AFFECT REGULATION MODULATES BRAIN RESPONSE TO FOOD PICTURES IN OBESE PARTICIPANTS

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Abstract

The present study uses functional magnetic resonance imaging (fMRI) to investigate whether differences in self-reported emotion regulatory ability are associated with differential patterns of responding to food images. Thirty-five obese individuals were scanned while viewing images of food (Food) and animals (Nonfood) both in both fasted (Pre-meal) and fed (Post-meal) states. Emotion regulation was measured using The Emotion Amplification and Reduction Scales (TEARS), and a subset of the participants were chosen for analyses based on a quartile split of the subscale (Amplification, Reduction) scores, resulting in High and Low TEARS Reduction (HTR, LTR) and High and Low TEARS Amplification (HTA, LTA) groups. HTR versus LTR group differences were found in the dorsolateral prefrontal cortex (DLPFC) and orbitofrontal cortex (OFC). HTA versus LTA group differences were found in OFC, lateral prefrontal cortex (PFC), anterior cingulate cortex (ACC), and medial prefrontal cortex (MPFC). Findings suggest that differences in emotion regulatory ability are related to differential brain response to food and hunger.

Keywords: emotion, brain, obesity

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Table of Contents

List of Tables

List of Figures

Appendix: Questionnaires Administered

Health History Questionnaire

The Emotion Amplification and Reduction Scales (TEARS)

Pittsburgh Sleep Quality Index (PSQI)

Center for Epidemiologic Studies Depression Scale (CES-D)

Affect Regulation Modulates Brain Response to Food Pictures in Obese Participants

Overview

Obesity (body mass index [BMI] ≥ 30 kg/m^2) has become a significant public health problem in the United States despite the popularity of a variety of diets and the increased accessibility of health information. It is evident that people experience great difficulty achieving weight loss and maintaining healthy weight over time (Weiss, Galuska, Khan, Gillespie, & Serdula, 2007), but a conclusive reason for this difficulty remains elusive. In an effort to better understand vulnerability to obesity, various lines of research have addressed the interactions between the neural, cognitive, and emotional correlates of eating behavior (e.g., Macht, 2008; Polivy, Heatherton, & Herman, 1988; Polivy & Herman, 1999; Simmons, Martin, & Barsalou, 2005). Recent neuroimaging studies (e.g., Martin et al., 2010) have focused on clarifying the relations among reward motivation, related constructs such as behavioral impulsivity, and brain response to food cues. Findings from these studies show that the brain responds to food images with changes in brain activity in limbic, paralimbic, and various prefrontal cortical regions, all of which mediate the experience and regulation of emotion. It is known that food has an affect-inducing quality that heavily influences eating behavior. Taken together, affect regulation at a neurobiological systems level of analysis appears to be inextricably related to a person's ability to down-regulate the salience of affect-inducing cues, including food images, and to control his or her behavioral response to these cues. The present study will examine whether self-reported ability to regulate emotion is a predictor of brain activation response patterns to images of appetizing food in obese participants. It is anticipated that a better understanding of these relationships may contribute to individually tailored and more effective obesity treatments.

The Problem of Obesity

Obesity in the United States has risen at staggering rates over the past 20 years to a current prevalence of roughly 34% of American adults (Ogden, Carroll, McDowell, & Flegal, 2007). The health consequences of obesity include premature death, cardiovascular disease, diabetes, cancer, breathing problems, arthritis, reproductive complications, increased surgical risk, and emotional problems such as depression. In the year 2000, obesity-related health care cost about $117 billion (U.S. Department of Health and Human Services [USDHHS], 2001). Additionally, from 1987 through 2001, the treatment of obesity-related diseases has accounted for 27% of medical costs (Centers for Disease Control and Prevention [CDC], 2009). Clearly obesity has created a significant burden on the healthcare system, the economy, and the well-being of the general public.

Obesity is a complex medical condition, and parsing the biological and psychological mechanisms behind overeating remains a significant challenge. However, it has been established that obesity is fundamentally a problem of energy balance in which calories consumed exceeds calories expended. Recently, the etiological contribution of eating behavior, specifically overeating, to obesity has gained increased attention among researchers. A multitude of biochemical and neurally mediated hunger and satiety signals (see Erlanson-Albertsson, 2005, for a review) interact with brain responses, psychological factors, and environmental cues (Lowe & Levine, 2005) to influence the initiation and cessation of eating behavior. Individual differences in the ability to regulate hunger and satiety cues therefore play an important role in mediating food motivation and eating behavior.

Psychobiological Motivation of Eating Behavior

Psychological theories of eating behavior emphasize that the reward-related and emotion

regulatory properties of food provide powerful motivation to consume and even over-consume highly palatable food (e.g., Baumeister & Heatherton, 1996; Lowe & Levine, 2005; Muraven, Tice, & Baumeister, 1998; Stroebe, Mensink, Aarts, Schut, & Kruglanski, 2008; Stroebe, Papies, & Aarts, 2008; Vohs & Heatherton, 2000). Reward sensitivity and BMI have been shown to have an inverse U-shaped relationship (Davis & Fox, 2008; Davis, Strachan, & Berkson, 2004) such that individuals with BMI greater than 30 have lower levels of reward sensitivity on various self-report measures. Davis and Fox (2008) argued that low reward sensitivity might result in self-medication with food in order to increase positive affect. Indeed, eating is a common strategy for regulating negative emotion (e.g., Polivy & Herman, 1999; Spoor, Bekker, Van Strien, & van Heck, 2007). Furthermore, negative emotional states can bias food choice and/or quantity of food consumed (e.g., Killgore & Yurgelun-Todd, 2006; for reviews, see Gibson, 2006; Macht, 2008). Individuals experiencing negative affect tend to engage in coping behaviors such as procrastination and eating if and when they believe that these behaviors will improve their mood, despite being aware of the negative consequences of doing so (Tice, Bratslavsky, & Baumeister, 2001). Under these circumstances, the goal of immediate gratification overrides the pursuit of long-term health-related goals. The rewarding and emotion regulatory properties inherent in food reinforce this pattern of eating behavior that appears to be instrumental in the development and persistence of obesity (Epstein, Leddy, Temple, & Faith, 2007).

Despite psychological motivators to eat, it cannot be concluded from this literature alone that these are responsible for high rates of obesity. On the contrary, there appear to be differences in brain response to food images, hunger, and satiety between obese and non-obese individuals. For example, data from positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies (e.g., Gautier et al., 2001; Martin et al., 2010;

Stoeckel et al., 2008) suggest that, in obese individuals compared with their healthy weight peers, brain regions and circuits associated with reward processing and emotion regulation may respond differently to food cues and hunger/satiety states. Recent psychological and neuroscientific research has investigated the individual differences in neural, emotional, and behavioral responses to food cues. It may be inferred from this literature that the eating behavior that contributes to obesity is driven in part by vulnerability at a neural systems level of analysis.

Self-Regulation

Self-regulation encompasses a variety of "processes that enable an individual to guide his/her goal-directed activities over time and across changing circumstances (contexts)," and more specifically refers to "modulation of thought, affect, behavior or attention via deliberate or automated use of specific mechanisms" (Karoly, 1993, p. 25). In behavioral and psychological research, self-regulation has typically been operationalized in terms of executive function (EF). EF is a broad category of constructs that encompasses a multitude of discrete but related emotional, cognitive, and behavioral processes (Banich, 2009). In general, researchers agree that inhibition of a learned, automatic, or prepotent response is one category or type of EF that is inherent in self-regulation (Banich, 2009; Miyake et al., 2000). Inhibitory control across neurobiological and emotional levels of analysis appears to be critical to the decision-making process involved in eating behavior.

Affect regulation

The maintenance of physical and mental health is determined in part by an individual's self-regulatory ability. Affect regulation, or the ability to modify valenced states to achieve optimal well-being, is closely linked with an individual's capacity to selectively engage in behavior congruent with long-term goals while simultaneously avoiding behavior that is solely

focused on temporarily improving affect (Tice, Bratslavsky, & Baumeister, 2001). Gross (1998) defined emotion regulation as the "processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions" (p. 275). In general, affect regulation serves two self-regulatory goals, either to decrease negative emotions or increase positive emotions (Hamilton et al., 2007). Polivy (1998) argued that self-regulation depends on a balance between immediate and long-term goal states in order to create a positive emotional experience. The processes employed to achieve these self-regulatory goals include controlling attention, controlling cognitive antecedents, and controlling behavioral responses (Gross, 1998). Together, these processes are integral in the selection of long-term goals over short-term gratification (Karoly, 1999).

