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FOOD ATTENTIONAL BIASES AND ADIPOSITY: ARE ENERGY INTAKE AND EXTERNAL EATING MEDIATORS OF THIS RELATIONSHIP?

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ABSTRACT

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Obesity is a substantial threat to the health of over a third of adults in the United States. Some evidence suggests that food attentional bias, or the tendency to automatically direct attention toward food-related stimuli in the environment, may contribute to the development of obesity in susceptible individuals. This study hypothesized that (1) food attentional bias would be positively associated with adiposity, (2) food attentional bias would be positively associated with energy intake and external eating, and (3) energy intake and external eating would partially mediate the association between food attentional bias and adiposity. Data were collected from a sample of 120 undergraduate students. Three measures of food attentional bias were obtained: reaction time bias obtained from a visual dot-probe task and direction bias and duration bias obtained from eye tracking. Adiposity indices of body mass index (kg/m^2) and body fat percent were measured using standard medical devices. Data were obtained for two mediators: 1) energy intake was assessed by web-based automated 24-hour dietary recall and 2) external eating was assessed using the External Eating Subscale of the Dutch Eating

Behavior Questionnaire. Separate linear regression models examining the association between each measure of food attentional bias with each measure of adiposity (adjusted for age, sex, race/ethnicity, and subjective hunger) indicated no associations. Similarly, linear regression analyses revealed no associations between measures of food attentional bias and energy intake or external eating. Models testing for statistical mediation demonstrated that energy intake and external eating were not significant mediators. However, mediation analyses demonstrated a significant overall effect and direct effect between direction bias and BMI in a reduced sample used to test for energy intake as a mediator, suggesting the presence of an association which may not have been detected in the larger sample due to methodological issues, measurement error, or type I error. Despite the overall null results, these findings, in conjunction with previous studies on food attentional biases and adiposity, highlight the need for future investigations examining prospective associations between food attentional bias and adiposity.

CHAPTER 1. INTRODUCTION

Obesity is a substantial threat to the health and well being of more than one third of adults in the U.S. Therefore, effort has been put forth to identify factors that contribute to the development of obesity. In addition to traditional risk factors, a potential psychological risk factor for obesity may be attentional bias for food cues – i.e., increased attention to food cues in the environment. Evidence suggests that, although all people have food attentional biases when hungry, people who are obese tend to also have these biases when they are not hungry.

It has been hypothesized that people who pay more attention to food cues in their environment may have increased food cravings, eating behaviors, and energy intake, which could ultimately result in the development of obesity. A core assumption of this model is that food attentional biases increase eating behavior and energy intake. Few studies, however, have investigated whether increased food attentional bias is associated with greater energy intake or eating in response to food cues.

Several topics will be discussed to provide an introduction to the variables of interest. First, obesity will be introduced, including its significance, pathophysiology, and risk factors. Second, the definition, development, and measurement of food attentional biases will be reviewed. Third, the possible role of food attentional biases in obesity development will be discussed. Fourth, candidate mediators of the food attentional bias-obesity relationship will be reviewed. Candidate mediators in this study are energy intake and external eating. Finally, the conceptual model and hypotheses of the present study will be presented. The primary objective of this study is to examine the association between food attentional biases and adiposity, as well as the role of two candidate mediators.

1.1 Obesity

Obesity is a condition characterized by excess body fat mass, or adiposity, resulting from a chronic imbalance between energy intake and energy expenditure (Rosenbaum, Leibel, & Hirsch, 1997). Although there are numerous approaches used to measure adiposity, two common methods are body mass index (BMI) and body fat percent. BMI is computed from body weight and height (kg/m^2) (Roche, Sievogel, Chumlea, & Webb, 1981). According to the World Health Organization (World Health Organization, 1995, 2000), individuals with BMI values between 18.5-24.9 are of normal body weight, 25.0-29.9 are overweight, and greater than 30 are obese. BMI is the internationally accepted method for determining overweight and obesity in adults due to it being accurate and easy to measure (Luecken & Gallo, 2007). However, BMI is limited in that it does not distinguish adiposity from other body mass, such as muscle, bone, and fluid (Luecken & Gallo, 2007). For instance, an adult with high muscle mass may have a high BMI with relatively little adiposity. Additionally, BMI does not account for variability in body type based on demographic factors, such as age, sex, or race/ethnicity (Luecken & Gallo, 2007).

Body fat percent is computed as the ratio of adipose tissue to the other tissues of the body. Body fat percent is measured through bioelectrical impedance, which involves sending a low current through the body (Luecken & Gallo, 2007). Because the flow of the current is impeded by adipose tissue, bioelectrical impedance can be used to estimate the percentage of adipose tissue compared to other tissues (Luecken & Gallo, 2007). Measuring body fat percent is advantageous for two reasons: (1) it assesses adipose tissue independently of other tissues, and (2) it is calculated with height, weight, age, and sex taken into account (Luecken & Gallo, 2007). However, bioelectrical impedance devices are costly, and body fat percent measurements can be influenced by hydration, consumption of food or beverages, and recent exercise (Luecken & Gallo, 2007).

1.1.1 Significance

Based on estimates from 2010, 36% of adults and 17% of children and adolescents in the U.S. are obese (Centers for Disease Control and Prevention, 2012). Obesity affects some demographic groups at disproportionately high rates. For instance, 50% of non-Hispanic blacks, 40% of Mexican Americans, and 39% of all Hispanics are obese, compared with 34% of non-Hispanic white adults (Centers for Disease Control and Prevention, 2012). Although there are relatively equal rates of obesity in men (36%) and women (36%), the highest rates of obesity are found among African American women (59%) (Flegal, Carroll, Kit, & Ogden, 2012). Additionally, adults 60 years and older are more likely to be obese than younger adults (Centers for Disease Control and Prevention, 2012). Between 1980 and 2008, the prevalence of obesity worldwide increased more than two fold (World Health Organization, 2015). Similar increases in obesity prevalence have been noted in the U.S., growing from less than 15% in 1990 to 36% in 2010 (Centers for Disease Control and Prevention, 2012). In addition to being highly prevalent, the healthcare costs for the management and treatment of obesity are concerning. Obesity is responsible for approximately $147 billion dollars in health-related medical costs each year (CDC, 2009), and obese people have annual medical costs that are approximately $1,429 greater than costs for normal-weight people (Finkelstein, Fiebelkorn, & Wang, 2003).

Obesity is also associated with increased morbidity and mortality. Overweight and obese individuals are at an elevated risk of developing numerous medical conditions, including cardiovascular disease, type 2 diabetes, and certain cancers (Barness, Opitz, & Gilbert-Barness, 2007; Franken & Muris, 2005). Although BMI cut points are used to identify overweight and obesity categories, BMI appears to be linearly related to health risk (Willett et al., 1995; World Health Organization, 2000). Obesity may impact the body in a variety of ways by putting extra burden on organs and joints (Luecken & Gallo, 2007). In addition, excess adipose tissue can increase the release of hormones and proinflammatory cytokines, which are thought to be involved in the pathophysiology of cardiovascular disease and type 2 diabetes (Luecken & Gallo, 2007; Shoelson, Herrero, & Naaz, 2007). Evidence suggests that the life expectancy of obese people is reduced by 5-20 years due to these comorbid conditions (Flegal, Graubard, Williamson, & Gail, 2007). To summarize, obesity is an important public health problem due to its high prevalence and substantial health and economic ramifications.

1.1.2 Pathophysiology of and Traditional Risk Factors for Obesity

Obesity is caused, at the most basic level, by a chronic imbalance between energy intake and energy expenditure (Hofbauer, 2002; Rosenbaum et al., 1997). Energy intake is comprised of the fuel people consume, usually in the form of food and beverages, and it is measured in calories. Energy expenditure is the energy burned by bodily functions (i.e., basal metabolism and thermogenesis), lifestyle activities (i.e., walking up stairs), and formal exercise (i.e., going for a run). In normal weight individuals, energy intake is typically balanced with energy expenditure (Lenard & Berthoud, 2008). If an individual consumes more energy than he or she expends, then the excess energy is converted into triglycerides, the fat deposits of the body, which are stored in adipocytes (Racette, Deusinger, & Deusinger, 2003). The balance between triglyceride synthesis (lipogenesis) and metabolism (lipolysis) into fatty acids and glycerol determines the amount of lipid storage within the adipocyte (Barness et al., 2007). A chronic imbalance between energy intake and energy expenditure can result in ongoing lipogenesis and resultant increases in adiposity. Although body fat mass is primarily determined by the energy intake-expenditure balance, body weight can be influenced by several other risk factors, including genetics, neuroendocrine function, lifestyle behaviors, and environmental factors (Aronne, Nelinson, & Lillo, 2009).

It is estimated that genetic factors account for 25-40% of individual differences in body mass (Ravussin & Bouchard, 2000). A genetic vulnerability may result in obesity when it is paired with high energy intake and low energy expenditure. Neel (1962) coined the term “thrifty gene” to describe a genetic predisposition that allowed some people to more effectively store extra energy in adipose tissue. From an evolutionary perspective, this predisposition was an adaptive trait that promoted survival and reproduction. When food is plentiful, individuals with the “thrifty gene” are better able to store excess energy, which can be used for energy during times of famine (Barness et al., 2007; Hill & Peters, 1998). However, in western society where food scarcity and famine are rare, an enhanced ability to store energy may promote the development of obesity.

Neuroendocrine dysregulation – namely leptin and insulin dysregulation – has also been implicated in the development of obesity. Leptin is synthesized in adipose tissue, and it communicates information about satiety and adiposity stores to the hypothalamus (Barness et al., 2007). Leptin is produced in greater quantities in individuals with higher adiposity (Hofbauer, 2002). Excess circulating leptin causes the leptin receptors in the hypothalamus to become less responsive, resulting in reduced transmission of the satiety signal. This dysregulation can result in obese individuals not properly experiencing fullness, which may promote increased food consumption and obesity (Caro, Sinha, Kolaczynski, Zhang, & Considine, 1996). Insulin is secreted by the pancreas following the consumption of food (Shepherd & Kahn, 1999). It promotes the conversion of energy into triglycerides for storage in adipocytes and inhibits the breakdown of triglycerides for energy consumption (Barness et al., 2007). As adiposity increases, insulin levels in the blood also increase, which facilitates the conversion of energy into triglycerides and prevents the breakdown of stored triglycerides (Barness et al., 2007); therefore excess insulin may contribute to the development of obesity.

Demographic factors are also risk factors for obesity (Labarthe, 1998). Specifically, older individuals are at increased risk; more than 70% of adults aged 60+ years are overweight or obese, which is notably higher than among younger adults (Wang

& Beydoun, 2007). Further, racial or ethnic minorities are at increased risk of obesity, as indicated by high prevalence rates among African Americans and Mexican Americans (Wang & Beydoun, 2007). Although being female has previously been considered a risk factor for obesity, recent evidence suggests that the rates of obesity are essentially equal among men and women (Flegal et al., 2012). Socioeconomic status (SES) is also likely a risk factor for obesity, with individuals of lower SES, especially women, being at elevated risk (McLaren, 2007; Sobal & Stunkard, 1989).

Lifestyle behaviors, such as the quality and quantity of food consumed (energy intake) and the amount of physical activity engaged in (energy expenditure), also contribute to the development of obesity. In addition, lifestyle behaviors related to eating and physical activity can be influenced by environmental factors. Generally, people are consuming more calories now than in the past. Between 1970 and 2003, average daily energy intake increased by 523 calories (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2005). There has also been a shift over the last 100 years to deriving more calories from fat (Faulconbridge & Wadden, 2010), which has a higher caloric density than other macronutrients. Over time, food has become more accessible and portion sizes have increased (Young & Nestle, 2002). Insufficient physical activity has been proposed as a lifestyle behavior contributing to the development of obesity (Rising et al., 1994). For instance, frequent television watching has been associated with increased adiposity later in life (Parsons, Manor, & Power, 2008). Additionally, normal weight people, on average, walk 152 minutes/day more than obese individuals (Levine et al., 2005). Of note, other evidence indicates that physical activity accounts for only 10% of energy expenditure (Barness et al., 2007). Because energy expenditure has remained relatively constant over the past 30 years, increases in obesity during this period appear to be largely due to increased energy intake (Westerterp & Speakman, 2008).

1.2 Attentional Bias for Food Cues as a Potential Risk Factor for Obesity

In addition to lifestyle behaviors, other psychological factors may contribute to obesity development. One such psychological factor that has received increased attention in recent years is attentional bias for food cues. In the following subsections, I define attentional bias, review the development and assessment of food attentional biases, discuss the possible role of food attentional biases in obesity development, and review two candidate mechanisms (i.e., increased energy intake and external eating) that may underlie the food attentional bias-obesity relationship.

1.2.1 Definition of Attentional Bias

Attentional bias is a form of cognitive bias in which an individual pays more attention to salient environmental stimuli (Faunce, 2002), such as those that have been associated with a rewarding outcome repeatedly over time. Attentional biases have been identified for a variety of stimuli. The most commonly researched domains of attentional bias are drug use (Copersino et al., 2004; Franken, Kroon, & Hendriks, 2000; Hester, Dixon, & Garavan, 2006; Lubman, Peters, Mogg, Bradley, & Deakin, 2000), alcohol use (Field, Mogg, Zetteler, & Bradley, 2004; Lusher, Chandler, & Ball, 2004), tobacco use (Bradley, Mogg, Wright, & Field, 2003; Field & Cox, 2008; Johnsen, Thayer, Laberg, & Asbjornsen, 1997), eating disorders (Dobson & Dozois, 2004), and depression and anxiety (Williams, Mathews, & MacLeod, 1996). A less researched domain is attentional biases for food cues (Castellanos et al., 2009; Mogg, Bradley, Hyare, & Lee, 1998; Nijs, Muris, Euser, & Franken, 2010; Werthmann et al., 2011). To illustrate this form of attentional bias, an individual with a food attentional bias would be more likely to notice cookies sitting on a table or an image of a cheeseburger on a billboard advertisement than someone who does not have this bias. For these individuals, food-related stimuli have become more salient.

