981.

Serdula, M.K., Ivery, D., Coates, R.J., Freedman, D.S., Williamson, D.F. & Byers, T. (1993). Do obese children become obese adults? A review of the literature. *Preventative Medicine*, *22*, 167-177.

Soetens, B., & Braet, C. (2007). Information processing of food cues in overweight and normal weight adolescents. *British Journal of Health Psychology, 12*, 285-304.

Sjoberg, R.L., Nilsson, K.W., & Leppert, J. (2006). Obesity, shame, and depression in school-aged children: A population based study. *American Academy of Pediatrics*, 116, 389-392.

Smith, G.P. & Gibbs, J. (2002). Peripheral physiological determinants of eating and body weight. In C.G. Fairburn & K. Brownell (Eds.), Eating disorders and obesity (2nd ed., pp. 11-15). New York: Guilford Press.

Stephenson, D. & Siddle, D.A. (1983). Theories of habituation. In Siddle, D. (Ed), *Orienting and Habituation: Perspectives in Human Research*. 183-236. New York: Wiley.

Stettler, N., Signer, T.M., & Suter, P.M. (2004). Electronic games and environmental factors associated with childhood obesity in Switzerland. *Obesity Research*, *12*, 896-903.

Stice, E., Spoor, S., Bohon, C., Veldhuizen, M.G., & Small, D.M. (2008). Relation of reward from food intake and anticipated food intake to obesity: A functional magnetic resonance imaging study. *Journal of Abnormal Psychology, 117*, 924-935.

Strasburger, V.C. (2001). Children and TV advertising: Nowhere to run, nowhere to hide. *Journal of Developmental & Behavioral Pediatrics*, *22*, 185-187.

Strauss, R.S. (1999). Childhood obesity. *Current Problems in Pediatrics*, *29*, 1-29.

Strauss, R.S. & Pollack, H.A. (2003). Social marginalization of OW children. *Archives of Pediatric Adolescent Medicine*, *157*, 746-752.

Striegel-Moore, R.H., Fairburn, C.G., Wilfley, D.E., Pike, K.M., Dohm, F.A. & Kraemer, H.C. (2005). Toward an understanding of risk factors for binge-eating disorder in black and white women: A community-based case-control study. *Psychological Medicine*, *35*, 907-917.

Striegel-Moore, R.H., Schreiber, G.B., Lo, A., Crawford, P., Obarzanek, E., & Rodin, J. (2000). Eating disorder symptoms in a cohort of 11-16-year-old black and white girls: The NHLBI growth and health study. *International Journal of Eating Disorders*, *27*, 49-66.

Swallen, K. C., Reither, E. N., Haas, S. A., & Meier, A. M. (2005). OW, obesity, and health-related quality of life among adolescents: The National Longitudinal Study of Adolescent Health. *Pediatrics, 115*, 340-347.

Swithers, S.E. & Hall, W.G. (1994). Does oral experience terminate ingestion? *Appetite*, *23*, 113-138.

Summerbell, C. D., Ashton, V., Campbell, K.J., Edmunds, L., Kelly, S. & Waters, E. (2003). Interventions for treating obesity in children. *Cochrane Database Systematic Reviews*, *3*, CD001872.

Sussman, S., Valente, T.W., Rohrbach, L.A., Skara, S., & Pentz, M.A. (2006). Translation in the healh professions: converting science into action. *Evaluation and the Health Professions*, *29*, 7-32.

Tanofsky-Kraff, M., H. Hayden-Wade, P. Cavazos, & D.E. Wilfley. (2003). Pediatric overweight treatment and prevention, in R. Anderson (Ed), *Overweight: Etiology, Assessment, Treatment and Prevention* (pp. 155-176). Champaign, IL: Human Kinetics.

Tanofsky-Kraff, M., Yanovski, S.Z., Wilfley, D.E. et al. (2004). Eating-disordered behaviors, body fat, and psychopathology in overweight and normal-weight children. *Journal of Consulting and Clinical Psychology, 72*, 53-61.