Historically, the affect regulation process has been difficult to measure. Multiple scales and questionnaires have been developed to assess individual differences in emotion and emotion regulation. For example, the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) inquires as to the extent to which individuals have felt a variety of positive and negative valenced emotions. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) assesses individuals' appraisal of their awareness and clarity of their emotions, as well as emotion regulation strategies and ability to engage in goal-directed behavior. However, the DERS focuses on negative affect regulation, as evidenced by the fact that most of the items begin with the stem "When I'm upset." Similarly, the Expectancies for Negative Mood Regulation Scale (NMR; Catanzaro & Mearns, 1990) employs the stem "When I'm upset, I believe that" in order to determine whether an individual expects that his or her efforts to regulate negative affect will be successful. The Trait Meta-Mood Scale (TMMS; Salovey, Mayer, Goldman, Turvey, & Palfai, 1995) assesses similar constructs (i.e., attention, clarity, and

repair) as those investigated by the DERS.

Hamilton and colleagues (2009) argued that these assessments do not measure the process of emotion regulation. Instead, these instruments measure self-reported felt emotion (e.g., PANAS), are confounded with personality traits such as optimism (e.g., TMMS), substantially overlap with the measured outcome (e.g., DERS), assess strategies for coping with emotional experiences and events (e.g., NMR), and inquire about metacognitive function such as awareness of emotional experience (subscales from DERS and TMMS). This lack of construct specificity may in part explain why affect regulation and overeating together have not been widely researched.

The Emotion Amplification and Reduction Scales (TEARS). TEARS (Hamilton et al., 2009) were developed with these limitations in mind. TEARS were designed to capture the fundamental processes of emotional self-regulation and is intended to be valence nonspecific. The scale is intended to measure the extent to which individuals feel they are able to upregulate and downregulate emotion states, regardless of whether these states are positively or negatively valenced. Therefore, the focus is less on how effectively individuals regulate positive versus negative emotions, and more on whether individuals feel they can change the trajectory of an emotional response. The emotion amplification subscale is characterized by a person's ability to *intensify*, *harness*, or *prolong* emotions or the effects of an emotion. Emotion reduction is characterized by a person's ability to *soften*, *shorten*, *stop*, or *prevent* emotions or their effects. Factor analyses of the emotion reduction and amplification subscales demonstrated evidence of both convergent and discriminant validity. The emotion reduction subscale covaried inversely with depressive symptoms, fatigue, and negative affect and was uncorrelated with positive affect. The emotion amplification subscale covaried with positive affect, fatigue, and inversely with

negative affect and was uncorrelated with depressive symptoms. These data indicate that emotion amplification and reduction are related but discriminable processes.

Self-Regulation of hunger and eating behavior: Why we eat and overeat

In general, eating behavior is governed by two self-regulatory tasks, that of knowing when one is hungry and that of knowing when one is full. In particular, the self-regulation of hunger functions to prioritize proximal (e.g., food-seeking) goals with more distal (e.g., weight management) goals. Food motivation, or the desire to seek and consume food, comprises at least two separate but interactive processes (Berthoud, 2004). The biological need to eat (homeostatic hunger) and the psychological desire to eat (hedonic hunger) are difficult to parse when considering food motivation and eating behavior. Homeostatic hunger is driven by central and peripheral biochemical signals that balance energy intake with energy expenditure (Erlanson-Albertsson, 2005; Yeomans, Blundell, & Leshem, 2004). Thus, homeostatic hunger drives eating behavior when individuals are in a fasted state. This is a complex process best understood through biological models and is therefore beyond the scope of this paper. However, it is important to note that the majority of people living in industrialized nations do not experience severe food and energy deprivation, and therefore most eating behavior is motivated by something other than biological need (Lowe & Levine, 2005). In contrast to homeostatic hunger, hedonic hunger is primarily motivated by biopsychosocial factors such as reward sensitivity, impulsivity, and limited availability of self-regulatory resources. The many affectively pleasant aspects of food and eating, including but certainly not limited to taste (e.g., sweet, salty, fatty) and context (e.g., dining at a restaurant with close friends, parties and celebrations), have likely contributed to the development of an obesigenic environment by increasing the salience and reinforcement value of hedonic hunger cues (Lowe & Butryn, 2007).

Hedonic hunger. Hedonic hunger may be further parsed into two separate processes of 'liking' versus 'wanting' food (Berridge & Robinson, 2003). 'Liking' is affective in nature, driven by the hedonic salience of food, whereas 'wanting' is behavioral, characterized by the motivation to seek food. Berridge and Robinson (2003) suggested that 'liking' and 'wanting' refer respectively to the pleasure involved in eating and the desire to eat. It is important to note that 'wanting' food may occur independently of reduction of physiological hunger (Finlayson, King, & Blundell, 2008), implying that hedonic food motivation is driven by separate processes from those that modulate homeostatic food motivation. Rothemund and colleagues (2007) found that obese individuals rated low calorie foods as more appetizing than high calorie foods, but still displayed greater brain activation in reward processing regions when presented with high versus low calorie food pictures. Multiple interpretations of this finding can be made, but it is possible that 'wanting' high calorie foods registers at a neural level, even when 'liking' is not consciously recognized or overtly acknowledged. This implicit wanting of food may be a risk factor for overeating, because failure to attenuate food motivation at a neural level appears to significantly influence the development and perpetuation of obesigenic eating behavior (Finlayson, King, & Blundell, 2008).

Food is a powerful reinforcer across multiple levels of analysis, though there is some evidence to suggest that food may be a more powerful reinforcer for obese compared with healthy weight individuals (Epstein, Leddy, et al., 2007; Epstein, Temple, et al., 2007). For example, obese individuals may be more motivated to work for food rewards than are their non-obese peers (Saelens & Epstein, 1996). In a decision-making task of smaller, immediate, and higher certainty versus larger, delayed, lower certainty food and money rewards, Rasmussen, Lawyer, & Reilly (2010) found that higher percent body fat was associated with greater temporal

and probability discounting for food. However, the researchers did not find a significant relationship between percent body fat and temporal and probability discounting for money. The authors inferred that, for the overweight and obese individuals in their study, food carries greater reward and incentive value than does money. These findings support the hypothesis that, especially among obese individuals, the affectively pleasant aspects of food and eating act as powerful reinforcers that are inadequately limited by biological and psychological inhibitory mechanisms.

Self-regulatory failure: A story of disinhibition

The self-regulation of hunger and satiety states depends on the integrity of systems that inhibit behavior and efficiently modulate emotion states. Low inhibitory control at both emotional and behavioral levels (i.e., disinhibition) may increase the likelihood of overeating (Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2008). Behavioral disinhibition of eating is characterized by patterns of overeating, especially when this eating serves an emotion regulatory function and/or is perpetuated by the rewarding properties of food (Bryant, King, & Blundell, 2007).

The limited strength model of self-regulation: 'When' are people vulnerable to disinhibition? Self-regulatory strength refers to an individual's personal (e.g., cognitive) resources that enable him or her to override automatic behaviors and impulses. Feelings and emotions such as fatigue and stress increase demands on and therefore limit self-regulatory strength (Baumeister & Heatherton, 1996; Muraven, Tice, & Baumeister, 1998; Vohs & Heatherton, 2000). Demands for self-control tend to be followed by difficulty maintaining regulatory focus, even when these demands are in unrelated domains. In other words, self-regulatory demands increase the likelihood of disinhibition.

Many obese individuals display restrained eating habits, or dietary restraint, wherein they control food intake in an effort to control their weight. This places a significant demand on self-regulatory strength (Stroebe, Mensink, Aarts, Schut & Kruglanski, 2008; Stroebe, Papies & Aarts, 2008). Some restrained eaters seem more vulnerable to the hedonic aspects of food than do unrestrained eaters (Stroebe, Mensink et al., 2008). Particularly for restrained eaters, the self-regulation involved in dietary restraint may draw from the limited resources. When other regulatory demands, such as regulating unpleasant emotions and feelings, are placed on the individual, he or she is left with fewer personal resources with which to control eating behavior.

A significant demand on self-regulatory resources is the need to regulate affective states. The self-referential processing (i.e., self-monitoring) required for successful goal-directed (e.g., weight loss) behavioral regulation (Karoly, 1993) represents a catch-22 for many people attempting to regulate their eating behavior. On one hand, individuals engaging in dietary restraint (i.e., dieting or controlling food intake) who fail to self-monitor may actually become disinhibited under certain conditions and eat more than intended (Heatherton, Polivy, Herman, & Baumeister, 1993). On the other hand, excessive or inappropriate self-focus may interfere with both regulatory efforts and goal achievement if it results in heightened awareness of negative characteristics of the self (e.g., body image dissatisfaction). Negative self-esteem has been hypothesized to contribute to dietary disinhibition (Polivy, Heatherton, & Herman, 1988). The need to regulate the consequent negative affect is an additional self-regulatory demand that could contribute to difficulty controlling eating behavior. Specifically, self-referential negative affect, particularly among restrained eaters, appears to motivate mood reparative behaviors such as eating (Heatherton, Striepe, & Wittenberg, 1998) or even binge eating (Heatherton & Baumeister, 1991). Thus, individuals who experience difficulty controlling negative emotions,

particularly self-related negative affect, may be more vulnerable to disinhibited eating.