1.2.2 Development of Food Attentional Biases

The development of food attentional biases may be best explained by describing reward processes and how these processes are thought to result in increased attention to rewarding stimuli. Neurological processing of pleasure and reward occurs in the mesocorticolimbic dopamine system (Kelley & Berridge, 2002). In this system, dopamine regulates communication between several brain structures, including the ventral tegmental area (VTA), ventral striatum, and nucleus accumbens (NAc) (Kelley & Berridge, 2002). Higher levels of dopamine activate these regions and result in the experience of reward and pleasure (Kelley & Berridge, 2002; Wise, 1998).

One theory regarding the development of attentional biases is the incentive sensitization theory of addiction, which includes processes of neuroadaptation and classical conditioning. According to this theory, addictive substances and other rewarding stimuli activate the release of dopamine in the VTA and NAc (Robinson & Berridge, 1993; Wise, 1996), which produces a pleasure sensation. Over repeated exposure, neuroadaptations occur in the mesocorticolimbic dopamine system in susceptible individuals (Robinson & Berridge, 1993). These neuroadaptations result in hypersensitivity to the substances, which is characterized by an increase in dopamine release in response to the substances. Classical conditioning also contributes to incentive sensitization (Robinson & Berridge, 1993). An association is formed between the addictive substance (unconditioned stimulus; UCS) and increased dopaminergic activity (unconditioned response; UCR). Over repeated exposure to the substance (UCS), increased dopaminergic activity (UCR) becomes associated with other stimuli in the environment (conditioned stimulus; CS). Once an association has been learned, the CS has the power to elicit dopaminergic responses, cravings, and substance-seeking behaviors (conditioned responses; CR) (Kiyatkin & Stein, 1996; Robinson & Berridge, 1993; Schiff, 1982). The ability of the UCS and CS to elicit dopaminergic responses and pleasure sensations results in an individual paying more attention to these stimuli in their environment.

The incentive sensitization theory has also been used to explain the development of food attentional biases (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Although food does not cause physical dependence in the same way addictive substances do, it does have strong incentive properties (Tapper, Pothos, Fadardi, & Ziori, 2008) and can activate the mesocorticolimbic dopaminergic system (Cannon & Bseikri, 2004; Fadardi & Bazzaz, 2011; Robinson & Berridge, 2001). Over repeated exposure to the paired association of food (UCS) and dopaminergic activation (UCR), food cues (CS) can develop incentive salience and elicit a dopamine response (CR). Food cues may include the sight, smell, or taste of food as well as associated stimuli, such as packaging, eating routines, and cutlery (Hermans et al., 2012). Sensitization of the reward system in response to food cues can cause individuals to pay more attention to those cues in their environment, which can lead to food cravings and potentially eating in response to those cues (external eating), increased energy intake, and obesity (Berridge et al., 2010).

1.2.3 Measurement of Attentional Biases

The three most common approaches to assessing food attentional biases are the modified Stroop tasks, visual dot-probe tasks, and eye tracking paradigms. Modified Stroop tasks have the longest history of use in the measurement of attentional biases (Kemps & Tiggemann, 2009). These tasks were adapted from the original Stroop task, in which participants are presented with a series of cards that have the name of a color written in a different color ink (Stroop, 1935). The participant's task is to name the color of the ink (Stroop, 1935). In research on food attentional biases, the Stroop task has been modified by comparing response latencies in color naming for food-related ("cake") and neutral ("pencil") words (Williams et al., 1996). It is thought that a delay in color naming indicates the presence of cognitive interference resulting from the word content drawing the participant's attention. Therefore, a delay may indicate attentional bias to the content of a word. However, it has argued that the delay may result from avoidance of, rather than attention to, the content-relevant cue (de Ruiter & Brosschot, 1994; Phelan et al., 2011). For instance, if an obese person feels that he or she should avoid food in order to lose weight, he or she may initially notice the food content of a cue and then try to avoid that cue, which results in a slower response time. Because the Stroop task may assess attentional bias toward and away from content-relevant cues, it is difficult to identify which process is occurring.

In visual dot-probe tasks, a series of paired cues are displayed on a computer screen. Either images or words can be used as cues, but images are considered to be more ecologically valid (Brignell, Griffiths, Bradley, & Mogg, 2009). These paired cues include one content-related cue (“cake”) and one neutral cue (“pencil”) presented simultaneously. After the presentation of each set of paired cues, a dot-probe appears in the same location as either the content-relevant or neutral cue. Once the dot-probe appears, the participant’s task is to identify the orientation of the dot-probe by pressing a key as quickly as possible. Reaction time latencies for dot-probes presented under content-related cues are compared to those of neutral cues. It is thought that, because people with attentional biases tend to be looking at content-related cues at picture offset, they would be able to respond more quickly to dot-probes presented under the content-related cue versus the neutral cues. Therefore, in the measurement of food attentional bias, faster reaction times to probes under food cues compared to non-food cues are considered to reflect food attentional bias (e.g., Mogg et al., 1998).

Although the visual dot-probe task is a validated measure of attentional biases, there is an important limitation. This task may assess different components of attentional bias depending on the duration of the image presentation. For instance, a short cue duration (< 100 ms) is intended to measure the automatic attentional response, but it may actually measure where the participant happened to be looking at picture onset (e.g., Nijs et al., 2010). In contrast, with longer cue durations (> 200 ms), participants may shift their gaze back and forth between the images, and reaction time may reflect the image the participant happened to be looking at during picture offset (Nijs & Franken, 2012). Dot-probe tasks using longer stimulus durations (> 1250 ms) are said to be tapping into sustained attentional processes (Bradley et al., 2003).

Eye tracking, a newer approach to assessing food attentional biases, records eye movements while food and neutral cues are simultaneously presented on a computer screen. When the participant looks at the computer monitor, light enters the eye, and some of that light is reflected as it hits the cornea and the retina. Eye tracking devices use these reflections to determine where the participant is looking, which is then mapped onto the computer screen. When used in food attentional bias research, eye tracking provides information about which image (food or non-food) the participant looked at first (gaze direction) and how long the participant looked at food or non-food images (gaze duration). Because eye tracking monitors the participant’s gaze throughout the entire trial, it may be a more comprehensive measure of attentional bias than are dot-probe tasks (Nijs & Franken, 2012). Although it has been proposed that eye tracking is the most direct and comprehensive approach to assessing attentional biases (Hermans et al., 2012), it assumes that the act of looking at an image indicates attention.

1.2.4 Possible Role of Food Attentional Biases in Obesity

It has been proposed that food attentional biases may lead to increased food cravings, eating behaviors, and energy intake, which could result in the development of obesity over time (Berridge et al., 2010). Therefore, individual differences in food attentional biases may partially explain why some people develop obesity and others do not (Berridge et al., 2010).

Recent empirical findings provide initial support for this theory. A study conducted by Castellanos et al. (2009) examined food attentional biases in 18 obese and 18 normal weight women who completed a visual dot-probe task in both hunger and satiety conditions. Multiple measures of attentional bias were obtained during the task: reaction times during a visual dot-probe task and gaze direction and duration assessed by eye tracking. These researchers found that, in the hunger condition, both obese and normal weight women had increased food attentional bias, as indicated by both direction and duration biases for food cues. In the satiety condition, obese women had increased attentional bias for food cues (direction and duration bias), whereas normal weight women showed no such bias. No attentional bias was found for either group in either condition using the reaction time data.

In a similar study, 26 obese/overweight and 40 normal weight women who were randomly assigned to hunger and satiety conditions completed a visual dot-probe task with eye tracking (Nijs et al., 2010). It was found that all women, regardless of weight group or condition, had attentional bias for food cues as indicated by higher direction bias and duration bias scores as well as faster reaction times to food images in the dot-probe task. The dot-probe task also showed that obese/overweight women had faster reaction times and, thus, greater attentional bias to food cues than normal-weight women across both hunger and satiety conditions. These findings suggest that, although all women may exhibit food attentional bias, this bias may be stronger in obese/overweight women.

A third study involving 22 overweight/obese and 29 normal weight women examined food attentional biases as well as craving and overeating (Werthmann et al., 2011). Similar to the other studies, participants completed a visual probe task with eye tracking. In addition, craving was measured by self-report and overconsumption was measured by tracking food intake during a fake taste-test. Overweight/obese women had higher rates of direction bias than normal weight women, but no group differences were observed for duration bias or reaction time. Among obese/overweight women, self-reported craving was positively correlated with direction bias, which was not found in the normal weight group. This association suggests that food attentional bias may lead to food craving.

In summary, these three studies provide preliminary evidence that obese/overweight women pay more attention to environmental food cues than do normal weight women. Although all women may exhibit food attentional bias when hungry, this bias may be more chronically present among obese/overweight individuals. Initial evidence also suggests that food attentional bias may be associated with increased food craving.

In these studies, the findings are mixed across the various measures of food attentional bias, possibility due to differences in stimulus duration and approach-avoidance behavior. First, the duration of stimulus presentation across studies ranged from 500-2000 ms. A longer stimulus presentation would allow for shifting of gaze between one image and the other. Therefore, reaction time could reflect where the participant happened to be looking during picture offset. For instance, the longer stimulus duration of 2000 ms in Werthmann et al. (2011) may explain why there were no group differences in reaction time measures. Second, inconsistent findings across studies may also be due to approach-avoidance behavior to food cues among obese individuals. Approach-avoidance behavior can be best illustrated by the results of Werthmann et al.

(2011). In that study, obese individuals tended look at the food image first (approach behavior) but then diverted their attention away from the image (avoidance behavior), resulting direction bias but not duration bias. Theoretically, approach-avoidance behavior to food cues occurs due to the conflict between having an attentional bias for food cues and also being motivated to lose weight (Macht, Gerer, & Ellgring, 2003). Consequently, direction bias, but not duration bias and reaction time bias, can be used to detect attentional biases in individuals avoiding food cues. This could explain why Castellanos et al. (2009) and Werthmann et al. (2011) detected differences in direction bias, but not duration bias and reaction times, when comparing obese/overweight women to normal weight women.

1.2.5 Increased Energy Intake and External Eating as Candidate Mediators of the Food Attentional Bias-Obesity Relationship

Two possible mechanisms that may underlie the relationship between food attentional bias and obesity are increased energy intake and external eating. Preliminary findings suggest that food attentional biases are associated with increased food cravings (Werthmann et al., 2011), which in turn could produce increases in eating behaviors and energy intake. Energy intake refers to the fuel people consume measured in calories (Faulconbridge & Wadden, 2010). To date, only two studies have examined the association between food attentional bias and overeating (Nijs & Franken, 2012). Werthmann et al. (2011) measured energy intake during a fake taste test that required participants taste four different foods (chocolate, biscuits, chips, and salted peanuts) and rate them on attractiveness, smell, and taste. Obese/overweight participants had greater food attentional bias and energy intake than did normal-weight participants; however, there was not a significant correlation between food attentional bias and energy intake. A second study using a similar fake taste test among 26 obese/overweight and 40 normal weight women also found that measures of food attentional bias did not significantly correlate with energy intake (r = .07 for reaction time; r = .10 for direction bias; r = .24 for duration bias) (Nijs et al., 2010); however, these correlations are small to moderate in size and could have been significant in a larger sample with more power. Because the laboratory setting and fake taste test context may have limited the ecological validity of these assessments of energy intake, there is a need for studies utilizing measures that reflect average energy intake during daily life. Furthermore, studies testing energy intake as a candidate mediator of the association between food attentional bias and adiposity are needed.

A second candidate mediator is external eating, defined as the tendency to eat in response to external food cues (Hou et al., 2011). Theoretically, a person with elevated food attentional bias and external eating may be more likely to notice food cues in the environment and eat in response to those cues, which could result in overeating, increased energy intake, and obesity. The few studies that have examined the relationship between food attentional bias and external eating have yielded mixed results. In a visual probe task using food-related and neutral words, individuals high on self-reported external eating were more likely to direct their attention away from food cues than those who are low on external eating (Johansson, Ghaderi, & Andersson, 2004). In another study using a visual dot-probe task using food-related and neutral images, food attentional bias was greater in high-external eaters than in low-external eaters (Hepworth, Mogg, Brignell, & Bradley, 2010). These divergent findings may be due to methodological factors, such as the use of words versus images. It has been proposed that assessments using pictorial food-cues may have more ecological validity than those using word cues (Nijs & Franken, 2012). Because adiposity has not been incorporated into prior studies in this area, there is a need not only to further investigate the relationship between food attentional biases and external eating, but also to test external eating as a candidate mediator of the food attentional bias-adiposity association.

1.3 The Present Study

Despite preliminary evidence of a positive relationship between food attentional bias and adiposity, there are several gaps that need to be addressed. First, the relationship between food attentional bias and adiposity needs to be further clarified. This literature consists of few studies, and the results have been somewhat mixed. Second, it is unknown whether food attentional biases are associated with increased energy intake in daily life and increased external eating. These literatures are also small and contain mixed findings. Third, although energy intake and external eating have been implicated in the food attentional bias-adiposity relationship, they have not been formally tested as mediators.

Accordingly, the primary objective of the present study is to examine the association between three measures of attentional bias for food cues (direction bias, duration bias, and reaction time bias) and adiposity (body mass index and body fat percent), as well the role of two candidate mediators – energy intake and external eating.

The conceptual model guiding this study is depicted in Figure 1. To achieve this objective, six hypotheses were tested:

Hypothesis 1: Measures of attentional bias for food cues (direction bias, duration bias, and reaction time) are positively related to BMI (H_1).

Hypothesis 2: Measures of attentional bias for food cues are positively related to body fat percent (H_2).

Hypothesis 3: Measures of attentional bias for food cues are positively related to energy intake (H_3).

Hypothesis 4: Measures of attentional bias for food cues are positively related to external eating (H_4).

Hypothesis 5: Energy intake partially mediates the relationship between food attentional bias and two measures of adiposity - BMI and body fat percent (H_5).

Hypothesis 6: External eating partially mediates the relationship between food attentional bias and two measures of adiposity - BMI and body fat percent (H_6).