Tataranni, P.A. & DelParigi, A. (2003). Functional neuroimaging: A new generation of human brain studies in obesity research. *Obesity, 4*, 229-238.

Temple, J.L., Giacomelli, A.M., Roemmich, J.N., & Epstein, L.H. (2007).Overweight children habituate slower than non-overweight children to food. *Physiology and Behavior*, *91*, 250-254.

Temple, J.L., Kent, K.M., Giacomelli, A.M., Paluch, R.A., Roemmich, J.N., & Epstein, L.H. (2006). Habituation and recovery of salivation and motivated responding for food in children. *Appetite*, *46*, 280-284.

Thompson, D. & Wolf, A.M. (2001). The medical-care cost burden of obesity. *Obesity Review*, *2*, 189-197.

Tucker, L. A. & Bagwell, M. (1991). Television viewing and obesity in adult females. *American Journal of Public Health, 81*, 908-911.

Van Strien, T., Frijters, J.E.R., Bergers, G.P.A., & Defares, P.B. (1986). The Dutch Eating Behaviour Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behaviour. *International Journal of Eating Disorders*, *5*, 747–755.

Wang, G. & Dietz, W.H. (2002) Economic burden of obesity in youths aged 6 to 17 years: 1979-1999. *Pediatrics*, *109*, E81-1.

Whitaker, R.C., Wright, J.A., Pepe, M.S., Seidel, K.D., & Dietz, W.H. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *New England Journal of Medicine*, *337*, 869-873.

WHO. Obesity: Preventing and managing the global epidemic. Geneva' World Health Organization; 1998.

WHO. (2003). Diet, Nutrition, and Prevention of Chronic Diseases: Report of a joint

WHO/FAO Expert Consultation, Geneva.

Wilfley, D.E., Passi, V.A., Cooperberg, J., & Stein, R.I. (2006) Cognitive-Behavioral

therapy for youth with eating disorders and obesity. In P. Kendall (Ed.), *Child

and adolescent therapy: Cognitive-behavioral procedures* (3rd ed.). New York:

Guilford Press.

Wilfley, D.E. & Saelens, B.E. (2002). Epidemiology and causes of obesity in children. In

C.G. Fairburn & K. Brownell (Eds.), *Eating disorders and obesity* (2nd ed., pp.

162-170). New York: Guilford Press.

Wilfley, D.E., Stein, R. I., Saelens, B.E., Matt, G.E., Mockus, D.S., & Hayden-Wade,

H.A., et al. (2007). Efficacy of maintenance treatment approaches for childhood

overweight: A randomized controlled trial. *Journal of the American Medical

Association*, *298*, 1661-1673.

Wilson, G.T. (1994). Behavioral treatment of childhood obesity: Theoretical and practical

implications. *Health Psychology*, *13*, 371-372.

Wisniewski, L., Epstein, L.H., & Caggiula, A.R. (1992). Effect of food change on

consumption, hedonics, and salivation. *Physiology and Behavior*, *52*, 21-6.

Wisniewski, L., Epstein, L.H., Marcus, M.D., & Kaye, W. (1997). Differences in salivary

habituation to palatable foods in bulimia nervosa patients and controls.

Psychosomatic Medicine, *59*, 427-433.

Wooley, S.C. & Wooley, O.W. (1973). Salivation to the sight and smell of food: A new

measure of appetite. *Psychosomatic Medicine*, *35*, 136-142.

Wooley, O.W. & Wooley, S.C. (1976). Deprivation, expectation and threat: Effects on

salivation in the obese and nonobese. *Physiology and Behavior*, *17*, 187-193.

Wrotniak, B.H., Epstein, L.H., Paluch, R.A., & Roemmich, J.N. (2005). The relationship

between parent and child self-reported adherence and weight loss. *Obesity

Review*, *13*, 1089-1096.