Critically, suppression is distinct from regulatory processes that result in a change in the trajectory of an emotional response. Suppression of emotion is a specific, effortful process in which an individual attempts to inhibit conscious experience of thoughts and feelings associated with a particular emotion (Gross, 1998). To some extent, then, emotion suppression is an indication of failure to regulate emotions efficiently. Given the demanding, resource consuming nature of suppression, it is not surprising that it has been linked to dietary disinhibition during a task immediately following emotion regulation demands. Individuals already experiencing persistent negative affect, such as depression, who then suppress their emotional responses to distressing stimuli may be especially vulnerable to subsequent disinhibition of eating behavior, leading to increased caloric consumption (Dingemans, Marijn, Jansen, & van Furth, 2009). Again, these data suggest that self-regulation can be easily derailed by negative moods and stress. Consequently, efficient regulation of negative affective states (as opposed to suppression of unwanted feelings) may be an essential skill for inhibiting the 'wanting' of food and maintaining a consistent level of dietary restraint. Difficulty with emotion regulation may therefore contribute to chronic overeating.

Various psychobiological factors have been found to influence both affective states and a person's ability to regulate his or her experience and expression of emotion. For example, disrupted quality of sleep is a symptom of depression (Radloff, 1977) and likely moderates a person's ability to cope with pain and adversity (Hamilton, Catley, & Karlson, 2007; Hamilton, Nelson, Stevens, & Kitzman, 2007). Additional, individual differences in the motivation to approach or withdraw from rewarding or punishing stimuli are likely a behavioral marker of affect regulatory ability. Therefore, in examining a person's self-reported ability to regulate

affective states, it is necessary to consider a multitude of other closely related constructs that index emotional and behavioral regulation and dysregulation.

Self-Regulation, Affect, and Eating Behavior: Neural Systems Level of Analysis

The reinforcing power of food, as well as the abundance of food and pervasiveness of eating occasions, means that humans must expend a great deal of self-regulatory strength in order to modulate behavioral responses to the affective components of food. The additional resources required for suppression and inhibition place additional demands on the neural systems responsible for controlling the salience of reward. Neural antecedents such as cortical activation and dopamine metabolism influence behavioral abilities such as emotion regulation and executive functions. In turn, these behavioral abilities directly impact behavioral outcomes such as goal-directedness, response flexibility, and psychological well-being (Declerck, Boone, & De Brabander, 2006). If this model is extended to understanding eating behavior, then affect regulation may contribute to brain response to food cues. Considering the brain activation patterns of emotional self-regulation may help to clarify the motivation behind eating-related decisions and the behavioral mechanisms that contribute to obesity.

Brain imaging methods

Brain imaging research using techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have contributed a wealth of information toward understanding the neural systems involved in psychological constructs such as reward sensitivity and impulsivity, as well as processes such as emotion generation and regulation and decision-making. Furthermore, brain imaging has begun to highlight how obese and non-obese individuals differ at a neural level in response to food images and hunger and satiety states. Findings from this literature indicate that there is significant overlap in regions of the brain that

process food motivation as well as emotion. For example, the rewarding properties of food appear to be processed in many of the ventral and medial PFC areas, including specific regions such as the medial PFC (MPFC) and orbitofrontal cortex (OFC). In contrast, the self-regulation of eating behavior appears to be modulated by dorsal and lateral PFC areas, including dorsolateral PFC (DLPFC).

Food motivation at a neurobiological level of analysis: Sensitivity to food-related reward. Given the rewarding nature of food, it is not surprising that neuroimaging studies have consistently reported activation in areas of the brain considered to mediate reward motivation in response to food cues. In particular, the mediation of positive affect, reward processing, and integration of affective and somatic information appears to differentially activate ventral and medial regions of the prefrontal cortex (PFC), including the medial PFC, anterior cingulate cortex (ACC) and the orbitofrontal cortex (OFC). Indeed, Potts et al. (2006) argued that the medial frontal cortex is heavily influenced by the dopamine reward system, a circuit critically involved in the amplification of positive affect and the motivation of behavior. Furthermore, the OFC is functionally connected with the ventral striatum, a limbic region heavily involved in reward processing (Di Martino et al., 2008). Within the ventral striatum, the nucleus accumbens has functional projections to the medial OFC. Individuals high in trait impulsivity (Martin & Potts, 2004) and reward sensitivity (Beaver et al., 2006) display elevated OFC activity in response to rewards.

The OFC is a convergence point for somatic, sensory, and affective information as evidenced by its direct connections with both cortical and subcortical (i.e., limbic) structures (Price, 1999; Rolls, 2000). For example, gustatory, olfactory, visual and somatic information is qualitatively encoded (e.g., pleasant or aversive) in the OFC. Thus, the OFC is especially

involved in processes guided by anticipated rewards and punishments, such as decision-making (Rolls & Grabenhorst, 2008) and goal-directed learning (Valentin, Dickinson, & O'Doherty, 2007). The left OFC has been shown to be involved the taste and reward aspects of eating (Kringelbach, O'Doherty, Rolls, & Andrews, 2003; O'Doherty, Kringelbach, Rolls, & Andrews, 2001; Simmons et al., 2005). For example, a group of non-obese, non-dieting women showed greater left OFC activation in response to pictures of "fattening" foods compared with nonfood objects (Schur et al., 2009). However, Zald (2009) argued that the left OFC is more heavily involved in processing unpleasant taste, whereas it is the right OFC that activates in response to pleasant taste. Despite these apparent discrepancies, it can be concluded that, in general, the role of the OFC in reward processing is to encode the reward value of stimuli, including food, and to facilitate stimulus-reward association learning (Rolls, 2000; 2004).

Interestingly, satiety signals in the OFC influence taste and flavor responses, thereby influencing the reward value of food (Rolls, 2006). Goldstone et al. (2009) found that presentation of high and low calorie food images to healthy weight participants resulted in higher medial and lateral OFC activation to high calorie foods when the participants were fasted versus fed. In this same study, low calorie images evoked the opposite activation pattern (fed was greater than fasted). In other words, hunger was associated with greater medial and lateral OFC activation to high calorie foods and lower activation to low calorie foods. Individuals craving chocolate displayed greater OFC response to both taste and sight of chocolate than did individuals who were not craving chocolate (Rolls & McCabe, 2007). In sum, it appears that the OFC differentially processes visual food cues in conditions of high food motivation (e.g., hunger, high calorie).

The processing and integration of affective and somatic information associated with

reward-related decision-making (Blair et al., 2006; Northoff et al., 2006) has been associated with elevated blood flow to the vmPFC (Bechara, Damasio, Damasio, & Lee, 1999; Blair et al., 2006) and the ACC (Blair et al., 2006; Paus, 2001). The ACC integrates somatic, autonomic, motor, cognitive, and emotional information and is part of a network that determines attention and subsequent responses to these potentially conflicting stimuli (Bush, Luu, & Posner, 2000; MacLeod & MacDonald, 2000; see Paus, 2001 for a review). Because eating behavior is a decision-making process requiring conflict monitoring and overriding automatic responses, the role of the ACC is likely to regulate these processes (Botvinick, 2007; Paus, 2001).

The MPFC has been shown to activate during self-referential processing and to deactivate during tasks of working memory and cognitive demand (Greicius, Krasnow, Reiss, & Menon, 2003; Kelley et al., 2002; Pochon et al., 2002). In an event-related fMRI study, Piech et al. (2010) presented participants with appetizing menu options and instructed participants to choose which option they would prefer to "eat" (based on appetizing value, an affective decision) and which they would prefer to "cook" (based on ease of cooking, a cognitive decision). Piech et al. found that in an "eat" greater than "cook" contrast, the MPFC was significantly activated. In the opposite contrast, the lateral OFC was activated, suggesting that medial PFC regions play a greater role in affective and reward-related decision-making, whereas lateral PFC regions play a greater role in cognitive decision-making.

Several of the areas of the brain involved in motivating eating behavior include the amygdala, medial frontal cortex (including the OFC and vmPFC), ACC, and basal ganglia (e.g., Grabenhorst, Rolls, Parris, & d'Souza, 2009). Together, these regions likely modulate food motivation by detecting and encoding the affective and rewarding properties of food. The processing of some orosensory cues such as fat content appears to be associated with activity in

the ACC and the OFC (de Araujo & Rolls, 2004). In a study of healthy weight individuals, viewing high calorie versus low calorie images in a fasted state resulted in reward system activation, including medial and lateral OFC activation (Goldstone et al., 2009). Thus, coherence of individual differences in the ability to manage reward cues is evident across neural, personality, and behavioral levels. Management of reward-related information relies heavily on both the OFC and ACC, and both regions tend to activate during the presentation of conflicting information. Taken together, the fact that somatic and affective information processing converge in the same regions of the brain provides support for the hypothesis that affect regulation and control of eating behavior share a common basis for regulatory control.