CHAPTER 2. METHODS

2.1 Design

The present study was a cross-sectional laboratory study. It utilized data that were collected as a part of an ongoing study, “Attentional Biases to Food-related Stimuli as a Potential Mechanism of the Depression-to-Obesity Relationship” (IRB Protocol #: 1112007694). This study began in February 2012, and I have served as the project coordinator since August 2012. Out of the 120 participants in this study, I conducted data collection on 59 participants, and I supervised a research assistant who collected data on 25 participants.

2.2 Participants

Participants were 120 undergraduate students at IUPUI who were seeking research credit for a psychology course. Students elected to participate through SONA, the Psychology Department’s undergraduate research recruitment website (http://iupui.sona-systems.com/; Retrieved January 13, 2014). Students were not eligible if they were < 18 years of age, pregnant, or taking antidepressant medication. Participants who were pregnant were not eligible because BMI and body fat percent among pregnant women may not accurately reflect adiposity. Participants taking antidepressants were not eligible because these medications could confound the depression-obesity relationship, which is the focus of the parent study. An undergraduate student sample was appropriate for this study, given that this is a critical age at which young adults have increased autonomy regarding food choices.

2.3 Measures

2.3.1 Attentional Bias

Several measures of attentional bias were obtained. Dot-probe reaction time was assessed using a computerized task adapted from Castellanos et al. (2009). In this task, a fixation cross was presented in the middle of the computer screen for 1,000 ms. Next, a pair of images were presented side by side, one on the left and one on the right, for 2,000 ms. A total of 60 image pairs were presented. Forty of the image pairs were experimental images, which include one food-related image and one non-food related image. These images were matched for color, shape, and size (see Figure 2 for a sample image pair). The remaining 20 image pairs were control images, comprised of nature scenes that were matched for color, shape, and size. A dot-probe appeared underneath one of the two images and remained on the screen until the participants pressed either a “1” or a “2” on the keyboard. At the start of the task, participants were instructed to respond to the probe quickly and accurately by pressing either a “1” for dots that are up and down (“:”) or a “2” for dots that are side-by-side (“..”). Dot-probe reaction time was computed by subtracting mean reaction time to dots presented under food images from mean reaction time to dots presented under non-food images. Positive values indicate attentional bias toward food images, and negative values indicate attentional bias away from food images.

Eye tracking data were collected using the EyeTrac® Series 6 Desk-mounted Eye Tracking Device. This device recorded participants' gaze location as they look at the computer screen. To ensure proper recording of gaze, participant's eye movements were calibrated using a nine-point procedure. Nine dots were presented on the screen in three rows of three, and participants were asked to look at each dot consecutively while their eye gaze location was tracked. Eye movements were recorded as participants completed the visual dot-probe task. Using computer software, Applied Science Laboratories (ASL) Results, I identified food-related and non-food-related images as "areas of interest" within the visual dot-probe task. For each experimental image pair, ASL Results determined the number and duration of gaze fixations (gazes lasting ≥ 100 ms) within the food and non-food image areas of interest. Fixations were used to calculate two indices of attentional bias: direction bias and duration bias. Direction bias was computed as a proportion of total paired image presentations in which the participant's first gaze was on the food image. Values over 0.5 indicate a direction bias for food images. Duration bias was calculated as the proportion of time that the participant spent looking at food images divided by the total time spent looking at either food or non-food images. Values over 0.5 indicate a duration bias for food images.

2.3.2 Adiposity

Two measures of adiposity were obtained: BMI and body fat percent. BMI was calculated as weight in kilograms divided by height in meters squared. Height and weight were measured using a standard medical scale. Body fat percent was measured using a Tanita Body Composition Analyzer Model TBF-300A. Body fat percent was measured by having a participant make contact with a metal conductor, which sends a low current through the body. The degree to which the current is impeded indicates the quantity of body fat because the current moves more easily through muscle and other tissues than through adipose tissue. Evidence supports body fat percent as a valid measure for individuals who are adequately hydrated with BMIs between 16 and 34 (Kyle, Morabia, Schutz, & Pichard, 2004).

2.3.3 Energy Intake

To measure daily energy intake, participants completed the Automated Self-Administered 24 Hour Recall (ASA24). The ASA24 is a web-based tool developed by the National Cancer Institute (NCI) that allows researchers to measure dietary intake over the previous 24 hours among adults. This tool is highly interactive and has a user-friendly interface. The present study was approved by the NCI ASA24 administrators to use the tool free of charge. The format and design of the ASA24 are based on the Automated Multiple Pass Method (AMPM) dietary recall, an interviewer-administered dietary recall developed for use in the National Health and Nutrition Examination Survey. The AMPM approach has been established as a valid measure for obtaining usual energy intake (Moshfegh et al., 2008).

The ASA24 asks participants to report their food intake systematically to minimize accidental omission, overreporting, or underreporting. First, participants are asked to report what meals they have eaten in the past 24 hours (i.e., breakfast, lunch, dinner, and snacks). Next, the program asks participants to provide a list of foods and beverages consumed during each meal. Participants search for foods using a comprehensive food database that was adapted from the AMPM recall protocol. The program also prompts participants about food items that may have been overlooked. For instance, if the participant reported eating chicken at dinner, the program would ask the participant how that chicken was prepared (baked vs. fried, seasoning and oils used, etc.). After the participants have identified all food items, they are asked to indicate the quantity and portion size of each food item. Then, the program asks about commonly overlooked foods, such as water, juice, coffee, and small snacks. Finally, there is a review of the day's intake. Throughout the entire process, the participants can go back and amend any of the food items they previously entered. It is important to note that the ASA24 uses an automated guide to narrate the instructions to the participants in a user-friendly, interactive fashion.

In the present study, participants were asked to complete the ASA24 on two occasions, during the in-person data collection session and remotely one week later. For this second administration, login information was emailed to the participant, who was asked to complete the measure from any internet-connected computer. If participants did not complete the second recall within one week, a reminder email was sent. No further contact was made after the second email. Research supports collecting 24-hour dietary recall on two days for two reasons: (1) it reduces the chance that the data collected were from an unusual day and (2) multiple recalls increases reliability of summary measures (Blanton, Moshfegh, Baer, & Kretsch, 2006). Various indices of dietary intake are automatically generated by the ASA24 system. I used total kilocalories (kCal) from the ASA24 as in index of daily energy intake. The ASA24 was added to the study protocol on November 30, 2012. Among the 120 study participants, 75 (62.5%) completed day 1 and 41 (34.2% of the entire sample, 54.7% of participants who completed day 1) completed day 2. Among the participants who completed day 2, the average time between administrations was 13.3 days. Data from day 1 were used in the present study due to the low response rate for day 2. Of note, there was a large correlation between day 1 and day 2 kilocalories in the high-quality eye tracking data sample ($r = .82$, $p < .001$).

2.3.4 External Eating

External eating was measured using the Dutch Eating Behavior Questionnaire (DEBQ) (van Strien, Frijters, Bergers, & Defares, 1986). The DEBQ is a 33-item questionnaire that asks participants to endorse how often they engage in certain eating behaviors, ranging from never to very often. This questionnaire was originally developed in Dutch but has since been translated into English (Wardle, 1987). Factor analysis supports a three-factor structure, comprised of restrained eating, emotional eating, and external eating subscales (van Strien et al., 1986). Restrained eating, or limiting dietary intake with the aim of losing weight, is measured by 10 items. Emotional eating, or eating in response to an emotional state, is measured by 13 items. External eating, or eating in response to external stimuli, is measured by 10 items. Internal consistency is acceptable for each of these factors, evidenced by Cronbach's α of .95 for restrained eating, .94 for emotional eating, and .80 for external eating (van Strien et al., 1986). Further, this measure has been shown to be equally reliable in obese and non-obese samples (van Strien et al., 1986). In the present study, the DEBQ external eating subscale was used to assess external eating. Mean scores for external eating were calculated the average of the 10 items that load onto that factor

2.3.5 Covariates

Data regarding demographic factors (age, gender, and race/ethnicity) were collected using an online survey comprised of standard questions. Because research supports a relationship between hunger and food attentional bias (Castellanos et al., 2009; Mogg et al., 1998; Placanica, Faunce, & Soames Job, 2002), a visual analogue scale (VAS) was administered to assesses current hunger level (“How hungry do you feel?” and “How strong is your urge to eat?”). For both questions, participants were instructed to rate level of agreement along a linear continuum line, ranging from “Not very” to “Very much.” There is evidence to support the use of these items in visual analogue form in the assessment of appetite and hunger, with correlations to subsequent energy intake ranging from 0.50 to 0.53 (Flint, Raben, Blundell, & Astrup, 2000). Mean VAS scores were computed by averaging values for the two hunger questions.

Physical activity was measured using the International Physical Activity Questionnaire (IPAQ), a quantity-frequency measure of weekly physical activity in work and recreational activities. The measure asks participants to report the number of days per week and hours per day they engage in vigorous activity, moderate activity, walking, and sedentary activity. For each level of activity, a unique metabolic rate (MET) constant is multiplied by the time spent on that level of activity times the number of days per week engaged in that activity level. The values for each activity level are then summed to produce an overall MET value, which is an estimate of metabolic intensity of activities over the past week. In a multi-site validation study, the IPAQ was found to have reasonable test-retest reliability, indicated by 75% of sites reporting reliability coefficients above 0.65 (Craig et al., 2003). Criterion validity was demonstrated by a fair to moderate agreement between the IPAQ and an accelerometer measure of physical activity (Craig et al., 2003). Although the IPAQ was proposed as a covariate in the present study, several extreme outliers raised serious concerns regarding the accuracy of the data collected. Specifically, 6 participants reported spending an average of more than 24 hours a day engaged in physical activity, and 17 additional participants reported spending more than 12 hours a day engaged in physical activity. This issue may have resulted from the wording of the items or the construction of the IPAQ, especially in its adaptation to the online format. Based on an examination of the data and subsequent pilot testing of the self-report online version of the IPAQ, a common issue I observed was that some participants reported hours per week for items that ask for hours per day. For example, a participant may report engaging in vigorous activity 7 days per week and spending 7 hours per day on this activity. Although the participant likely meant that he or she does 1 hour of vigorous activity on each day, totaling to 7 hours across the week, his/her data would be coded as 49 hours (7 days/week x 7 hours/day) of vigorous activity per week. Another common issue was that participants wrote values in the wrong text box in the online format. For instance, one participant reported walking for 55 hours and 0 minutes per day, but they probably meant 0 hours and 55 minutes per day. Due to these concerns, I decided not to include the IPAQ total score as a covariate.

2.4 Procedure

Participants completed a 1-hour 40-minute laboratory session. In addition, participants were asked to complete a 20-minute follow-up one week later, which could be completed online from home. At the start of the session, participants provided written informed consent for all study procedures. Then participants completed the visual analogue scale to assess current hunger and the Patient Health Questionnaire-8 to assess depressive symptom severity, which was being examined in the parent study.

Next, each participant was escorted to the laboratory where the food attentional bias assessment was conducted. The participant sat at a computer desk and was oriented to the desk-mounted eye tracking camera and chin rest that was used to minimize head movement. The research assistant explained the order of events for the computer task: the participant will practice the task, the eye tracking camera will be set up and calibrated, and the participant will complete the task while reaction time and eye tracking data are collected. The practice visual dot-probe task was identical to the experimental task other than the images being shapes rather than the food and non-food images. The research assistant next described the practice task instructions to the participant. Once the task was understood, the research assistant left the room and allowed the participant to complete the practice task independently. If the participant scored below 85%, the practice task was repeated until a score of 85% or higher was attained. Next, the research assistant calibrated the eye tracking device. Settings were adjusted on the eye tracker until adequate readings of pupil and corneal reflection were obtained. Then, the research assistant set up the calibration screen, described the calibration process to the participant, and conducted the calibration. If the program accepted the calibration, the research assistant proceeded. If the program did not accept the calibration, the settings on the eye tracker were further adjusted to obtain proper readings of pupil and corneal reflection.

Once the eye tracking camera was calibrated, then the experimental visual dot-probe task was completed. For details regarding this task, see the Attentional Bias subsection of the Measures section above.

After completing the visual dot-probe task with eye tracking, the participant completed a battery of computerized questionnaires on SurveyMonkey, a password-protected website. This battery included the standard questions assessing demographic factors, as well as questionnaires assessing external eating (DEBQ) and physical activity (IPAQ). The participant was next directed to the web-based ASA24, where he or she was asked to recall dietary intake for the previous 24 hours. The research assistant measured the participant's height, weight, and body fat percent using a standard medical scale and a body composition analyzer. Then, the researcher informed the participant that he or she would receive an email invitation in a week to complete the ASA24 a second time. Finally, the research assistant provided the debriefing document, which explains the purpose of the study, and a copy of the informed consent. One week after the completion of the study, participants were contacted via email to complete the second ASA24. If participants did not complete the dietary recall within seven days, one follow-up email was sent.

2.5 Data Analysis

2.5.1 Data Cleaning and Reduction

Frequencies were run on all raw variables to assess for out-of-range values. When out-of-range values were found, I checked for data entry errors. Across all variables, except for the IPAQ variables, no out-of-range values were identified. Normality was assessed for all continuous variables, ensuring that skewness was less than 3.0 and kurtosis is less than 10.0 (Kline, 2004). Data completeness was also examined.

All relevant variables were calculated and checked for outliers (z scores ≥ 3.0). BMI was computed as weight in kilograms divided by height in meters squared (Roche et al., 1981), and body fat percent was generated by the Tanita Body Composition Analyzer. The energy intake variable was derived from day 1 of the ASA24, which automatically generated a value for kilocalories. One outlier was found for the ASA24 kilocalories variable (5,454 kilocalories, z score = 3.38), which was retained because it was a possible value and it did not impair normality. Based on van Strein and colleague's (1986) three-factor structure for the DEBQ, the external eating subscale was computed as the mean of items 24, 25, 26, 27, 28, 29, 30, 31, 32, and 33. Prior to computing the external eating subscale, within-subject mean imputation was performed for 6 participants missing only one item on the DEBQ. Age was computed from date of birth and is reported in years. Four outliers were identified (adults aged 42.5-51.4 years) but were retained. Sex was coded as 0 = female and 1 = male, and a 7-level race/ethnicity variable (1 = White, 2 = Black, 3 = Hispanic/Latino, 4 = Asian, 5 = Native Hawaiian/Pacific Islander, 6 = American Indian, Alaskan Native, 7 = Other) was recoded as a dichotomous variable (0 = non-white, 1 = white) due to the low number of participants in the non-white race/ethnicity groups in the high-quality eye data sample (2 Black, 4 Hispanic/Latino, 3 Asian, 1 American Indian/Alaskan Native, and 4 Other participants). Subjective hunger score was computed as the mean of VAS ratings for the items "How hungry do you feel?" and "How strong is your urge to eat?"