Zametkin, A.J., Zoon, C.K., Klein, H.W., & Munson, S. (2004). Psychiatric aspects of

child and adolescent obesity: A review of the past 10 years. *Journal of the

American Academy of Child and Adolescent Psychiatry, 43*, 134-160.

Appendix A : Glossary

Body mass index (BMI): weight in kilograms divided by the square of height in meters

Cephalic Phase Response (CPR): The first responses from the body elicited by exposure to sensory properties of food (i.e., color, flavor and aroma), which includes increases in saliva, heart rate, temperature and gastric activity.

Habituation: A general process of decreasing responsiveness after repeated exposures to a stimulus and recovering when a new stimulus is presented; Habituation is measured via salivation in the context of this proposal

Salivary flow: An example of a CPR

Translational research: The adaptation and application of methods and principles from one area of science to another

Weight definitions:
At risk of overweight: Children with a BMI between the 85th and 95th percentiles for age and sex

Overweight: Children with a BMI at or above the 95th percentile for age and sex

Obese: Adults with a BMI of 30 kg/m^2 or greater

Appendix B: Questionnaires

MODIFIED DEBQ

PLEASE DRAW A CIRCLE AROUND THE ANSWER THAT IS TRUE FOR YOU.

1. If I feel fat, I try to eat less.

NEVER   SOMETIMES   VERY OFTEN

2. I try not to eat foods that might make me fat.

NEVER   SOMETIMES   VERY OFTEN

3. I have tried to lose weight

NEVER   SOMETIMES   VERY OFTEN

4. If I have eaten too much, I try to eat less the next day.

NEVER   SOMETIMES   VERY OFTEN

5. I try not to eat between meals because I want to be thinner.

NEVER   SOMETIMES   VERY OFTEN

6. I try to eat less because I don't want to get fat.

NEVER   SOMETIMES   VERY OFTEN

7. I try to get thinner by doing more exercise

NEVER   SOMETIMES   VERY OFTEN

8. If my mom feels fat, she tries to eat less.

NEVER   SOMETIMES   VERY OFTEN

9. If my dad feels fat, he tries to eat less.

NEVER   SOMETIMES   VERY OFTEN

MARY JANE THOUGHT THAT SHE WAS GETTING FATTER.
HOW OFTEN SHOULD SHE TRY TO DO THESE THINGS:

1. Do you think that she should try to eat less?

NEVER   SOMETIMES   VERY OFTEN

2. Do you think that she should try not to eat foods that might make her fatter?

NEVER   SOMETIMES   VERY OFTEN

3. Do you think she should try to lose some weight?

NEVER   SOMETIMES   VERY OFTEN

4. Do you think she should eat less if she has eaten too much the day before?

NEVER   SOMETIMES   VERY OFTEN

5. Do you think she should try not to eat between meals?

NEVER   SOMETIMES   VERY OFTEN

6. Do you think she should try to eat less if she doesn't want to get fatter?

NEVER   SOMETIMES   VERY OFTEN

7. Do you think she should try to do more exercise?

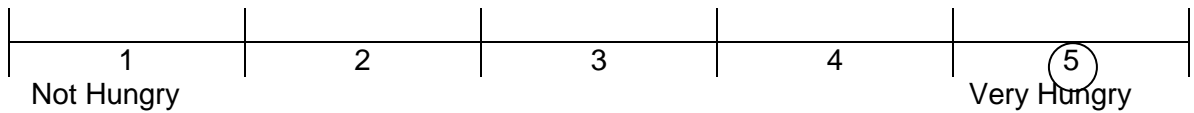NEVER   SOMETIMES   VERY OFTEN

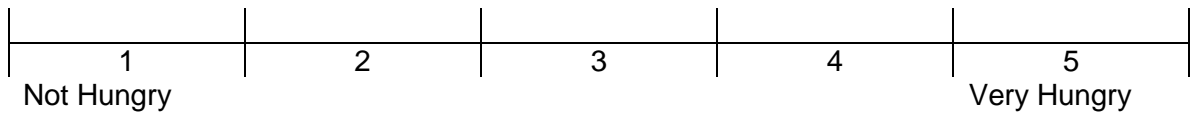Participant ID: _____

Date: _____

HUNGER SCALE

Directions: Circle the number between 1 and 5 that best represents your answer to the following question.