Brain regions of self-regulatory resources and inhibition. Whereas reward sensitivity and impulsivity appear to differentially activate portions of the ventral and medial prefrontal cortex, the dorsal and lateral prefrontal cortex appear to be heavily involved in the inhibition and regulatory control of emotion and behavior. The DLPFC has reciprocal connections between sensory, motor, and limbic regions of the brain, facilitating convergence of sensory, motor, affective, and motivational information in this region (Miller & Cohen, 2001). The DLPFC is highly involved in executive function because of its role in complex cognitive, emotional, and behavioral regulation. For example, the DLPFC is implicated in voluntary suppression of negative affect, a form of effortful emotion regulation that demands cognitive, autonomic, and behavioral resources (Phan et al., 2005). The ventrolateral prefrontal cortex (VLPFC) is known to play a role in inhibition and shifting (Aron, Robbins, & Poldrack, 2004; Hedden & Gabrieli, 2010). Additionally, the left VLPFC may be involved in the downregulation of affective and somatosensory response to visual information (Anders et al., 2009).

In addition to areas such as the DLPFC, the OFC and ACC have been found to contribute

to the process of self-regulation, likely due to their involvement in encoding and interpreting somatosensory information and visual cues such as food images. Whereas it seems counterintuitive to suggest that these regions are involved in both food motivation and control of eating behavior, it is possible that the OFC processes the conflict between choosing immediate versus delayed rewards. Individual differences in the magnitude of anticipated immediate rewards would explain this region's response to both rewarding stimuli and regulatory demands. Additionally, regulation of attention to affective states such as negative emotions may be directly mediated by the ACC. The regulation of negative affect, which also requires awareness and interpretation of sensory (e.g., visual), somatosensory, and affective information, as well as of rewards and punishment, may be mediated by this region as well. Given the need to inhibit the positive affect (i.e., reward) induced by food cues, it is not surprising that successful control over eating behavior is characterized by brain regions typically active when individuals suppress or inhibit their responses to stimuli.

Neural systems of self-regulatory resources, inhibition, and eating behavior. Neuroimaging studies have demonstrated that processes of emotion regulation and control over eating behavior are mediated by many of the same regions of the brain. These processes appear to be driven in large part by the ACC, dorsal and lateral PFC, and medial and orbital PFC (Eippert et al., 2007; Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Kim & Hamann, 2007; Ochsner et al., 2004). For example, restrained eaters who consumed a liquid meal to satiety had greater dorsal PFC activation in response to satiety than did unrestrained eaters (Del Parigi et al., 2007). Additionally, successful dieters had enhanced blood flow to inhibitory control regions during while viewing food pictures (McCaffery et al., 2009). It is apparent that dietary restraint, particularly in healthy weight individuals, appears to be controlled by many of

the same brain regions as those that control emotion regulatory processes, suppress thoughts, and inhibit behavior.

In some studies, satiety has been positively associated with activation in the left DLPFC (Pannacciuli et al., 2007), bilateral DLPFC and ventrolateral PFC (VLPFC; Tataranni et al., 1999), and the left lateral PFC (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). In contrast, other studies have found a positive association between the left DLPFC and food motivation stimuli such as food images and taste. Kringelbach, de Araujo, and Rolls (2004) argued that the left DLPFC might be involved in taste processing. In their fMRI study, participants tasted various flavors (e.g., chocolate, tomato), including a tasteless control liquid. An unexpected finding of the study was that response in the left DLPFC was associated with taste stimuli. The authors suggested that this finding might indicate that the DLPFC plays a role in generating cognitive and behavioral processes related to the initiation and cessation of eating. Similarly, Schur et al. (2009) found that the left DLPFC was more responsive to images of "fattening" foods compared with objects.

Food Motivation and Obesity

There is converging evidence suggesting that, at a neural level, obese individuals react to food cues differently than do healthy weight individuals. Studies in which food motivation has been manipulated have yielded different response patterns between obese compared with healthy weight (BMI between 18.5 and 24.9 kg/m^2) individuals. These differences have been observed in regions known to process food-related reward, such as MPFC, OFC, and ACC, as well as those regions believed to be involved in self-regulation, such as DLPFC.

During a pre-meal food motivation paradigm, obese participants displayed greater ACC and MPFC activation compared to healthy weight participants (Martin et al., 2010). During a

post-meal food motivation paradigm in this same study, MPFC activation remained greater in obese versus healthy weight participants. A critical finding from this study is that, not only were emotion regulatory regions of the brain activated, but this activation was sustained in obese participants even after eating. It is important to note that these participants were not afforded the opportunity to eat until satiety. Therefore, one possible explanation of these findings is that eating had a priming effect, which led to continued activation in the MPFC post-meal. Gautier et al. (2001) found that obese compared with lean women showed significantly greater decrease in OFC activation from hunger to satiated states. This finding indicates that, among the group of obese women, the OFC response while in a state of hunger was greater than the OFC response among lean women.

These differing response patterns have also emerged in comparisons of high and low calorie food pictures (Rothemund et al., 2007). When participants in a eucaloric state were shown images of high and low calorie foods in an fMRI block design, obese, but not healthy weight, women activated the OFC in response to pictures of high calorie foods. Furthermore, BMI was positively correlated with OFC response to images of high calorie foods. Similarly, Stoeckel et al. (2008) found that obese compared with healthy weight individuals displayed greater OFC and ACC activation to pictures of both low and high calorie foods. Moreover, these two studies also found that obese individuals responded to food images with enhanced activity in the brain reward system, including ventral and dorsal striatal structures such as caudate, putamen, and nucleus accumbens, as well as limbic structures such as amygdala and hippocampus. Given the role of these regions in mediating emotion-related response to food cues, it may be inferred that obesity is associated with heightened affective reactivity (e.g., reward sensitivity) to energy dense, highly palatable food.

Self-regulation of eating behavior in obesity. It appears that dorsal and lateral PFC areas of the brain are critical in the behavioral inhibition responsible for the cessation of eating. Both obese men and women have been found to exhibit less dorsolateral PFC activation following consumption of liquid nutrition until satiation (Le et al., 2006). Formerly obese women high in dietary restraint (as measured by the Three Factor Eating Questionnaire) are not only leaner than their non-dieting obese peers, but are also more effective at recruiting areas of the brain responsible for behavioral control (e.g., dorsal PFC; Del Parigi et al., 2007; Le et al., 2007). Dietary restraint appears to be positively correlated with dorsal PFC activity and negatively correlated with OFC activity (Del Parigi et al., 2007). Additionally, successful dieters displayed enhanced blood flow to inhibitory control regions while viewing food pictures (McCaffery et al., 2009). Collectively, these findings argue for the engagement of dorsal and lateral PFC in control of eating behavior.

It may be concluded from the neuroimaging literature that dorsal and lateral PFC regions may be responsible for top-down inhibitory control of medial portions of the PFC. Taken together, neuroimaging research indicates that, when obese individuals are presented with food cues while in a hunger state, they display elevated levels of activity in brain areas associated with affect and reward processing and diminished levels of activity in regions typically involved in behavioral inhibition. Furthermore, increased demands on self-regulatory resources via top-down affect regulation processes may further compromise control over eating behavior, particularly when the emotion-related response to food cues is heightened or intense. Failure to recruit brain regions responsible for affect regulation may result in overeating (i.e., hedonic eating), leading to overweight and obesity. In contrast, healthy weight individuals are able to selectively recruit areas of the dorsal and lateral PFC and suppress activity of the ACC and OFC

after they have eaten. It appears that the capacity for lean people to maintain a healthy body weight relies on neural pathways that promote inhibition of eating.

Conclusions from Neuroimaging Research

Given this literature, there is ample evidence suggesting that affective states and food motivation are processed in the same regions of the brain. Likewise, emotion regulation and control over eating behavior appear to be modulated by many of the same brain regions. Findings from neuroimaging research suggest that for most individuals, regardless of body composition, food cues typically evoke powerful affective, and specifically reward-related, responses within the brain. However, for obese individuals, the immediately rewarding properties of food are powerful enough to consistently override long-term health-related goals. At a neural level, ventral and medial PFC activation has been observed in response to the affective properties of food stimuli. These regions are likely influence eating behavior by modulating food motivation and the salience of affective food properties. Dorsal and lateral PFC regions appear to downregulate the neural response to affective and somatic information, resulting in enhanced inhibitory control over food-related reward. The fact that there are differing patterns of neural activity in these regions between obese and healthy weight individuals lends support to the argument that obesity may be driven at least in part by enhanced affective reactivity to food cues coupled with failure of inhibitory control regions to "come online" and promote cessation of eating. Thus, capacity for self-regulation, including regulation of affective and specific emotional responses to food, may play a critical role in the perpetuating eating behavior that contributes to moderate obesity.

The Present Study

Purpose

The aim of the present study was to build on existing research on individual and group differences in brain response to visual food cues. To this end, this study aimed to determine whether perceived ability to self-regulate emotion would be associated with differences among obese individuals in how the brain responded to highly appetizing food pictures when hungry (Pre-meal) and after eating a small meal (Post-meal). In an effort to examine brain response to food cues in fasted (before eating lunch) and fed (after eating lunch) states, participants were shown images of food and animals (nonfood). A whole-group analysis was conducted to investigate the effects of food motivation conditions on all participants. Following the whole-group analysis, two separate analyses were conducted using TEARS Reduction and Amplification subscales. Selection of participant data for these analyses was based on a quartile split of the data for each subscale, with individuals scoring in the highest quartile being "high" amplifiers or reducers and those in the lowest quartile being "low" amplifiers or reducers. Between-group fMRI analyses were conducted to assess for response differences between high and low reducers as well as between high and low amplifiers.