The three variables for food attentional bias required notable cleaning and reduction. For the reaction time bias variable, prior to computing bias scores, reaction time data for each image pair (event) was examined. Reaction time events were excluded when the participant responded incorrectly to the dot-probe (92 events, 1.06% of events) (Nijs et al., 2010). The modal number of errors was 0, and 93.2% of participants had 2 or fewer errors. Reaction time events with durations lasting less than 200 ms or longer than 1500 ms were also excluded (28 events, 0.32% of events) (Nijs et al., 2010). For all events that remained, reaction time bias scores were computed (see Methods section). Data for two participants was lost due to improper saving of the dot-probe reaction time task, and one participant was excluded due to an extreme low value (reaction time bias score = -183.13, z-score = -3.71). Accordingly, reaction time data is reported for 117 participants (97.5%).

For variables using eye tracking (direction bias and duration bias), data quality was examined at the event level and at the participant level. At the event level, ASL Results automatically generates direction and duration of gaze fixations in each area of interest (food image and non-food image regions of the computer monitor) and outside of the areas of interest. When data are not reported for an event, that indicates that ASL Results did not detect a gaze fixation that remained stable for ≥ 100 ms in any area of the computer monitor during that image pair presentation. Only 23 participants (19.2%) had eye tracking data for all 40 experimental events, and 74 participants (61.7%) had data for 20 or more experimental events (50% of events).

For each participant who was missing data for one or more events, I examined the participant’s raw data to identify why fixations were not detected. Two common sources of data loss were identified: (1) poor measurement of the pupil or corneal reflections and (2) extreme gaze coordinate values. Pupil or corneal reflection measurement are established during the calibration process, but these measurements may be lost after calibration. From my observations during eye tracking sessions, some cases of pupil and corneal reflection loss occurred as the result of participant head movement or changes in brightness of the computer monitor (e.g., transitioning from the dark calibration screen to a light task screen), which changed the pupil dilation. In addition, the eye tracker appeared to have difficulty maintaining measurement of the pupil reflection for some participants with darker iris colors. For many participants, loss of pupil or corneal reflection resulted in missing data for part of an event or for only a few events. In more extreme cases, loss of pupil or corneal reflection resulted in missing data for most of the experimental image pair events. Extreme gaze coordinate values, the second source of data loss, occurs when the eye gaze is read to be outside of the region of the computer monitor. ASL Results did not report data for those samples. From my observations, it appeared that extreme gaze coordinate values sometimes result from the participant looking away from the computer monitor or the eye tracking equipment measuring a reflection on glasses lenses.

Heat maps and fixation plots were also spot checked to ensure that they matched event data for gaze direction and duration. For instance, Figure 3 displays the fixation plots and heat maps for a participant who had a direction bias value of .54 and a duration bias value of .55, suggesting a slight attentional bias toward food cues. Notice that the fixation plots show more fixations in the food image area of interest, and the heat maps show more gradation (red versus green coloration) in the food image area of interest.

After examining the quality of the data at the event level, I examined the quality of the data at the participant level. First, I computed the total number of experimental events in which any eye tracking data were collected for each participant. Then, for each event, I examined whether the participant looked at the areas of interest (food or non-food images). I excluded participants with eye tracking data for fewer than 20 experimental events in which they looked at either a food or non-food area of interest. Excluding these individuals was designed to exclude participants with major data quality issues, including: 1) not acting naturally (e.g., keeping their fixation in the center of the computer screen and avoiding looking at food or non-food images), 2) data loss due to poor quality of measurement of the pupil or corneal reflection, loss of head tracking, and extreme gaze coordinate values (see above more information on data quality issues). I selected fewer than 20 events as a cutoff because it represents < 50% of the experimental events in the task and fewer than 20 events may not adequately capture attentional bias in a representative manner and thus may not provide stable estimates of direction and duration bias.

Among 120 participants who participated in this study, eye tracking data were not recorded for five participants due to inability to calibrate (n = 2) and errors in saving the eye tracking session (n = 3). Forty-one participants were excluded because eye tracking data were not recorded for more than 20 experimental events. Among the 74 participants with eye tracking data for 20 or more events, 11 participants were excluded because they had multiple trials in which they did not look at an area of interest, resulting in their number of events looking at an area of interest being below 20. Accordingly, high-quality eye tracking data, used in the computation of direction and duration bias, were available for 63 participants (55.0%). See the Methods section for computation of direction and duration bias.

Of note, considerable effort went into mitigating problems with eye tracking data loss. After consultation with Applied Science Laboratories (ASL), I employed multiple strategies to improve head tracking and measurement of pupil and corneal reflection. To improve head tracking, I used a chin rest to minimize head movement, and I added a white screen to a black one-way mirror window that is located behind the participant's head to enhance detection of the head. To improve measurement of the pupil and corneal reflection, I used several strategies to reduce glare. First, I lowered the computer brightness to as low as possible while still affording viewing of the task. Second, I revised the calibration screen from a black background to a white background so that the screen brightness remained consistent when transitioning from calibration to the task. Third, I placed an anti-glare screen on the computer monitor to further reduce screen brightness and to reduce the reflective glare from the monitor. Fourth, I placed a black curtain along the wall in front the participant to reduce glare that could be produced by a white wall. Fifth, I dimmed the lights in the room to increase pupil size. These strategies improved my ability to calibrate participants, but data loss still occurred even after these strategies were put into place.

2.5.2 Tests of Hypotheses

Six hypotheses were tested in the present study (see Figure 2). Hypothesis 1 (H_1) was tested using a series of multiple linear regression analyses. Separate models were constructed for each of the three predictor variables – direction bias, duration bias, and reaction time bias. The outcome variable was BMI, and the covariates were age, sex, race/ethnicity, and subjective hunger VAS score. Hypothesis 2 (H_2) was tested employing a parallel set of analyses with body fat percent as the outcome variable. Hypotheses 1 and 2 were tested on a sample of 61 participants. Although eye tracking and visual dot probe reaction time data were collected from all 120 participants, high-quality eye tracking data were obtained from only 63 participants (53%), and usable dot-probe reaction time data were obtained from 117 participants (98%). BMI and body fat percent were measured for all 120 participants. Accordingly, analyses were performed on the 61 participants with high-quality and complete data for all food attentional bias measures. This sub-sample will be called the "high-quality eye tracking data sample" throughout this document. Note that among the 63 participants with high-quality eye tracking data, two participants' data were not included due to improper saving of reaction time data ($n = 1$) or being an outlier on the reaction time bias value ($n = 1$; z-score = -3.17). Also of note, supplemental analyses for Hypotheses 1 and 2 were performed on the sample with usable reaction time data ($n = 117$).

Hypotheses 3 (H_3) and 4 (H_4) were tested using a similar series of multiple linear regression analyses, with separate models for each of the three food attentional bias measures as predictor variables and age, sex, race/ethnicity, and subjective hunger VAS score as covariates. Parallel analyses were conducted for both outcome variables – mean ASA24 total kilocalories (energy intake) and the DEBQ external eating subscale score (external eating). Because the ASA24 was added after data collection for the parent project had begun, energy intake data were obtained from 75 (63%) of the 120 participants who comprised the entire sample. In the high-quality eye tracking data sample, ASA24 data was not recorded for 19 participants, so Hypothesis 3 was tested on a sample of 42 participants. Hypothesis 4 was tested on the sample of 61 participants comprising the high-quality eye tracking data sample.

To test Hypotheses 5 (H_5) and 6 (H_6), Hayes' SPSS bootstrapping macro called PROCESS (Hayes & Preacher, 2013) was used to determine if the indirect effect of food attentional bias on adiposity through either of two candidate mediators – energy intake or external eating – was significant. Separate analyses were conducted for each candidate mediator. Bootstrapping is a nonparametiric approach to effect size estimation that uses resampling to test the indirect effect of a mediator (MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2004). Using sampling with replacement, bootstrapping takes subsamples from the original sample and computes the indirect effect within each subsample. By repeating this process thousands of times, bootstrapping can estimate the shape of the sampling distribution of the indirect effect. From this, the upper and lower estimates of the indirect effect can be identified, and a confidence interval can be computed. Bootstrapping is appropriate to use in the present study for two reasons. First, it does not require that the sampling distribution be normally distributed, which allows for testing mediation in cases where there is asymmetry in that distribution (Hayes & Preacher, 2014; Hayes & Scharkow, 2013; Preacher & Hayes, 2004). Second, it can be used on smaller samples with more confidence because it has greater statistical power while also minimizing the type I error rate (Hayes & Preacher, 2014; Hayes & Scharkow, 2013). For this study, a 95% confidence interval was used with 10,000 bootstrap resamples. I am able to conclude that a variable is a significant mediator if the upper and lower bounds of the 95% confidence interval do not include the value of zero. Hypothesis 5 was tested using the sample of 42 participants with high-quality eye tracking data who completed the ASA24. Hypothesis 6 was tested using the sample of 61 participants comprising the high-quality eye tracking data sample.

CHAPTER 3. RESULTS

3.1 Characteristics of Participants

One hundred and twenty undergraduate students were enrolled in this study. Descriptive statistics for the demographic, food attentional bias, adiposity, and mediator variables are presented in Table 1. Descriptive statistics are reported for the entire sample (*n* = 120; noting missing data for reaction time, eye tracking, and energy intake variables) and the high-quality eye tracking data sample (*n* = 61). In the entire sample, the mean age was 22 years, there was an even split of men and women, and 34% of participants were non-white. The mean subjective hunger score was 3 out of 10, suggesting a relatively low level of hunger. Mean scores for direction bias and duration bias fall near .50, suggesting an even split of individuals who exhibited evidence of food attentional bias versus those who did not. The mean score for reaction time bias fell below 0 ms, suggesting slightly faster reaction times toward non-food images at the group level. For the adiposity variables, the mean BMI score for the entire sample was 25.50 kg/m^2, which falls in the lower end of the overweight BMI category. The mean body fat percent was 23.94. Although there are not established standards for body fat percent, this mean body fat percent corresponds with BMI values in the normal rage for males and females ages 20-39 years (Gallagher et al., 2000). Regarding the mediator variables, energy intake fell near the national averages for caloric intake, with males eating slightly less than the national average of 2,640 calories per day and females eating slightly more than the national average of 1,785 calories per day (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). The mean score for the external eating subscale of the DEBQ was comparable with scores from other studies examining food attentional bias (Castellanos et al., 2009; Nijs et al., 2010).

The only meaningful difference between the entire sample (n = 120) and the high-quality eye tracking data sample (n = 61) is that there were fewer non-white participants in the high-quality data sample. Eye tracking data were lost for some non-white individuals due to difficulty with head tracking and calibration. The eye tracking device was less reliable when tracking the head movements of individuals with darker complexions. In addition, the eye tracker had difficulty with measurement of the pupil reflection for some participants with dark iris colors. For information on strategies used to mitigate eye tracking data loss, particularly from non-white participants, see the Data Cleaning and Reduction section.

Table 2 shows correlations among the three measures of food attentional bias. Direction bias and duration bias had a moderate positive correlation (r = .562, p < .001), suggesting that the image participants look at first is also the image they look at the longest. In contract, reaction time bias was not correlated with direction bias (r = .024, p = .86) or duration bias (r = .055, p = .68), suggesting that there was not a relationship between the image participants looked at last and either the image they looked at first or for the longest. This pattern of correlation among attentional bias variables is consistent with Nijs et al. (2010), who found a positive correlation between direction and duration bias (r = .54) but not between reaction time and direction or duration bias (r = -.09-.19).

Table 3 shows correlations between the two measures of adiposity, with BMI and body fat percent having a strong positive correlation ($r = .704$, $p < .001$), as was expected.

3.2 Association of Food Attentional Bias with Adiposity

3.2.1 Food Attentional Bias and Body Mass Index (Hypothesis 1)

As shown in Table 4, separate linear regression analyses, adjusted for demographic factors (age, sex, and race/ethnicity) and subjective hunger score, revealed that direction bias, duration bias, and reaction time bias were not associated with BMI (all *p*s ≥ .41). Among the covariates, in each of the models, age (direction bias model $\beta = 0.44$, $p = .001$; duration bias model $\beta = 0.45$, $p < .001$; reaction time bias model $\beta = .46$, $p < .001$) and subjective hunger score (direction bias model $\beta = -0.25$, $p = .04$; duration bias model $\beta = -0.26$, $p = .03$; reaction time bias model $\beta = -0.24$, $p = .04$) were associated with BMI, whereas sex (direction bias model $\beta = 0.03$, $p = .83$; duration bias model $\beta = 0.03$, $p = .78$; reaction time bias model $\beta = 0.04$, $p = .74$) and race/ethnicity (direction bias model $\beta = -0.07$, $p = .54$; duration bias model $\beta = -0.08$, $p = .52$; reaction time bias model $\beta = -0.10$, $p = .42$) were not associated with BMI.

3.2.2 Food Attentional Bias and Body Fat Percent (Hypothesis 2)

A parallel set of separate linear regression analyses, adjusted for demographics and subjective hunger score, were performed to examine the association between food attentional bias measures and body fat percent (see Table 5). Similar to the results for BMI, there was not an association between any of the food attentional bias variables and body fat percent (all *p*s ≥ .34). Across all models, the covariates of age (direction bias model β = 0.31, p = .003; duration bias model β = 0.31, p = .002; reaction time bias model β = 0.32, p = .002), sex (direction bias model β = 0.61, p < .001; duration bias model β = 0.61, p < .001; reaction time bias model β = 0.62, p < .001), and subjective hunger score (direction bias model β = -0.27, p = .006; duration bias model β = -0.28, p = .004; reaction time bias model β = -0.26, p = .007) were associated with body fat percent. Race/ethnicity was not associated with body fact percent (direction bias model β = -0.13, p = .18; duration bias model β = -0.13, p = .17; reaction time bias reduced sample model β = -0.15, p = .14).