Example:

Jane is extremely hungry, so she circled a 5 on the scale below.

1	2	3	4	(5)
Not Hungry				Very Hungry

1. How hungry are you right now?

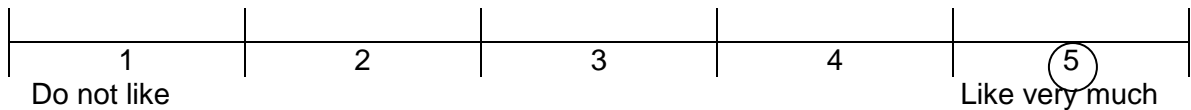|				
1	2	3	4	5
Not Hungry				Very Hungry

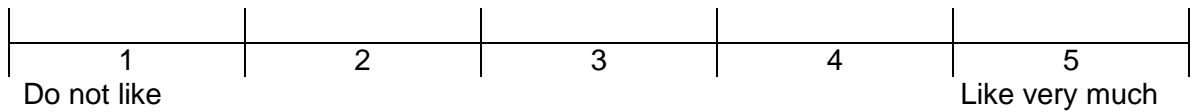Participant ID: _____
Date: _____

FOOD SCALE

Directions: Circle the number between 1 and 5 that best represents your liking to the following foods.

Example:

Jane likes French fries a lot, so she circled a 5 on the scale below.

1	2	3	4	5
Do not like				Like very much

1. How much do **you** like French fries?

1	2	3	4	5
Do not like				Like very much

Date: _____

FOOD PREFERENCES QUESTIONNAIRE

Items:	Do Not Like				Like Very Much
1. Chocolate bar	1	2	3	4	5
2. Frozen yogurt	1	2	3	4	5
3. Brownies	1	2	3	4	5
4. Cracker Jacks	1	2	3	4	5
5. Doughnut	1	2	3	4	5
6. Danish	1	2	3	4	5
7. Ice Cream	1	2	3	4	5
8. Cookies	1	2	3	4	5
9. Apple Pie	1	2	3	4	5
10. Cake	1	2	3	4	5
11. French fries	1	2	3	4	5
12. Potato Chips	1	2	3	4	5
13. Chicken Wings	1	2	3	4	5
14. Bacon	1	2	3	4	5
15. Doritos	1	2	3	4	5
16. Hamburger	1	2	3	4	5
17. Corn Chips	1	2	3	4	5
18. Cheese Balls	1	2	3	4	5
19. Tortilla Chips	1	2	3	4	5

Items:	Do Not Like				Like Very Much
20. Bologna	1	2	3	4	5
21. Chicken Fingers	1	2	3	4	5
22. Soda	1	2	3	4	5
23. Chocolate Syrup	1	2	3	4	5
24. Gum Drop	1	2	3	4	5
25. Hard Candy	1	2	3	4	5
26. Lollipop	1	2	3	4	5
27. Italian Lemon Ice	1	2	3	4	5
28. Carmel Candies	1	2	3	4	5
29. Fruit Juices	1	2	3	4	5
30. Jelly Beans	1	2	3	4	5
31. Kool-Aid	1	2	3	4	5
32. Bagel	1	2	3	4	5
33. English Muffin	1	2	3	4	5
34. White Rice	1	2	3	4	5
35. Corn	1	2	3	4	5
36. Baked Potato	1	2	3	4	5
37. Peas	1	2	3	4	5
38. Peppers	1	2	3	4	5
39. White Bread	1	2	3	4	5

Items:	Do Not Like				Like Very Much
40. Graham Crackers	1	2	3	4	5
41. Oyster Crackers	1	2	3	4	5
42. Broccoli	1	2	3	4	5
43. Oranges	1	2	3	4	5