This study was part of an ongoing longitudinal fMRI and diet intervention study investigating brain function predictors of weight loss and weight loss maintenance. In the longitudinal study, obese participants were recruited to participate in a nine-month weight loss and maintenance behavioral intervention that involved moderate calorie restriction, titrated physical activity, and group psychoeducation. Healthy weight controls were also recruited to undergo fMRI and cognitive testing but not the weight management intervention. Scanning sessions took place both before and after the three-month weight loss portion of the intervention.

The present study was a cross-sectional investigation that included only obese participants at their baseline (pre-diet) fMRI sessions.

Hypotheses

Primary hypotheses. Consistent with prior research, it was hypothesized that participants would display significant differences in brain activation patterns between Food and Nonfood images, as well as between Pre-meal and Post-meal conditions. It was also hypothesized that self-reported ability to regulate emotions would modulate brain activation patterns among conditions of food motivation.

Images and conditions. For all participants, it was expected that there would be a main effect for food images, such that participants would display greater brain activation in reward processing areas such as the mPFC, OFC, and ACC while viewing Food versus Nonfood and baseline pictures. It was also expected that this effect would be significant during the Pre-meal condition, but not during the Post-meal condition. Additionally, the activation of inhibitory centers was hypothesized to be associated with reduction of food motivation centers. Thus, BOLD activation in the DLPFC was expected to be greater in the Post-meal condition than in the Pre-meal condition when participants were viewing food pictures.

In and above between-condition responses to the experimental conditions, it was predicted that individual differences in responses to conditions would be related to perceived and reported ability to reduce and amplify emotional states.

TEARS emotion reduction. Participants reporting strong ability to reduce emotional responses (high reducers) were hypothesized to display appropriate suppression of reward response to food pictures after eating. It was predicted that this reaction would be manifested by increased lateral frontal (e.g., DLPFC) activity and decreased medial frontal (e.g., MPFC, OFC)

activity from the Pre-meal scanning session to the Post-meal scanning session. In contrast, participants reporting difficulty with emotion reduction (low reducers) were expected to display no difference in medial frontal activity (i.e., MPFC, OFC) from Pre-meal to Post-meal. Among low reducers, it was expected that DLPFC activation would not be observed, and the inverse relationship between medial and lateral brain regions would not be observed.

TEARS emotion amplification. Given that affect intensity appears to correlate positively with both ventrolateral (VLPFC) and dorsomedial PFC (DMPFC; Grimm et al., 2006), it was hypothesized that individuals who report strong ability to intensify emotions (high amplifiers) would display enhanced BOLD activation in these regions in response to food pictures during both Pre-meal and Post-meal conditions.

Method

Participants

The participants were 35 (28 female) young to middle-aged adults who were obese but otherwise healthy (see Table 1). Participants were residents of northeast Kansas or northwest Missouri and were recruited through the Weight Control Research Project at the Energy Balance Laboratory on the University of Kansas-Lawrence campus. Exclusion criteria included recent weight loss or gain, high physical activity, smoking or other history of drug addiction, special dieting, medications that affect metabolism or appetite, inability to exercise, eating disorders, psychological disorders, metabolic disease, recent or current pregnancy or breastfeeding, and serious medical conditions such as diabetes, cancer, or cardiac event. All participants provided a signed physician's authorization form to participate in a weight loss program.

Questionnaires

Health History Questionnaire. This questionnaire gathered information about

demographics, general health status, medication use (prescription, over-the-counter, contraceptive, complementary or supplementary), and selected health behaviors that might represent cause for ineligibility to participate in the research study.

The Emotion Amplification and Reduction Scales (TEARS). TEARS (Hamilton et al., 2009) are an 18-item self-report index of an individual's emotion regulation ability through amplification and/or reduction of emotions. TEARS were developed in order to assess emotion regulation as a process, namely individuals' self-reported ability to amplify or reduce an emotional response. This measure has demonstrated high internal consistency and criterion validity in preliminary studies. Responses are on a 4-point rating scale, ranging from 1 (*not at all true of me*) to 4 (*very true for me*).

Center for Epidemiologic Studies – Depression Scale (CES-D). The CES-D (Radloff, 1977) is a 20-item self-report measure designed to detect depressive symptoms in the general population. The CED-D has demonstrated adequate factorial validity (Orme, Reis, & Herz, 1986) and sufficient appropriateness for use as a depression screening tool in research studies (Myers & Weissman, 1980). Responses are on a 4-point scale ranging from 0 (*rarely or none of the time [<1 day]*) to 3 (*most or all of the time [5-7 days]*).

Pittsburgh Sleep Quality Index (PSQI). The PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1988) is a 24-item self-report questionnaire designed to measure subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The first 23 items are rated on a 4-point scale from 0 (*not during the past month*) to 3 (*three or more times a week*). The last item, related to sleep quality, is rated on a 4-point scale from 0 (*very good*) to 3 (*very bad*). The PSQI has good internal homogeneity and construct validity, and is an adequate instrument for assessing general

dissatisfaction with sleep quality (Carpenter & Andrykowski, 1998).

Wechsler Abbreviated Scale of Intelligence (WASI). The WASI (Wechsler, 1999), an abbreviated version of the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III; Wechsler, 1997), is a brief intelligence test, designed to assess verbal, nonverbal, and general intelligence and cognitive functioning. The WASI was standardized on a national sample of adults and children and is linked to both the WAIS-III (Wechsler, 1997) and the Wechsler Intelligence Scale for Children – Third Edition (WISC-III; Wechsler, 1991). Age-based norms have been developed for both two- and four-subtest versions of the WASI. In the four-subtest version, Verbal IQ (VIQ) is measured by the Vocabulary and Similarities subtests, and Nonverbal, or Performance, IQ (PIQ) is measured by the Block Design and Matrix Reasoning subtests. Full scale IQ (FSIQ) is derived by summing age-based VIQ and PIQ. In the two-subtest version, only the Vocabulary and Matrix Reasoning subtests are administered, and only FSIQ, not VIQ and PIQ, can be derived. The two-subtest version was used in this study. The WASI has been deemed appropriate for screening purposes, as well as for when the goal is to acquire a relatively quick estimate of general intelligence.

Procedure

All procedures were approved by the University of Kansas Medical Center Human Subjects Committee. Researchers reviewed the informed consent document with potential participants. All participants who agreed to participate were assigned a date and time for their MRI appointment. Participants also chose from among four lunch choices. All meal choices were standardized for energy [Kcal ≈ 500] and macronutrient content (e.g., a weighed lean meat [turkey, ham, roast beef, or tuna] sandwich, slice of American cheese, vegetable [baby carrots or lettuce], fruit [strawberries, grapes, orange], and skim milk or Lactaid). Prior to coming to their

appointments, participants were instructed to eat breakfast "as usual" but to refrain from consuming anything except clear liquids within four hours of their MRI appointment. All participants were run in the late morning to early afternoon (i.e., during lunch hours) in order to benefit from normal daily hunger cycles and enhance ecological validity.

At the start of each MRI appointment, participants completed MRI safety screening. Researchers reviewed the study protocol with participants. Blood pressure, pulse, height and weight (in stocking or bare feet, all participants wearing scrubs), and waist circumference measurements were taken. Females also underwent a urine pregnancy test. The remainder of the study procedure was counterbalanced to minimize order effects. Approximately half the participants completed a Pre-meal fMRI session, ate a small meal, completed the Post-meal fMRI session, underwent cognitive testing and behavioral assessment, and filled out questionnaires. The remaining participants ate a small meal, complete the Post-meal fMRI session, underwent testing and assessment and filled out questionnaires, and completed the Pre-meal fMRI session four hours after finishing their meal. At the conclusion of MRI appointments, participants were thanked and dismissed. Participants were compensated for their participation.

fMRI Methods

fMRI cognitive activation paradigm

The experimental paradigm was based closely on a paradigm developed by LaBar et al. (2001), as used in research studies with children (Bruce et al., 2010; Holsen et al., 2005) and adults (Martin et al., 2010). Participants viewed food, animal (nonfood), and blurry images during two scanning sessions: 1) after fasting for four hours (Pre-meal) and 2) immediately after eating a small meal (Post-meal). Visual stimuli were images of food and animals obtained from

professional stock photography. Animal (Nonfood) images were used in order to control for general interest and visual richness. In a food image validation pilot study, food and animal images were rated based on the extent to which they were appetizing, exciting (arousal), and pleasant (valence), using the methods of Lang, Bradley, and Cuthbert (1999). Selected food images were significantly more appetizing than selected animal images ($p<.001$). No significant difference existed between the food and animal image groups with regard to valence or arousal ($p>.05$). These same food and animal images were blurred so as to be unrecognizable with a phase randomized Fast Fourier Transform in the MATLAB (The MathWorks, Inc., Natick, MA) program. Blurred images were used as baseline visual comparison stimuli to control for visual cortex activation during the paradigm.