3.3 Association of Food Attentional Bias with Eating Behavior

Linear regression analyses were conducted to examine the association between each of the food attentional bias measures and two eating behavior variables – ASA24 energy intake and DEBQ external eating. Each food attentional bias measure was entered as a separate predictor of each eating behavior in a series of 8 analyses.

3.3.1 Food Attentional Bias and Energy Intake (Hypothesis 3)

No associations between the food attentional bias measures and ASA24 energy intake were detected (all *p*s ≥ .19; see Table 6). Despite these nonsignificant results, the magnitude of some of the standardized regression coefficients suggests trends in the hypothesized direction might have gone undetected due to insufficient power. For instance, greater duration bias was associated with increased energy intake (β = .21), although this relationship was nonsignificant (*p* = .19). Regarding covariates entered into these models, none was associated with ASA24 energy intake (all *p*s ≥ .07).

3.3.2 Food Attentional Bias and External Eating (Hypothesis 4)

The food attentional bias measures were also not associated with DEBQ external eating (all *p*s ≥ .62; see Table 7). The covariates of age (direction bias model $\beta = 0.37$, $p = .004$; duration bias model $\beta = 0.37$, $p = .004$; reaction time bias model $\beta = .37$, $p = .004$) and race/ethnicity (direction bias model $\beta = 0.41$, $p = .001$; duration bias model $\beta = 0.41$, $p = .001$; reaction time bias model $\beta = 0.40$, $p = .003$) were significantly associated with external eating, whereas sex (direction bias model $\beta = 0.14$, $p = .26$; duration bias model $\beta = 0.14$, $p = .25$; reaction time bias model $\beta = 0.14$, $p = .25$) and subjective hunger score (direction bias model $\beta = 0.16$, $p = .18$; duration bias model $\beta = 0.17$, $p = .16$; reaction time bias model $\beta = 0.16$, $p = .17$) were not.

3.4 Mediators of Food Attentional Bias-Adiposity Association

PROCESS (Hayes & Preacher, 2014) was used to test 12 mediation models. First, I examined ASA24 energy intake as a mediator between each of the three food attentional bias measures and the two adiposity measures. Second, I examined DEBQ external eating as a mediator between the three food attentional bias measures and the two adiposity measures.

3.4.1 Energy Intake as a Mediator (Hypothesis 5)

Using PROCESS model number 4, the three food attentional bias variables were entered separately as the predictor (X), the two adiposity variables were entered separately as the outcome (Y), and the ASA24 energy intake variable was entered as the mediator (M) (see Figure 4). In all models, age, sex, race/ethnicity, and subjective hunger score were included as covariates. All measures of food attentional bias were not associated with ASA24 energy intake (all path *a* *p*s ≥ .19), and ASA24 energy intake was not associated with either measure of adiposity (all path *b* *p*s ≥ .37) (see Figure 4). For associations between food attentional bias and adiposity, there was a significant overall effect between direction bias and BMI (path *c* point estimate = 30.60, *p* = .014, 95% *CI*: 6.69-54.50), and the association between direction bias and body fat percent fell just sort of significant (path *c* estimate = 31.99, *p* = .086, 95% CI: -4.79-68.77). Associations between all other measures of attentional bias and adiposity were nonsignificant (all other path *c* *p*s ≥ .18). Across all models, there was not an indirect effect of ASA24 energy intake on the food attentional bias-adiposity association (all indirect effect CIs overlapped with 0). Therefore, ASA24 energy intake was not a mediator of the food attentional bias-adiposity relationship. The direct effect of direction bias on BMI remained after accounting for ASA24 energy intake (path *c'* point estimate: 29.49, p = .019, 95% CI: 5.07 to 53.91), and the direct effect of direction bias on body fat percent remained just short of significant (*c'* path point estimate: 32.27, p = .076, 95% CI: -3.55 to 68.09). There is some preliminary evidence for an association of direction bias with BMI and possibly body fat percent, as observed in the mediation analyses using the sample of 42 participants with high-quality eye tracking data who completed the ASA24.

Of note, this association was not observed in the linear regression analyses using the high-quality eye tracking data sample of 61 participants.

3.4.2 External Eating as a Mediator (Hypothesis 6)

Using PROCESS model number 4, the three food attentional bias variables entered separately as the predictor (X), the two adiposity variables entered separately as the outcome (Y), and the DEBQ external eating subscale score variable was entered as the mediator (M) (see Figure 5). In all models, age, sex, race/ethnicity, and subjective hunger score were added as covariates. All measures of food attentional bias were not associated with DEBQ external eating (all path *a* ps $\geq .62$), and DEBQ external eating was not associated with either measure of adiposity (all path *b* ps $\geq .51$). In addition, measures of food attentional bias were not associated with measures of adiposity (all path *c* ps $\geq .34$). There was not an indirect effect of DEBQ external eating on the food attentional bias-adiposity association (all indirect effect CIs overlapped with 0). Therefore, DEBQ external eating is not a mediator of the food attentional bias-adiposity relationship. After accounting for the potential mediator of DEBQ external eating, there remained no direct effect of food attentional bias on adiposity (all path *c'* $ps \geq .36$).

CHAPTER 4. DISCUSSION

4.1 Summary of Results

The objective of the present study was to examine the association between food attentional bias and adiposity and to explore two potential mediators – namely, energy intake and external eating. The first and second hypotheses that food attentional bias is positively associated with BMI and body fat percent, respectively, were not supported. Regression analyses revealed that none of the three food attentional bias measures – direction bias, duration bias, and reaction time bias – was related to the adiposity measures. Of note, in mediation analyses involving a reduced sample of 42 participants, there was a significant positive association between direction bias and BMI and a nonsignificant positive trend between direction bias and body fat percent. The third hypothesis that food attentional bias is associated with energy intake and the fourth hypothesis that food attentional bias is associated with external eating were not supported. However, there the magnitude of the standardized regression coefficient for the association of duration and direction bias with energy intake suggested the presence of positive association that may have gone undetected due to insufficient power.

Although food attentional bias was not related to adiposity, energy intake, or external eating, I examined energy intake and external eating as potential mediators. The fifth hypothesis that energy intake mediates the food attentional bias-adiposity association was not supported. Likewise, the sixth hypothesis that external eating mediates the food attentional bias-adiposity association was not supported.

4.2 Fit with Existing Literature

The lack of associations between food attentional bias and adiposity in the high-quality eye tracking data sample contradicts findings from previous studies, which have shown that overweight/obese women exhibit greater attentional bias toward food cues compared to normal weight women (Castellanos et al., 2009; Nijs et al., 2010; Werthmann et al., 2011). However, there was a significant association between direction bias and BMI in the sample of 42 participants. It is possible that this association was detected in the reduced sample due methodological factors of improving the eye tracking assessment over time (i.e., training research assistant in eye tracking calibration, reducing the glare and ambient lighting in the participant chamber to improve pupil and corneal reflection measurement, and using a chin rest and adding a white screen behind the participant to improve head tracking) or changes in the order of the study tasks (i.e., moving the eye tracking task to before the administration of questionnaires). It is also possible that this association was detected due to type 1 error, given that I only found one association across 28 analyses.

Aside from this one significant relationship, results of the present study generally did not replicate past findings. Methodological factors may account for this discrepancy. For instance, this study analyzed food attentional bias and adiposity as continuous variables, whereas other studies have dichotomized these variables (Castellanos et al., 2009; Nijs et al., 2010; Werthmann et al., 2011). Food attentional bias and adiposity variables were modeled as continuous variables because attentional bias, BMI, and body fat percent are measured naturally as continuous variables and dichotomizing continuous variables can lead to lower statistical power (Babyak, 2004). In addition, the sample included both men and women, whereas past studies utilized samples of only women (Castellanos et al., 2009; Nijs et al., 2010; Werthmann et al., 2011). It is possible that this relationship may be predominantly found among women.

The two studies that have examined the association between food attentional bias and energy intake during laboratory taste test tasks observed no relationships (Nijs et al., 2010; Werthmann et al., 2011). The present results are consistent with these findings. However, the analyses revealed a moderate effect size between duration bias and energy intake, which suggest that there may be meaningful relationships that could be detected with larger samples and greater statistical power.

Previous studies examining the association between food attentional bias and external eating show mixed evidence. One study found that high external eating was associated with low food attentional bias (i.e., diverting attention away from food cues) (Johansson et al., 2004), whereas another study found the opposite pattern of results (Hepworth et al., 2010). Interestingly, the present study contrasted the previous studies in this area, as no association was observed between measures of food attentional bias and external eating.

4.3 Possible Explanations for Null Findings

Results of the present study did not support any of the hypotheses. According to Kazdin (2003), there are two possible explanations for null findings. The first explanation is that there truly is no association between food attentional bias and adiposity. The second explanation is that there is an association; however, I was unable to detect it due to methodological issues.

Assuming the first explanation is true, in the natural state of these variables, food attentional bias is not associated with adiposity. However, previous studies have found that food attentional bias is stronger for overweight/obese women compared to normal weight women (Castellanos et al., 2009; Nijs et al., 2010; Werthmann et al., 2011), suggesting that the present study may not have adequately captured the association.

Assuming the second explanation is true, methodological issues limited my ability to detect an association that occurs in nature. Potential methodological issues include inadequate measurement of key variables, limited variability in key variables, insufficient statistical power, uncontrolled error variability, or the influence of outside or confounding factors. Each of these issues is reviewed below.

With respect to the first issue (inadequate measurement of key variables), the present study included three state-of-the-art measures of food attentional bias – direction bias, duration bias, and reaction time bias. However, there were issues with eye tracking data quality and loss.

Primary analyses used a subsample of participants with high-quality eye track data, indicated by looking at either food or non-food images in 20 or more of the 40 experimental events. It is possible that 20 events are not enough events to provide a representative sample of the food attentional bias of a given participant**.** Conversely, it is also possible that I lost valuable information for participants with fewer than 20 events. Of note, a substantial amount of data loss occurred in participants in the non-white group.

Eye tracking data was lost for 61% of non-white participants, compared to 37% for white participants. If the food attentional bias-adiposity association is stronger among racial/ethnic minorities, this data loss could work against my ability to detect a relationship.

The second possible issue is the limited variability in the dependent variable, adiposity. More than half of the present sample fell in the normal or underweight BMI category (n = 34, 55.7%), and a smaller percentage fell in overweight (n = 20, 32.8%) or obese (n = 7, 11.5%) categories. Because obesity and overweight occur in two thirds of the U.S. population (Centers for Disease Control and Prevention, 2012), obese and overweight individuals may be under-represented in this sample. This limited variability in BMI may have impaired my ability to detect the hypothesized associations.

A third possible issue is likely insufficient statistical power. In this study, there were associations of moderate size that did not achieve statistical significance – namely, the direction bias-body fat percent relationship in mediation analyses (path c estimate = 31.99, p = .086, 95% CI: -4.79-68.77; c' path point estimate: 32.27, p = .076, 95% CI: -3.55 to 68.09) and the duration bias-energy intake relationship (β = .21, p = .19). These associations might have been detected in a larger sample with greater statistical power.

A fourth possible issue is unsystematic implementation of the protocol, which might have increased uncontrolled error variance (Kazdin, 2003). Three factors could have contributed to unsystematic implementation of the protocol: (1) training of new research assistants, (2) changing the order of study tasks, and (3) improvements in eye tracking measurement across the study. First, training of new research assistants was comprehensive and included the use of a protocol with detailed descriptions of study tasks and eye tracking procedures, several sessions of training on the use of the eye tracking device, and attending several data collection sessions where the trainee first shadowed and then was shadowed by a more senior research assistant. Despite comprehensive training, with the start of each new research assistant, there was a notable learning curve to obtaining high-quality eye tracking data. Second, changing the order of study tasks could have increased error variance. Initially, the questionnaires were administered prior to the dot-probe reaction time task. The questionnaire items related to eating and health behaviors alerted some participants to the objective of the dot-probe task, which led them to avoid food cues. To address this issue, I changed the order of study tasks by having participants complete the dot-probe task before completing the questionnaires. In addition, I excluded participants who looked only at the white space around the food and non-food images. Third, because the eye tracking equipment was new to all of the research assistants at the start of data collection, the collective ability of research assistants to obtain high-quality eye tracking data likely improved over time. This could explain why an association between direction bias and BMI was found in the reduced sample of 42 participants who completed the ASA24 (which was later added to the study) but not in the primary sample of 61 participants.

A fifth and final possible issue that may explain my null findings is the influence of outside or confounding factors – i.e., the younger age of the sample and the presence of approach-avoidance behavior during the food attentional bias assessments. This sample was comprised predominantly of young adults, who not have had enough time to experience the long-term cumulative effects of food attentional biases on adiposity. However, they may display increased energy intake, which represents an earlier stage in the attentional bias-adiposity process. This notion is consistent with my finding that the duration bias-energy intake association, although nonsignificant, had a moderate effect size.

Another possible confounding factor is approach-avoidance behavior in response to food cues, characterized by initially orienting to the food cue but then looking away from the food cue. Evidence suggests that some overweight/obese participants avoid food images or words (Nijs & Franken, 2012). If approach-avoidance occurred in this study, I would still expect to observe associations with the direction bias measure, which captures the initial gaze direction at picture onset. Consequently, approach-avoidance behavior is likely not the sole cause of my null findings, as direction bias-adiposity relationships were generally not observed.

4.4 Limitations and Future Directions

Given the ambiguity of my null results and the paucity of studies in this area, additional research should be conducted to further evaluate the potential relationships between food attentional biases and adiposity. Four key areas for future investigation include: (1) examining the food attentional bias-adiposity association in a larger, more diverse sample; (2) evaluating the prospective associations between food attentional biases and adiposity; (3) exploring potential moderators of these associations, and (4) performing a detailed assessment of the various measures of food attentional bias.

There are several limitations of the present study. First, the study used a moderately-sized sample of primarily young, Caucasian participants. Because older adults and racial/ethnic minorities are at increased risk of obesity (Wang & Beydoun, 2007), it is important to have at least adequate representation of these groups in studies examining risk factors for obesity. Relatedly, this study utilized a sample of undergraduate students, which is not an ideal sample to use to answer questions about the food-attentional biases and adiposity association. The sample included participants who were predominantly normal weight and who did not demonstrate high levels of food attentional bias. Therefore, there was not a clear match between the hypotheses proposed and the sample in which the hypotheses were tested. Given these sample limitations, future studies should examine the association between continuous variables of food attentional bias and adiposity in larger, more diverse samples and samples that are more representative of the true nature of food attentional biases and adiposity.

Second, all existing studies of food attentional bias and adiposity, including the present study, have examined cross-sectional associations. Therefore, it is not known whether food attentional bias predicts future changes in adiposity and/or whether adiposity predicts the development of food attentional bias. Based on the incentive sensitization theory of reward (Robinson & Berridge, 1993), it is conceivable that individuals who have developed incentive salience toward food cues have already engaged in eating of foods high in reward value. Thus, intake of these rewarding foods, which can cause weight gain, could occur before developing attentional biases. Accordingly, future studies should examine the bidirectional, prospective associations among food attentional bias, eating behavior, and adiposity.

Third, future studies should examine the role of potential moderators of food attentional bias-adiposity associations. Candidate moderators include sex, impulsivity, and physical activity. Although rates of obesity are equal among men and women (Flegal et al., 2012), it is possible that the mechanisms for the development of obesity differ by gender. Another potential moderator, impulsivity, has a positive association with food attentional bias as well as external eating (Hou et al., 2011). It has been theorized that individuals who are impulsive may have a diminished ability to regulate their responses to food cues (Hou et al., 2011). Therefore, those high in impulsivity with attentional bias to food cues may be more likely notice food and eat in response to food cues, thereby increasing their adiposity. A third potential moderator is physical activity. Evidence indicates that physical activity helps to maintain body weight and prevent weight gain (Fogelholm & Kukkonen-Harjula, 2000; Swift, Johannsen, Lavie, Earnest, & Church, 2014). Individuals with food attentional biases who also exercise regularly may be less likely to develop excess adiposity than those with the same biases who are more sedentary.

Fourth, more research should be conducted on the use of attentional bias measures. A limitation of the present study is that a substantial amount of data was lost using the eye tracking paradigm. More research should be conducted to elucidate sources of data loss and strategies to mitigate data loss, specifically for non-white participants. In addition, studies should examine differences in the measures of attentional bias to decipher whether or not they tap the same construct of attentional bias or whether they are measuring different aspects of attention to food cues.

4.5 Conclusion

Food attentional bias and adiposity were not associated in this sample of generally healthy young adults. In addition, food attentional bias was not associated with energy intake or external eating behavior. Several factors may have contributed to these null results, including the methodological issues of inadequate measurement of key variables, limited variability in key variables, insufficient statistical power, uncontrolled error variability, or the influence of outside or confounding factors. Although the present hypotheses were not supported, it is important to note that food attentional bias may still be a novel risk factor for obesity. Future studies should examine prospective associations between food attentional biases and adiposity in larger, more diverse samples, while also exploring potential mediators and moderators of these associations.

LIST OF REFERENCES

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Aronne, L. J., Nelinson, D. S., & Lillo, J. L. (2009). Obesity as a disease state: A new paradigm for diagnosis and treatment. *Clin Cornerstone, 9*(4), 9-25; discussion 26-29.

Babyak, M. A. (2004). What you see may not be what you get: A brief, nontechnical introduction to overfitting in regression-type models. *Psychosom Med, 66*(3), 411-421.

Barness, L. A., Opitz, J. M., & Gilbert-Barness, E. (2007). Obesity: Genetic, molecular, and environmental aspects. *Am J Med Genet A, 143A*(24), 3016-3034. doi: 10.1002/ajmg.a.32035

Berridge, K. C., Ho, C. Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Res, 1350*, 43-64. doi: 10.1016/j.brainres.2010.04.003

Blanton, C. A., Moshfegh, A. J., Baer, D. J., & Kretsch, M. J. (2006). The USDA Automated Multiple-Pass Method accurately estimates group total energy and nutrient intake. *J Nutr, 136*(10), 2594-2599.

Bradley, B. P., Mogg, K., Wright, T., & Field, M. (2003). Attentional bias in drug dependence: Vigilance for cigarette-related cues in smokers. *Psychol Addict Behav, 17*(1), 66-72.

Brignell, C., Griffiths, T., Bradley, B. P., & Mogg, K. (2009). Attentional and approach biases for pictorial food cues. Influence of external eating. *Appetite, 52*(2), 299-306. doi: 10.1016/j.appet.2008.10.007

Cannon, C. M., & Bseikri, M. R. (2004). Is dopamine required for natural reward? *Physiol Behav, 81*(5), 741-748. doi: 10.1016/j.physbeh.2004.04.020

Caro, J. F., Sinha, M. K., Kolaczynski, J. W., Zhang, P. L., & Considine, R. V. (1996). Leptin: The tale of an obesity gene. *Diabetes, 45*(11), 1455-1462.

Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *Int J Obes (Lond), 33*(9), 1063-1073. doi: 10.1038/ijo.2009.138

Centers for Disease Control and Prevention. (2012). Prevalence of obesity in the United States: 2009-2010. Retrieved January 15, 2015 from http://www.cdc.gov/nchs/data/databriefs/db82.pdf.

Copersino, M. L., Serper, M. R., Vadhan, N., Goldberg, B. R., Richarme, D., Chou, J. C., . . . Cancro, R. (2004). Cocaine craving and attentional bias in cocaine-dependent schizophrenic patients. *Psychiatry Res, 128*(3), 209-218. doi: 10.1016/j.psychres.2004.07.006

Craig, C. L., Marshall, A. L., Sjostrom, M., Bauman, A. E., Booth, M. L., Ainsworth, B. E., . . . Oja, P. (2003). International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc, 35*(8), 1381-1395. doi: 10.1249/01.MSS.0000078924.61453.FB de Ruiter, C., & Brosschot, J. F. (1994). The emotional Stroop interference effect in anxiety: Attentional bias or cognitive avoidance? *Behav Res Ther, 32*(3), 315-319.

Dobson, K. S., & Dozois, D. J. (2004). Attentional biases in eating disorders: A meta-analytic review of Stroop performance. *Clin Psychol Rev, 23*(8), 1001-1022.

Fadardi, J. S., & Bazzaz, M. M. (2011). A Combi-Stroop test for measuring food-related attentional bias. *Exp Clin Psychopharmacol, 19*(5), 371-377. doi: 10.1037/a0024045

Faulconbridge, L. F., & Wadden, T. A. (2010). Managing the obesity epidemic. *Handbook of Health Psychology and Behavioral Medicine*. New York, NY: Guilford Publication.

Faunce, G. J. (2002). Eating disorders and attentional bias: A review. *Eat Disord, 10*(2), 125-139. doi: 10.1080/10640260290081696

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug Alcohol Depend, 97*(1-2), 1-20. doi: 10.1016/j.drugalcdep.2008.03.030

Field, M., Mogg, K., Zetteler, J., & Bradley, B. P. (2004). Attentional biases for alcohol cues in heavy and light social drinkers: The roles of initial orienting and maintained attention. *Psychopharmacology (Berl), 176*(1), 88-93. doi: 10.1007/s00213-004-1855-1

Finkelstein, E. A., Fiebelkorn, I. C., & Wang, G. (2003). National medical spending attributable to overweight and obesity: How much, and who's paying? *Health Aff (Millwood), 22*, 219-226.

Flegal, K. M., Carroll, M. D., Kit, B. K., & Ogden, C. L. (2012). Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *JAMA, 307*(5), 491-497. doi: 10.1001/jama.2012.39

Flegal, K. M., Graubard, B. I., Williamson, D. F., & Gail, M. H. (2007). Cause-specific excess deaths associated with underweight, overweight, and obesity. *JAMA, 298*(17), 2028-2037. doi: 10.1001/jama.298.17.2028

Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord, 24*(1), 38-48.

Fogelholm, M., & Kukkonen-Harjula, K. (2000). Does physical activity prevent weight gain: A systematic review. *Obes Rev, 1*(2), 95-111.

Franken, I. H., Kroon, L. Y., & Hendriks, V. M. (2000). Influence of individual differences in craving and obsessive cocaine thoughts on attentional processes in cocaine abuse patients. *Addict Behav, 25*(1), 99-102.

Franken, I. H., & Muris, P. (2005). Individual differences in reward sensitivity are related to food craving and relative body weight in healthy women. *Appetite, 45*(2), 198-201. doi: 10.1016/j.appet.2005.04.004

Gallagher, D., Heymsfield, S. B., Heo, M., Jebb, S. A., Murgatroyd, P. R., & Sakamoto, Y. (2000). Healthy percentage body fat ranges: An approach for developing guidelines based on body mass index. *Am J Clin Nutr, 72*(3), 694-701.

Hayes, A. F., & Preacher, K. J. (2014). Statistical mediation analysis with a multicategorical independent variable. *Br J Math Stat Psychol, 67*(3), 451-470. doi: 10.1111/bmsp.12028

Hayes, A. F., & Scharkow, M. (2013). The relative trustworthiness of inferential tests of the indirect effect in statistical mediation analysis: Does method really matter? *Psychol Sci, 24*(10), 1918-1927. doi: 10.1177/0956797613480187

Hepworth, R., Mogg, K., Brignell, C., & Bradley, B. P. (2010). Negative mood increases selective attention to food cues and subjective appetite. *Appetite, 54*(1), 134-142. doi: 10.1016/j.appet.2009.09.019

Hermans, R. C., Larsen, J. K., Lochbuehler, K., Nederkoorn, C., Herman, C. P., & Engels, R. C. (2012). The power of social influence over food intake: Examining the effects of attentional bias and impulsivity. *Br J Nutr*, 1-9. doi: 10.1017/S0007114512001390

Hester, R., Dixon, V., & Garavan, H. (2006). A consistent attentional bias for drug-related material in active cocaine users across word and picture versions of the emotional Stroop task. *Drug Alcohol Depend, 81*(3), 251-257. doi: 10.1016/j.drugalcdep.2005.07.002

Hill, J. O., & Peters, J. C. (1998). Environmental contributions to the obesity epidemic. *Science, 280*(5368), 1371-1374.

Hofbauer, K. G. (2002). Molecular pathways to obesity. *Int J Obes Relat Metab Disord, 26 Suppl 2*, S18-27. doi: 10.1038/sj.ijo.0802124

Hou, R., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite, 56*(2), 424-427. doi: 10.1016/j.appet.2011.01.019

Johansson, L., Ghaderi, A., & Andersson, G. (2004). The role of sensitivity to external food cues in attentional allocation to food words on dot probe and Stroop tasks. *Eat Behav, 5*(3), 261-271. doi: 10.1016/j.eatbeh.2004.01.005

Johnsen, B. H., Thayer, J. F., Laberg, J. C., & Asbjornsen, A. E. (1997). Attentional bias in active smokers, abstinent smokers, and nonsmokers. *Addict Behav, 22*(6), 813-817.

Kazdin, A. E. (2003). *Research Design in Clinical Psychology, 4th ed.* Boston, MA: Allyn & Bacon.

Kelley, A. E., & Berridge, K. C. (2002). The neuroscience of natural rewards: Relevance to addictive drugs. *J Neurosci, 22*(9), 3306-3311. doi: 20026361

Kemps, E., & Tiggemann, M. (2009). Attentional bias for craving-related (chocolate) food cues. *Exp Clin Psychopharmacol, 17*(6), 425-433. doi: 10.1037/a0017796

Kiyatkin, E. A., & Stein, E. A. (1996). Conditioned changes in nucleus accumbens dopamine signal established by intravenous cocaine in rats. *Neurosci Lett, 211*(2), 73-76.

Kline, R. B. (2004). *Principles and Practice of Structural Equation Modeling, Second Edition.* New York, NY: Guilford Press.

Kyle, U. G., Morabia, A., Schutz, Y., & Pichard, C. (2004). Sedentarism affects body fat mass index and fat-free mass index in adults aged 18 to 98 years. *Nutrition, 20*(3), 255-260. doi: 10.1016/j.nut.2003.11.019

Labarthe, D. R. (1998). *Epidemiology and Prevention of Cardiovascular Diseases: A Global Challenge.* Gaithersburg, MD: Aspen Publishers.

Lenard, N. R., & Berthoud, H. R. (2008). Central and peripheral regulation of food intake and physical activity: Pathways and genes. *Obesity (Silver Spring), 16 Suppl 3*, S11-22. doi: 10.1038/oby.2008.511

Levine, J. A., Lanningham-Foster, L. M., McCrady, S. K., Krizan, A. C., Olson, L. R., Kane, P. H., . . . Clark, M. M. (2005). Interindividual variation in posture allocation: Possible role in human obesity. *Science, 307*(5709), 584-586. doi: 10.1126/science.1106561

Lubman, D. I., Peters, L. A., Mogg, K., Bradley, B. P., & Deakin, J. F. (2000). Attentional bias for drug cues in opiate dependence. *Psychol Med, 30*(1), 169-175.

Luecken, L. J., & Gallo, L. C. (2007). *Handbook of Physiological Research Methods in Health Psychology*. Thousand Oaks, CA: Sage Publications.

Lusher, J., Chandler, C., & Ball, D. (2004). Alcohol dependence and the alcohol Stroop paradigm: Evidence and issues. *Drug Alcohol Depend, 75*(3), 225-231. doi: 10.1016/j.drugalcdep.2004.03.004

Macht, M., Gerer, J., & Ellgring, H. (2003). Emotions in overweight and normal-weight women immediately after eating foods differing in energy. *Physiol Behav, 80*(2-3), 367-374.