The cognitive activation paradigm is represented in Figure 1. Each functional scan involved three repetitions of each block of each stimulus condition type (i.e., food, animal), alternated between blocks of blurred images. Visual stimuli were generated with Presentation (Neurobehavioral Systems, Inc., Albany, CA) on a Dell desktop computer running Windows 2000, and were then projected onto a screen behind the MRI scanner. Participants viewed the stimuli via a mirror that reflected the images on the screen. Stimulus presentation time was 2.5 seconds, with an interstimulus interval (ISI) of 0.5 seconds. Within each functional scan (6 minutes 36 seconds each), there was a total of 13 blocks of stimuli presentation; within each block, 10 images were presented, for a total of 130 data points per fMRI scan. Stimulus condition order was counterbalanced across subjects. As an attention check, participants completed a recognition memory task outside the scanner, immediately following each scanning session.

Image acquisition

Scanning was performed at the University of Kansas Medical Center Hoglund Brain Imaging Center (HBIC) on a 3 Tesla head-only Siemens Allegra scanner (Siemens, Erlangen, Germany) fitted with a quadrature head coil. Participants' heads were immobilized with head cushions. Following automated scout image acquisition and shimming procedures performed to optimize field homogeneity, a structural scan was completed. T1-weighted anatomic images were acquired with a 3D magnetization prepared rapid acquisition gradient echo (MPRAGE) sequence (TR/TE = 23/3.06 ms, flip angle = 8˚, field of view [FOV] = 192 x 100 mm, matrix = 192 x 192, slice thickness = 1 mm). This scan was used for slice localization for the functional scans, Talairach transformation, and co-registration with fMRI data. Following the MPRAGE sequence, two gradient echo blood oxygen level dependent (BOLD) sequences were acquired in 43 contiguous oblique axial slices at a 40º angle (repetition time/echo time [TR/TE] = 3000/30 ms, flip angle = 90˚, FOV = 220 mm, matrix = 64 x 64, slice thickness = 3 mm, 0.5 mm skip, in-plane resolution = 3 x 3 mm, 130 data points).

Method to minimize susceptibility artifact. In order to standardize head positioning across individuals, all participants were positioned in the scanner so that the angle of the anterior commissure-posterior commissure (AC-PC) plane was between 17˚ and 22˚ in scanner coordinate space. The angle was verified with a localization scan. To optimize signal in ventromedial prefrontal regions by minimizing susceptibility artifact from air sinuses, BOLD sequences were acquired in oblique slices at a 40º angle.

Data analysis

Based on analysis procedures used by Savage (2007), fMRI data was analyzed using the BrainVoyager QX statistical package and random effects (Brain Innovation, Maastricht,

Netherlands, 2004). Preprocessing steps included trilinear 3D motion correction, sinc-interpolated slice scan time correction, 3D spatial smoothing with 4-mm Gaussian filter, and high pass filter temporal smoothing. Collectively, these steps were intended to improve signal-to-noise ratio and reduce motion-related artifact, with the end result of increasing the likelihood of detecting true BOLD activation. Functional runs with motion of more than 4 mm along any axis (*x*, *y*, or *z*) were discarded. Functional images were co-registered to the anatomic images obtained within each session and normalized to the BrainVoyager template image, which conforms to the space defined by the Talairach and Tournoux's (1988) stereotaxic atlas. Regions of interest were verified by mapping Talairach coordinates onto two atlases (Mai, Assheuer, & Paxinos, 2003; Talairach & Tournoux, 1988). Functional MRI data were analyzed in two different steps: 1) whole brain statistical analysis and 2) region of interest analysis.

Whole brain statistical analyses. Activation maps were analyzed using statistical parametric methods (Friston et al.,1995) contained within the BrainVoyager QX software. Statistical contrasts were conducted using multiple regression analysis with the general linear model (GLM), allowing for multiple predictors (e.g., Pre- versus Post-Meal, Food versus Nonfood) to be built into the model. Regressors representing the experimental conditions of interest were modeled with a hemodynamic response filter and entered into the multiple-regression analysis using a random-effects model accounting for individual participant differences. Contrasts between conditions of interest were assessed with *t* statistics. Statistical parametric maps were overlaid on three-dimensional renderings of an averaged-group brain. Based on *a priori* regions of interest, voxel values in the OFC, MPFC, ACC, and DLPFC were considered significant if the activation survived a statistical threshold of $p<.001$ (uncorrected for multiple comparisons) and had a minimum cluster size of three contiguous voxels. Other areas

were considered significant if they exceeded a threshold of $p<.001$ (uncorrected for multiple comparisons) and had a minimum cluster size of six contiguous voxels. This approach was designed to ensure maximum statistical power for *a priori* regions in which there is strong evidence of activation from previous studies, while not missing other, potentially important yet unanticipated, activations (Holsen et al., 2005; Martin et al., 2010).

Region of interest (ROI) data analyses. Consistent with prior research studies using a similar cognitive activation paradigm, *a priori* regions of interest (ROIs) included MPFC, ACC, and OFC and the DLPFC. Follow-up analyses of *a priori* ROIs were conducted in regions noted above that achieved statistical significance in the group analyses. Mean percent signal change from baseline for each condition (Food Pre-meal, Food Post-meal, Nonfood Pre-meal, and Nonfood Post-meal) in the maximally activated voxel within each region was extracted and exported to Microsoft Excel for Mac 2008 (Microsoft Corporation).

TEARS data analysis. TEARS were analyzed as categorical variables with fMRI data. Reduction and Amplification subscale scores for all participants were ranked. Two groups were defined for each Reduction and Amplification, based on self-report scores that fell within the highest and lowest quartiles. High TEARS Reduction (HTR) relative to Low TEARS Reduction (LTR) scores reflect greater perceived ability to reduce emotion intensity or duration. Likewise, High TEARS Amplification (HTA) compared with Low TEARS Amplification (LTA) scores reflect greater perceived ability to increase or intensify emotion experience.

Results

Demographic and Behavioral Data

Independent samples *t*-tests determined that there were no gender differences for age, baseline BMI, TEARS Amplification, and CES-D. As a group, men reported higher scores on

the TEARS Reduction subscale than did women, indicating that men perceived themselves to be better at reducing emotions than did women. Table 1 contains a summary of the demographic information and questionnaire data, as well as comparisons between men and women.

TEARS subscales were correlated with other individual differences (see Table 2). Specifically, TEARS Amplification was significantly correlated with BMI, such that individuals with higher BMI also reported greater ability to amplify their emotions. TEARS Reduction was negatively correlated with symptoms of depression, as assessed by the CES-D, and sleep disruption, as assessed by the PSQI. Thus, participants who reported that they have difficulty reducing emotions also reported having more problems regulating mood and sleep quality. Importantly, neither of the TEARS subscale scores was significantly correlated with verbal, nonverbal, or general intelligence, as measured by the WASI, indicating that emotion regulation as measured by TEARS is a separate construct from both verbal and nonverbal intelligence.

As described in the Method section above and depicted in Figures 2 and 3, two groups for each subscale were created from the highest and lowest quartiles of TEARS Reduction and Amplification scores. High TEARS Reduction (HTR) was defined as Reduction scores greater than or equal to 27 (n = 9). Low TEARS Reduction (LTR) was defined as Reduction scores less than or equal to 19 (n = 11). As would be expected given the correlation data, the LTR group compared with the HTR group reported higher scores on the CES-D and PSQI. LTR and HTR groups did not differ in mean age, BMI, or IQ. Results of independent samples t-tests between LTR and HTR groups are reported in Table 3. Similarly, high TEARS Amplification (HTA) was defined as Amplification scores greater than or equal to 27 (n = 9). Low TEARS Amplification (LTA) was defined as Amplification scores less than or equal to 20 (n = 11). LTA and HTA groups did not differ in mean age or IQ, but they did differ significantly in BMI, with the mean

BMI being greater for the HTA than the LTA group. Results of independent samples *t*-tests between LTA and HTA groups are reported in Table 4.

fMRI Data

Whole group analysis

The first step was to determine whether there were between-condition changes in brain activation. To do this, three planned contrasts were analyzed: 1) Food versus Nonfood, Pre-meal versus Post-meal, 2) Pre-meal Food versus Nonfood, and 3) Post-meal Food versus Nonfood. These contrasts were designed to determine whether there were activation differences, respectively, between response to Food and Nonfood images at both Pre-meal and Post-meal, Food and Nonfood images at Pre-meal, and Food and Nonfood images at Post-meal. *A priori* and *post hoc* ROIs found to be significant are reported in Table 5.