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiol Rev, 29*, 29-48. doi: 10.1093/epirev/mxm001

Mogg, K., Bradley, B. P., Hyare, H., & Lee, S. (1998). Selective attention to food-related stimuli in hunger: Are attentional biases specific to emotional and psychopathological states, or are they also found in normal drive states? *Behav Res Ther, 36*(2), 227-237.

Moshfegh, A. J., Rhodes, D. G., Baer, D. J., Murayi, T., Clemens, J. C., Rumpler, W. V., . . . Cleveland, L. E. (2008). The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. *Am J Clin Nutr, 88*(2), 324-332.

Neel, J. V. (1962). Diabetes mellitus: A "thrifty" genotype rendered detrimental by "progress"? *Am J Hum Genet, 14*, 353-362.

Nijs, I. M., & Franken, I. H. (2012). Attentional Processing of Food Cues in Overweight and Obese Individuals. *Curr Obes Rep, 1*(2), 106-113. doi: 10.1007/s13679-012-0011-1

Nijs, I. M., Muris, P., Euser, A. S., & Franken, I. H. (2010). Differences in attention to food and food intake between overweight/obese and normal-weight females under conditions of hunger and satiety. *Appetite, 54*(2), 243-254. doi: 10.1016/j.appet.2009.11.004

Parsons, T. J., Manor, O., & Power, C. (2008). Television viewing and obesity: A prospective study in the 1958 British birth cohort. *Eur J Clin Nutr, 62*(12), 1355-1363. doi: 10.1038/sj.ejcn.1602884

Phelan, S., Hassenstab, J., McCaffery, J. M., Sweet, L., Raynor, H. A., Cohen, R. A., & Wing, R. R. (2011). Cognitive interference from food cues in weight loss maintainers, normal weight, and obese individuals. *Obesity (Silver Spring), 19*(1), 69-73. doi: 10.1038/oby.2010.138

Placanica, J. L., Faunce, G. J., & Soames Job, R. F. (2002). The effect of fasting on attentional biases for food and body shape/weight words in high and low Eating Disorder Inventory scorers. *Int J Eat Disord, 32*(1), 79-90. doi: 10.1002/eat.10066

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behav Res Methods Instrum Comput, 36*(4), 717-731.

Racette, S. B., Deusinger, S. S., & Deusinger, R. H. (2003). Obesity: Overview of prevalence, etiology, and treatment. *Phys Ther, 83*(3), 276-288.

Ravussin, E., & Bouchard, C. (2000). Human genomics and obesity: Finding appropriate drug targets. *Eur J Pharmacol, 410*(2-3), 131-145.

Rising, R., Harper, I. T., Fontvielle, A. M., Ferraro, R. T., Spraul, M., & Ravussin, E. (1994). Determinants of total daily energy expenditure: Variability in physical activity. *Am J Clin Nutr, 59*(4), 800-804.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Res Brain Res Rev, 18*(3), 247-291.

Robinson, T. E., & Berridge, K. C. (2001). Incentive-sensitization and addiction. *Addiction, 96*(1), 103-114. doi: 10.1080/09652140020016996

Roche, A. F., Sievogel, R. M., Chumlea, W. C., & Webb, P. (1981). Grading body fatness from limited anthropometric data. *Am J Clin Nutr, 34*(12), 2831-2838.

Rosenbaum, M., Leibel, R. L., & Hirsch, J. (1997). Obesity. *N Engl J Med, 337*(6), 396-407. doi: 10.1056/NEJM199708073370606

Schiff, S. R. (1982). Conditioned dopaminergic activity. *Biol Psychiatry, 17*(2), 135-154.

Shepherd, P. R., & Kahn, B. B. (1999). Glucose transporters and insulin action--implications for insulin resistance and diabetes mellitus. *N Engl J Med, 341*(4), 248-257. doi: 10.1056/NEJM199907223410406

Shoelson, S. E., Herrero, L., & Naaz, A. (2007). Obesity, inflammation, and insulin resistance. *Gastroenterology, 132*(6), 2169-2180. doi: 10.1053/j.gastro.2007.03.059

Sobal, J., & Stunkard, A. J. (1989). Socioeconomic status and obesity: A review of the literature. *Psychol Bull, 105*(2), 260-275.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643-662.

Swift, D. L., Johannsen, N. M., Lavie, C. J., Earnest, C. P., & Church, T. S. (2014). The role of exercise and physical activity in weight loss and maintenance. *Prog Cardiovasc Dis, 56*(4), 441-447. doi: 10.1016/j.pcad.2013.09.012

Tapper, K., Pothos, E. M., Fadardi, J. S., & Ziori, E. (2008). Restraint, disinhibition and food-related processing bias. *Appetite, 51*(2), 335-338. doi: 10.1016/j.appet.2008.03.006

U.S. Department of Agriculture, & U.S. Department of Health and Human Services. (2005). Dietary guidelines for Americans. Retrieved from May 30, 2015 http://www.health.gov/dietaryguidelines/dga2005/document/.

U.S. Department of Agriculture, & U.S. Department of Health and Human Services. (2010). Dietary Guidelines for Americans, 2010. Retrieved May 30, 2015 from http://www.health.gov/dietaryguidelines/2010.asp.

van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders, 5*(5), 295-315.

Wang, Y., & Beydoun, M. A. (2007). The obesity epidemic in the United States--gender, age, socioeconomic, racial/ethnic, and geographic characteristics: A systematic review and meta-regression analysis. *Epidemiol Rev, 29*, 6-28. doi: 10.1093/epirev/mxm007

Wardle, J. (1987). Eating style: A validation study of the Dutch Eating Behaviour Questionnaire in normal subjects and women with eating disorders. *J Psychosom Res, 31*(2), 161-169.

Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can(not) take my eyes off it: Attention bias for food in overweight participants. *Health Psychol, 30*(5), 561-569. doi: 10.1037/a0024291

Westerterp, K. R., & Speakman, J. R. (2008). Physical activity energy expenditure has not declined since the 1980s and matches energy expenditures of wild mammals. *Int J Obes (Lond), 32*(8), 1256-1263. doi: 10.1038/ijo.2008.74

Willett, W. C., Manson, J. E., Stampfer, M. J., Colditz, G. A., Rosner, B., Speizer, F. E., & Hennekens, C. H. (1995). Weight, weight change, and coronary heart disease in women: Risk within the 'normal' weight range. *JAMA, 273*(6), 461-465.

Williams, J. M., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychol Bull, 120*(1), 3-24.

Wise, R. A. (1996). Neurobiology of addiction. *Curr Opin Neurobiol, 6*(2), 243-251.

Wise, R. A. (1998). Drug-activation of brain reward pathways. *Drug Alcohol Depend, 51*(1-2), 13-22.

World Health Organization. (1995). Physical status: The use and interpretation of anthropometry (Report of a WHO Expert Committee). Retrieved January 15, 2015 from http://whqlibdoc.who.int/trs/WHO_TRS_854.pdf.

World Health Organization. (2000). Obesity: Preventing and managing the global epidemic (Report of a WHO Consultation). Retrieved January 15, 2015 from http://whqlibdoc.who.int/trs/WHO_TRS_894.pdf.

World Health Organization. (2015). Factsheet: Obesity and overweight. Retrieved January 15, 2015 from http://www.who.int/mediacentre/factsheets/fs311/en/.

Young, L. R., & Nestle, M. (2002). The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health, 92*(2), 246-249.

TABLES

Table 1. Characteristics of Participants

	Entire Sample **$N = 120$**	**High-Quality Eye Tracking Data Sample** **$N = 61$**
Age, years	22.04 (5.81)	20.63 (2.81)
Female, %	52.5	59.0
Non-white, %	34.2	23.0
Subjective Hunger Score	3.08 (2.67)	3.26 (2.87)
Direction Bias	.48 (.16)	.47 (.06)
Duration Bias	.50 (.17)	.50 (.06)
Reaction Time Bias, ms[†]	-20.80 (38.67)	-22.71 (37.69)
Body Mass Index, kg/m^2	25.50 (5.68)	24.55 (4.99)
Body Fat Percent	23.94 (9.86)	23.59 (9.49)
Energy Intake, kcal[†]	2,064.18 (939.66)	2,192.93 (963.88)
DEBQ External Eating	3.16 (.66)	3.19 (.66)

Note. Continuous variables are presented as mean (standard deviation), and categorical variables are presented as percentage. DEBQ represents the Dutch Eating Behavior Questionnaire.

[†]Reported on a reduced sample. Reaction time bias is reported on $n = 117$ due to missing data for two participants and an extreme value for a third participant. Energy intake is reported on $n = 73$ in the entire sample and $n = 42$ in the high-quality eye tracking data sample due to the measure being added later in data collection.

Table 2. Correlations Among Food Attentional Bias Measures

	Direction Bias	Duration Bias	Reaction Time Bias
Direction Bias	1		
Duration Bias	.562**	1	
Reaction Time Bias	.024	.055	1

N = 61
** $p<.001$

Table 3. Correlations Between Adiposity Measures

	Body Mass Index	Body Fat Percent
Body Mass Index	1	
Body Fat Percent	.704**	1

N = 61
** $p<.001$

Table 4. Separate Linear Regression Models Examining the Association Between Three Food Attentional Bias Measures and Body Mass Index

	n	***β***	***p*-value**
Direction Bias	61	0.03	0.774
Duration Bias	61	0.10	0.413
Reaction Time Bias (High-Quality Eye Tracking Data Sample)	61	-0.09	0.490
Reaction Time Bias (Entire Sample)	117	0.04	0.686

Note: All models are adjusted for age, sex, race/ethnicity, and subjective hunger.
β represents the standardized regression coefficient.

Table 5. Separate Linear Regression Models Examining the Association Between Three Food Attentional Bias Measures and Body Fat Percent

	n	***β***	***p*-value**
Direction Bias	61	0.01	0.918
Duration Bias	61	0.09	0.342
Reaction Time Bias (High-Quality Eye Tracking Data Sample)	61	-0.07	0.491
Reaction Time Bias (Entire Sample)	117	0.04	0.586

Note: All models are age, sex, race/ethnicity, and subjective hunger.
β represents the standardized regression coefficient.

Table 6. Separate Linear Regression Models Examining the Association Between Three Food Attentional Bias Measures and Energy Intake

	n	***β***	***p*-value**
Direction Bias	42	0.16	0.366
Duration Bias	42	0.21	0.190
Reaction Time Bias (High-Quality Eye Tracking Data Sample)	42	0.11	0.535
Reaction Time Bias (Entire Sample)	73	-0.08	0.527

Note: All models are adjusted for age, sex, race/ethnicity, and subjective hunger.
β represents the standardized regression coefficient.

Table 7. Separate Linear Regression Models with Examining Association Between Three Food Attentional Bias Measures and External Eating

	n	***β***	***p*-value**
Direction Bias	61	-0.01	0.943
Duration Bias	61	-0.06	0.616
Reaction Time Bias (High-Quality Eye Tracking Data Sample)	61	-0.04	0.747
Reaction Time Bias (Entire Sample)	117	-0.04	0.668

Note: All models are adjusted for age, sex, race/ethnicity, and subjective hunger.
β represents the standardized regression coefficient.

FIGURES

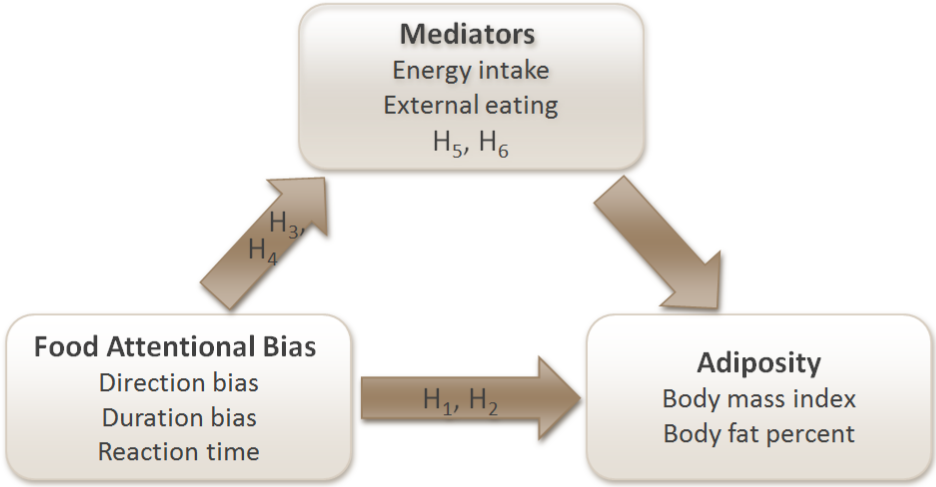

Figure 1. Conceptual Model

Conceptual model for the present study depicting energy intake and external eating as potential mediators of the cross-sectional association between food attentional bias and adiposity.

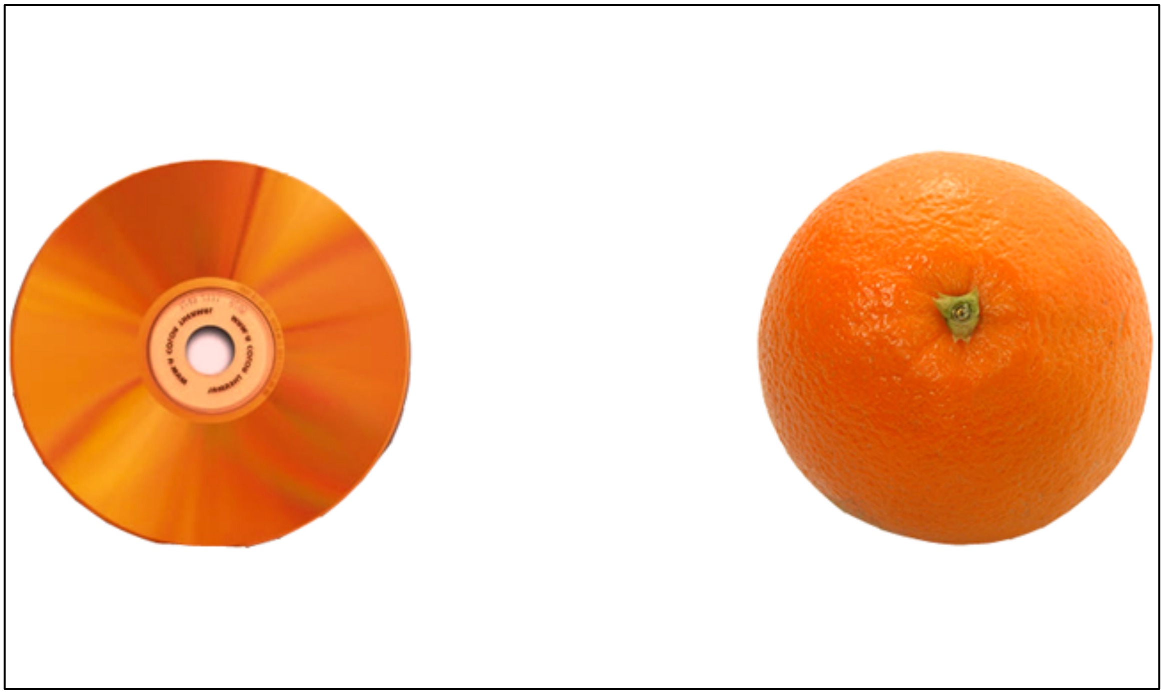

Figure 2. Sample Image Pair

Screen shot of an image pair used during the dot-probe task. Participants are presented with a series of image pairs that are matched for color, shape, and size. In the presented image, an orange and a CD are paired together.