Contrast 1: Food versus Nonfood, Pre-meal versus Post-meal. In the Image Type by Meal Condition interaction, participants displayed different BOLD response in three *a priori* regions, the right OFC (*x, y, z* = 30, 28, −8), the left OFC (*x, y, z* = −12, 56, −5), and left lateral PFC (*x, y, z* = −45, 44, −2). These relationships are represented in Figure 4. In all three of these clusters, there was a greater difference in activation between Food and Nonfood images at Post-meal compared with Pre-meal. Specifically, participants showed more activation to Nonfood images at Post-meal compared with Pre-meal.

Contrast 2: Pre-meal Food versus Nonfood. In the Pre-meal condition, participants displayed greater BOLD response to *Food* than to Nonfood images in the left posterior OFC (*x, y, z* =–24, 32, –2) and bilateral DLPFC corresponding to the middle frontal gyrus and Brodmann Area (BA) 46 (42, 32, 22; –45, 29, 22). Participants displayed greater BOLD response to *Nonfood* than to Food images in the right MPFC (*x, y, z* = 9, 41, 34), right dorsal ACC (*x, y, z* =

3, 44, 1), and bilateral DLPFC corresponding to the superior frontal gyrus and BA 9 (*x, y, z* = 36, 47, 31; –30, 35, 31).

Contrast 3: Post-meal Food versus Nonfood. In the Post-meal condition, none of the *a priori* regions showed greater activation to Food than to Nonfood images. However, participants did display greater BOLD response to *Nonfood* than to Food images in several of the *a priori* regions, including bilateral OFC (*x, y, z* = = –15, 23, –8; –9, 56, –5; 3, 56, 7), bilateral DLPFC (*x, y, z* = –36, 47, 7; 33, 44, 28; –33, 38, 31; 45, 20, 25), and bilateral ACC (*x, y, z* = –15, 38, 10; –3, 38, –5; 15, 38, 13).

Summary. In the OFC bilaterally and in the left lateral PFC, there was a greater difference in activation between images types at Post-meal (Nonfood greater than Food) than at Pre-meal. A cluster in DLPFC BA 46 as well as the cluster in the left OFC showed more activation Food versus Nonfood images at Pre-meal, but more activation to Nonfood versus Food images at Post-meal. In addition, viewing Food (versus Nonfood) images during the Pre-meal condition elicited greater activation in the left posterior OFC and bilateral DLPFC (middle frontal gyrus). In contrast, viewing Nonfood (versus Food) images at Pre-meal elicited greater activation in the right MPFC, right dorsal ACC, and bilateral DLPFC (superior frontal gyrus). At Post-meal, viewing Nonfood (versus Food) images was associated with greater activation in bilateral MPFC, left OFC, bilateral DLPFC, and bilateral ACC.

TEARS and fMRI Data

To test the hypothesis that emotion regulation skills are associated with food-related brain activation, TEARS Reduction and TEARS Amplification were used as classification variables. As described in the Method and Results sections above, two groups for each subscale were defined from the highest and lowest quartiles of Reduction and Amplification scores. High

reducers (HTR) reported greater perceived ability to reduce emotional arousal compared with low reducers (LTR). Likewise, high amplifiers (HTA) reported greater perceived ability to amplify emotional arousal compared with low amplifiers (LTA). The same series of contrasts were run for both HTR versus LTR and HTA versus LTA analyses.

Reduction. In order to test for group differences between LTR and HTR, Meal State (Pre, Post) by Image Type (Food, Nonfood) by TEARS Reduction Group (LTR, HTR) GLM with random effects was run. Three contrasts were examined: 1) Image Type (Food versus Nonfood) by Meal State (Pre-meal versus Post-meal) by TEARS Reduction (LTR versus HTR), 2) Pre-meal Food versus Nonfood for LTR versus HTR, and 3) Post-meal Food versus Nonfood for LTR versus HTR. *A priori* and *post hoc* ROIs found to be significant are reported in Table 6.

Contrast 1: Food versus Nonfood, Pre versus Post, LTR versus HTR. For the three-way interaction among Image Type, Meal State, and Group, there were clusters of activation in the *a priori* regions of left DLPFC (middle frontal gyrus, BA 9; *x, y, z* = –36, 20, 37), and bilateral OFC (x, y, z = –20, 14, –11; 12, 41, –5). These relationships are depicted in Figure 5. In the left DLPFC, the LTR group showed a greater difference in activation between image types at Pre-meal, whereas the HTR group showed a greater difference at Post-meal. Specifically, LTR was associated with greater activation to Nonfood than Food images at *Pre*-meal, and no difference in activation between image types at Post-meal. Conversely, HTR was associated with less activation to Food images and greater activation to Nonfood images at *Post*-meal, with no difference in activation between image types at Pre-meal.

In the left OFC, LTR was associated with less activation to Food than Nonfood images at *Pre*-meal, whereas HTR was associated with less activation to Food than Nonfood images at *Post*-meal. There was no difference in activation between image types for LTR at Post-meal and

HTR at Pre-meal. In the right OFC, neither LTR nor HTR was associated with a significant difference in activation between image types at Pre-meal. Additionally, the LTR group did not show a change in activation to either image type from Pre-meal to Post-meal. In contrast, the HTR group showed less activation to Food than Nonfood images at Post-meal.

Contrast 2: Pre-meal Food versus Nonfood, LTR versus HTR. For the Pre-meal Group by Image Type interaction, there was a cluster of activation in *a priori* regions of the left DLPFC (middle frontal gyrus, BA 9; *x, y, z* = –33, 17, 37; inferior frontal gyrus, BA 46; *x, y, z* = –33, 20, 22) and bilateral OFC (*x, y, z* = –20, 14, –11; 21, 11, –11). These relationships are depicted in Figure 6. In the middle frontal gyrus, the LTR group showed a greater difference in activation between Food and Nonfood images than did the HTR group. Specifically, the LTR group showed less activation to Food and more activation to Nonfood images. In the inferior frontal gyrus, the LTR group showed more activation to Food compared with Nonfood images. On the contrary, in both clusters within the left DLPFC, the HTR group showed no difference from baseline in activation to Food or Nonfood images, and no response difference between image types. In the OFC bilaterally, the LTR group showed less activation to Food than Nonfood images. Critically, the HTR group showed greater activation to Food than Nonfood images in the *right* OFC and no difference in activation between images types in the *left* OFC.

Contrast 3: Post-meal Food versus Nonfood, LTR versus HTR. For the Post-meal Group by Image Type interaction, there were clusters of activation in the right DLPFC (middle frontal gyrus, BA 9; *x, y, z* = 27, 23, 35; superior frontal gyrus, BA 9; *x, y, z* = 31, 41, 28) and right OFC (posterior orbital gyrus, BA 11; *x, y, z* = 24, 32, –11). These are shown in Figure 7. In both DLPFC clusters and in the right OFC, the HTR group, compared with the LTR group, showed greater difference in activation between images types, with a less activation to Food than

Nonfood images. In contrast, the LTR group did not show a difference in activation between Food and Nonfood images.

Summary. In a comparison of Image Type, Meal State, and Group, the left DLPFC and bilateral OFC showed differences in response patterns between LTR and HTR groups. Within the left DLPFC, LTR was associated with less activation to Food than Nonfood at Pre-meal, whereas the HTR group showed this difference at Post-meal. The left OFC showed a group difference for processing Food images at Pre-meal, and the right OFC showed a group difference for processing Nonfood images at Post-meal. For both differences, LTR was associated with less activation to Food than Nonfood images. The two-way interactions also revealed between-group differences in the left DLPFC and bilateral OFC at Pre-meal, as well as in the right DLPFC and right OFC at Post-meal. In all two-way interactions, the LTR group showed greater differences in activation than did the HTR group.

Amplification. In order to test for group differences between LTA and HTA, Image Type (Food, Nonfood) by Meal State (Pre, Post) by TEARS Amplification Group (LTA, HTA) GLM with random effects was run. As with Reduction, three contrasts were examined: 1) Image Type (Food versus Nonfood) by Meal State (Pre versus Post) by TEARS Amplification (LTA versus HTA), 2) Pre-meal Food versus Nonfood for LTA versus HTA, and 3) Post-meal Food versus Nonfood for LTA versus HTA. *A priori* and *post hoc* ROIs found to be significant are reported in Table 7.

Contrast 1: Pre-meal versus Post-meal, Food versus Nonfood, LTA versus HTA. For the three-way interaction among Image Type, Meal Condition, and Group, there was a cluster of activation in the left subgenual ACC (sgACC, BA 25; *x, y, z* = –9, 8, –11) and the right MPFC (superior frontal gyrus, BA 8; *x, y, z* = 9, 44, 37). As shown in Figure 8, in the left

sgACC, the LTA group showed greater activation to Food versus Nonfood images before eating and decreased activation to both Food and Nonfood images after eating. The HTA group showed no differences in activation to image type or meal state. In the right MPFC, there was a larger group difference in activation (Food versus Nonfood) at Post-meal compared with Pre-meal. After eating, HTA showed greater response to Food and lower response to Nonfood compared with the LTA group.