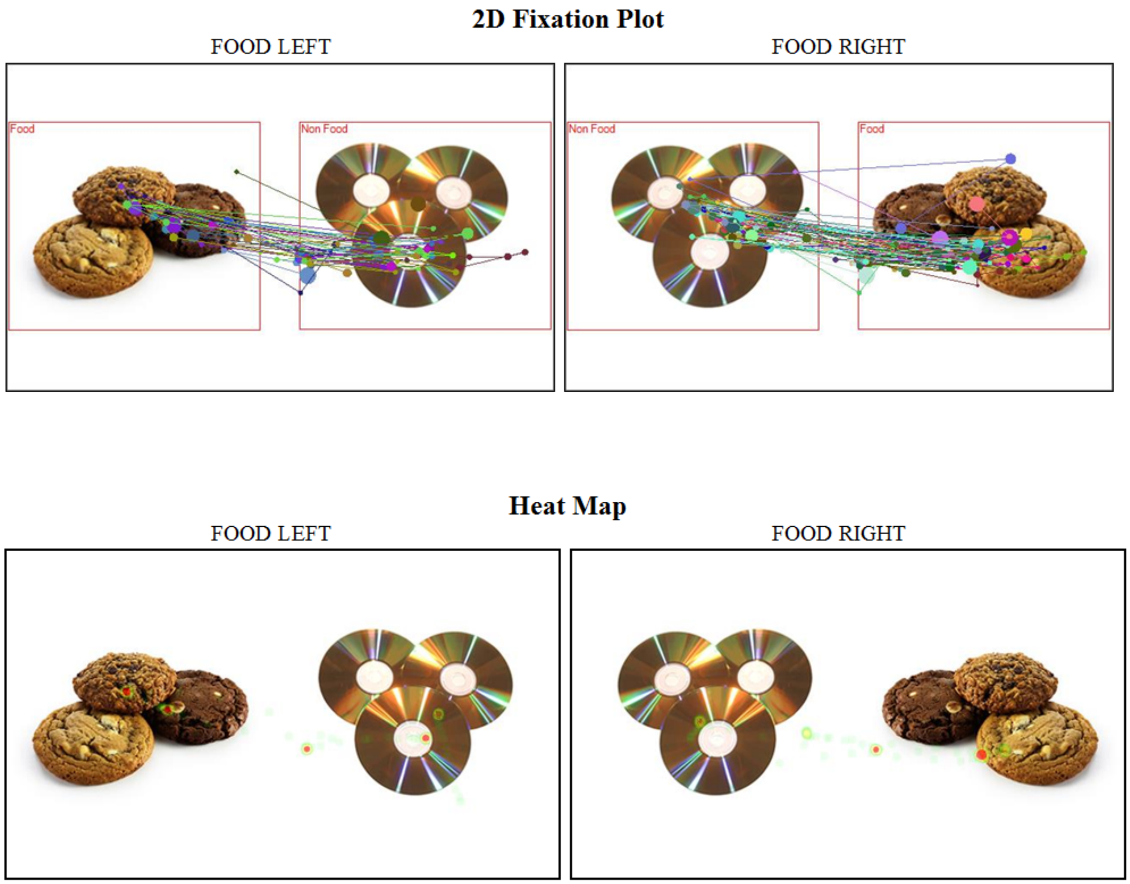

Figure 3. 2D Fixation Plot and Heat Map Summaries of Eye Tracking Data for One Participant

Heat map and 2D fixation plot summaries for one participant are shown. Summaries of experimental events where the food image was presented on the right (FOOD RIGHT) or on the left (FOOD LEFT) are depicted separately. The summary data is displayed on an exemplar experimental image pair that includes three cookies (food) and three CDs (non-food), which have been matched for color, shape, and size. This participant had a direction bias value of .55 and a duration bias value of .54, suggesting a slight attentional bias toward food images. These values are consistent with the heat map and fixation plot summaries, which show that the participant looked at both food and non-food images at a relatively high rate, but he or she appears to look at food images more often and for longer duration.

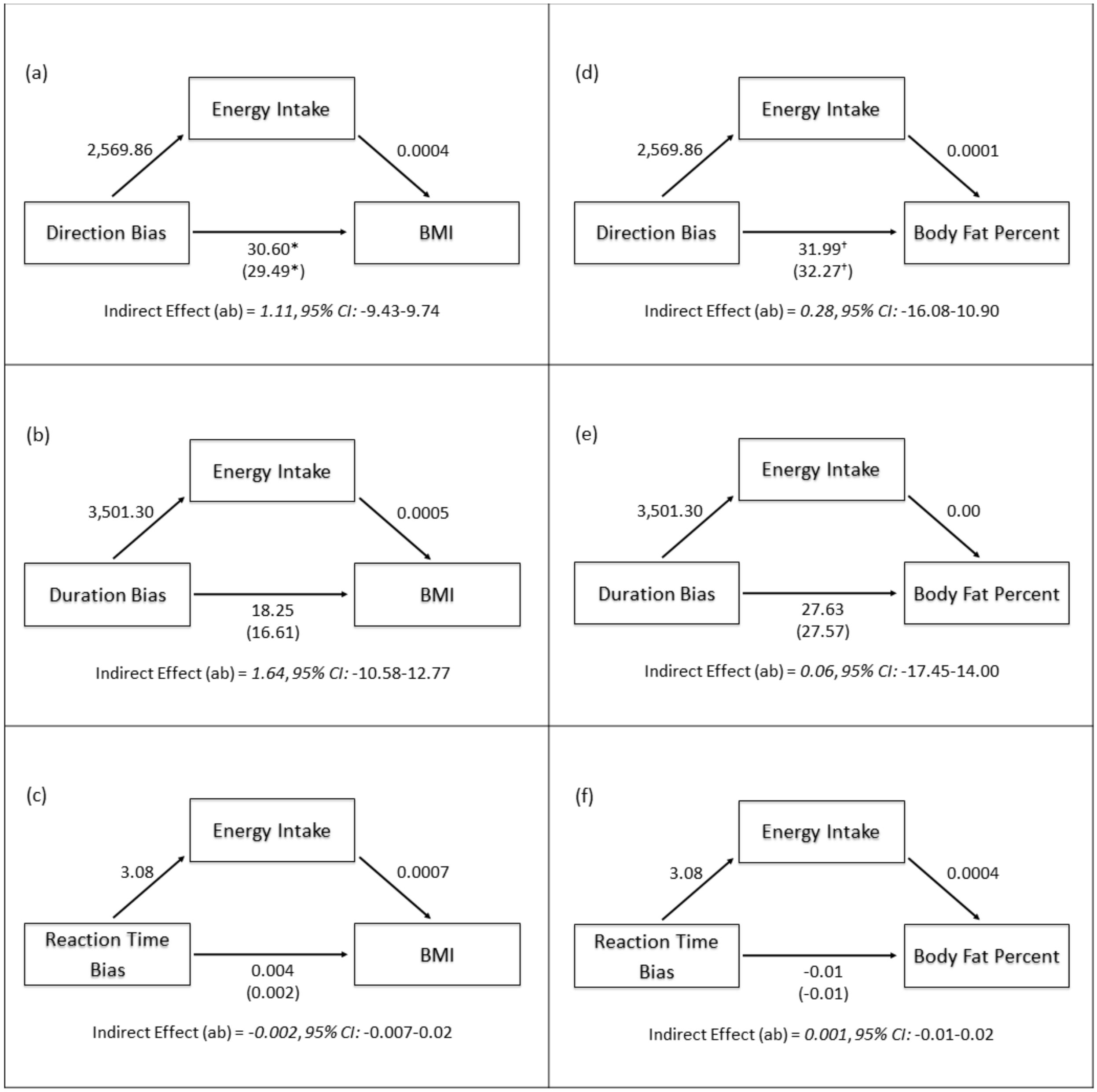

Figure 4. Energy Intake as a Mediator

Results of bootstrapping mediation analyses examining energy intake as a mediator between food attentional bias (direction bias, duration bias, and reaction time bias) and adiposity (body mass index [BMI] and body fat percent), adjusted for age, sex, race/ethnicity, and subjective hunger. All analyses were performed on participants in the high-quality eye tracking data sample who completed the ASA24 dietary recall on day 1

($n = 42$). Values represent the unstandardized regression coefficients for paths a, b, c (and path c' in parentheses). All models demonstrate that energy intake is not a significant mediator.

$*p < .05$ $^{\dagger}p < .10$

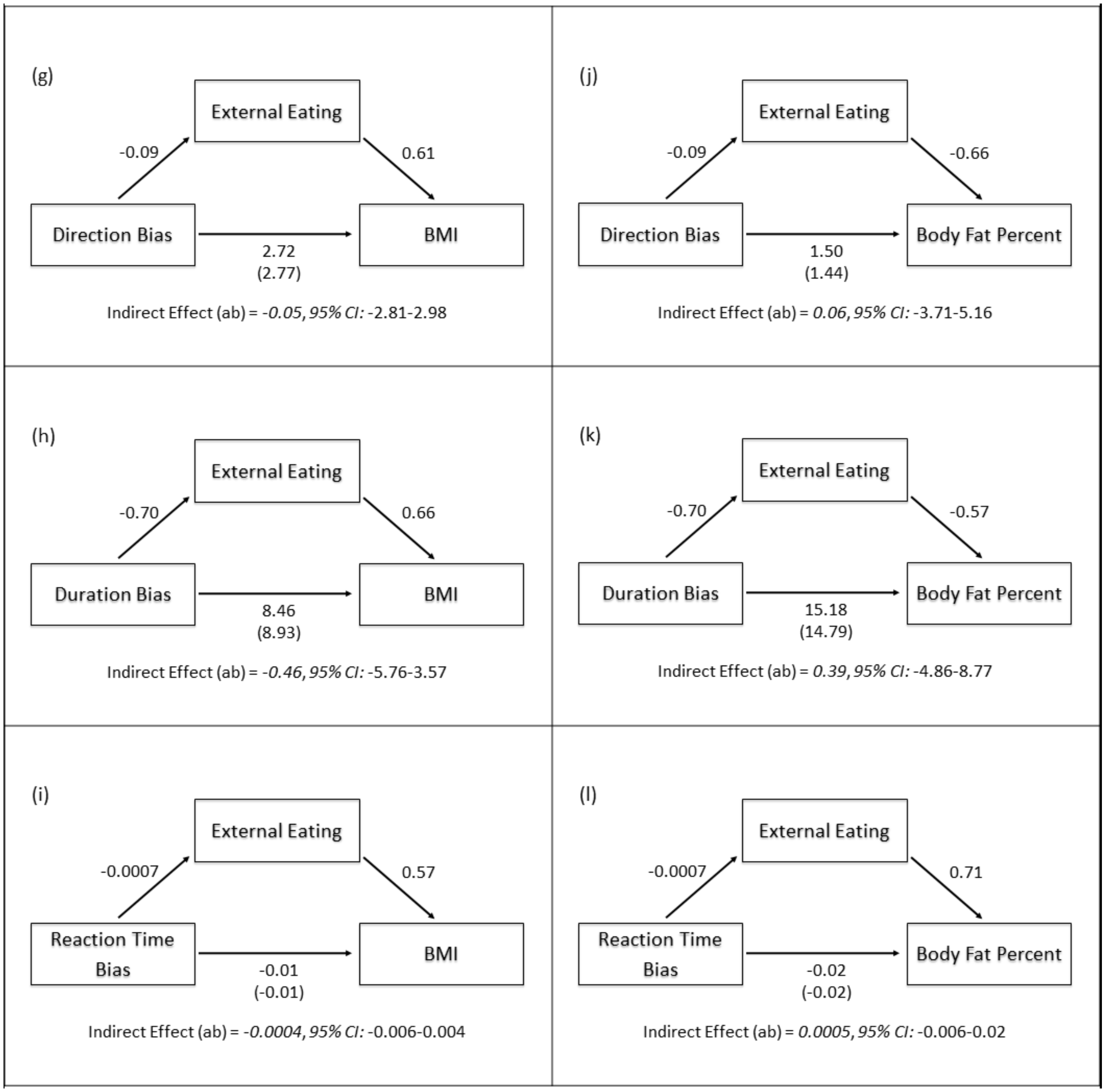

Figure 5. External Eating as a Mediator

Results of bootstrapping mediation analyses examining external eating as a mediator between food attentional bias (direction bias, duration bias, and reaction time bias) and adiposity (body mass index [BMI] and body fat percent), adjusted for age, sex, race/ethnicity, and subjective hunger. All analyses were performed on the high-quality eye tracking data sample (*n* = 61). Values represent the unstandardized regression coefficients for paths a, b, c (and path c' in parentheses). All models demonstrate that external eating is not a significant mediator.

$^{*}p < .05$ $^{\dagger}p < .10$