Contrast 2: Pre-meal Food versus Nonfood, LTA versus HTA. For the Pre-meal Group by Image Type interaction, there was a cluster of activation in the *a priori* region of left OFC (gyrus rectus, BA 10/11; *x, y, z* = –9, 42, –8). As shown in Figure 9, there was a group difference in response to Nonfood such that the LTA group showed less activation and the HTA group showed more activation to Nonfood images. There was no group difference in response to Food images.

Contrast 3: Post-meal Food versus Nonfood, LTA versus HTA. For the Post-meal Group by Image Type interaction, there were clusters of activation in the right and left lateral PFC (right inferior frontal gyrus, opercular part, BA 9; *x, y, z* = 45, 8, 22; right middle frontal gyrus, BA 46; *x, y, z* = 42, 18, 22; left middle/inferior frontal gyrus, BA 46; *x, y, z* = –39, 35, 13; left inferior frontal gyrus, BA 46; *x, y, z* = –45, 32, 4), left dorsal ACC (BA 32; *x, y, z* = –12, 17, 40), and left OFC (BA 10/11; –3, 35, –8). As shown in Figure 10, in both the right and the left lateral PFC, LTA and HTA groups responded with opposite patterns of activation to images and meal states. The LTA group showed more activation to Food than Nonfood images, whereas the HTA group showed more activation to Nonfood compared with Food images. In the left dorsal ACC, HTA and LTA groups showed similar amounts of activation to Food images. However, the LTA group showed less and the HTA group showed more activation to Nonfood images

compared with Food images. In the left OFC, the HTA group showed a greater difference in activation to Food versus Nonfood images than did the LTA group. Specifically the HTA group showed greater activation to Nonfood images than did the LTA group.

Summary. In a comparison of Image Type, Meal State, and Group, the left sgACC showed a group difference in response to both image type and meal state, such that the LTA group showed increased activation to Food images before eating and decreased activation to both Food and Nonfood images after eating. In the right MPFC, the group difference was greater at Post-meal than at Pre-meal, such that HTA was associated with greater response to Food and lower response to Nonfood compared with LTA. In two-way interactions, there was between-group differential responding in the left OFC at Pre-meal, and in the left dorsal ACC, left and right lateral PFC, and left OFC at Post-meal. Specifically, there was a greater difference between LTA and HTA in response to Nonfood than Food images in the two-way interactions.

Discussion

The aim of the present study was to investigate the relationship between emotion regulation and brain response to food images in a group of obese participants. Specifically, this study was intended to examine the extent to which self-reported emotion amplification and reduction ability is related to brain activation patterns to Food and Nonfood images in both Pre-meal (hungry) and Post-meal (after eating a small ~500 kcal meal) conditions. Results indicate that differences in emotion reduction are associated with differing patterns of activation in the OFC, DLPFC, and lateral PFC bilaterally. Findings also indicate that differences in emotion amplification are associated with differing patterns of activation in left OFC, left dorsal ACC, left sgACC, right MPFC, and lateral PFC bilaterally.

Whole-group analysis

In the whole-group analysis for the present study, when participants were hungry, they did indeed show greater activation in the left OFC to *Food* compared with Nonfood images. Additionally, participants showed greater right OFC activation to Food images when they were hungry compared to when they were full. The finding in the present study of OFC involvement in food motivation is consistent with other research findings (e.g., Goldstone et al., 2009) and provides converging support for the role of the OFC in hedonic-driven eating behavior.

Contrary to hypotheses, in the lateral PFC and DLPFC, participants showed more activation to Food images before eating than after eating. It should be noted that the participants in this study were all seeking a structured diet intervention; that is, they were motivated to change eating behavior and lose weight. Lateral regions of the PFC, including the DLPFC, have been associated with cognitive control processes, particularly those that are effortful (Aron, Robbins, & Poldrack, 2004; Hedden & Gabrieli, 2010). These unanticipated findings may reflect participants' efforts to control the salience and rewarding nature of Food images, which is most intense in the Pre-meal state.

Emotion reduction

Differences in emotion reduction were associated with differences in activation of the lateral PFC, the DLPFC, and the OFC. At a behavioral level, it was expected that Post-meal activation patterns of high reducers would suggest context-appropriate suppression of reward response to food pictures and enhanced recruitment of inhibitory control regions after eating. It was also anticipated that low reducers would fail to show this context-appropriate modulation of reward and inhibitory PFC regions. It was therefore unexpected that low reducers would show more hunger-related activation in the right and left lateral PFC in response to Food images.

Whereas high reducers showed the expected increase in right lateral PFC activation to Food images after they had eaten, they did not appear to recruit the left lateral PFC in this process (i.e., there was no difference from Pre-meal to Post-meal when viewing Food images).

Thus, a critical and unanticipated difference between the two groups is the finding that low reducers appear to show differences in hunger and fullness in both left and right lateral PFC, but high reducers may process this difference primarily in the right lateral PFC. It is possible that individual differences in emotion regulatory ability are associated with recruitment of different neural networks in response to food, hunger, and satiety related cues. An additional group difference is the opposite pattern of right lateral PFC activation seen in high versus low reducers: whereas high reducers showed recruitment of this region after eating, low reducers showed contextually inappropriate engagement of this region when hungry and decreased activation when full. One explanation is that for low reducers, this represents effortful suppression of the rewarding properties of highly appetizing food prior to being presented with actual food and disinhibited response to food-related reward after exposure to food (i.e., eating).

Low reducers showed greater difference (Food vs Nonfood) in the *left* DLPFC *before* eating, whereas high reducers showed greater difference in the *right* DLPFC *after* eating. This finding suggests that low reducers attempt to exert inhibitory control over food-related stimuli prior to eating, whereas high reducers exert this control after eating. This is especially critical given the tendency for self-regulatory strength to become depleted when demand for self-regulation is high (Baumeister & Heatherton, 1996; Muraven, Tice, & Baumeister, 1998; Vohs & Heatherton, 2000). Engagement of the DLPFC by low reducers prior to eating may actually result in subsequent disinhibition, or overeating, in the absence of adequate self-regulatory resources.

There were differing patterns of response to food motivation conditions in the OFC bilaterally. Low reducers showed a marked decrease in signal, whereas high reducers showed mildly increased signal in the OFC, in response to Food images when they were hungry. Post-meal, in a state of at least partial fullness, only the right OFC showed a group difference in activation. Whereas low reducers showed increased signal to both Food and Nonfood, high reducers showed decreased right OFC signal to Food and increased signal to Nonfood. Again, this set of findings suggests that high reducers appropriately activated reward centers of the brain when they were hungry and downregulated these centers after eating. In contrast, low reducers appeared to exhibit a contextually inappropriate pattern of response. Specifically, food images failed to activate a primary reward center when low reducers were hungry, but once they had been fed, the OFC reward center was hyperresponsive to food images. It may be that for low reducers, consuming food primes or disinhibits reactions to subsequent exposure to food. In other words, emotion reduction ability may be less related to how people respond to hunger cues than it is the ability to "turn off" the reward center once eating has begun.

Thus, individuals who reported being effective at decreasing emotional arousal (high reducers) also displayed contextually appropriate responses to changing conditions in food motivation in inhibitory brain regions. In contrast, individuals who reported difficulty with emotion reduction (low reducers) displayed a pattern of context-inappropriate response to food and hunger cues. Specifically, they recruited inhibitory resources at a time when it is "acceptable" to fail to inhibit approach behavior toward food. This neural and behavioral tendency might predispose low emotion reducers to subsequent disinhibited eating behavior when food motivation would expect to have declined (e.g., after eating). Coupled with the finding that low reducers endorsed more sleep difficulties as well as more symptoms of

depression on questionnaires, it can be inferred that low emotion reduction is associated with general dysregulation of eating, sleeping, and engaging in mood reparative behavior.

Emotion amplification

Differences in emotion amplification were associated with differences in activation of the lateral PFC, as well as OFC, ACC, and MPFC. It was expected that high amplifiers would display more food-related activation in lateral PFC regions. Data were at least partially consistent with this hypothesis. High amplifiers did show greater activation to Food images in left lateral PFC before compared with after eating, a pattern that was reversed after eating. In Post-meal comparisons of both Food and Nonfood images, low amplifiers showed greater lateral PFC activation to Food compared with Nonfood images. It is possible that these differences are representative of differences in cognitive processing of affective stimuli. Future research should be directed toward clarifying the significance of this finding.

It was not hypothesized that there would be differences in OFC activation between low and high amplifiers. In the present study, however, low and high amplifiers showed different patterns of activation to visual stimuli both before and after eating in the left OFC. During both meal states, left OFC response to Food images did not differ between groups. On the contrary, the primary between-group difference in activation was observed in response to Nonfood (animal) images. This set of findings may indicate that emotion amplification ability is related to Nonfood-related reward processing in the left OFC, such that high amplifiers are more responsive to these and other general rewards than are low amplifiers. If emotion amplification is related to the ability to become engaged or energized by environmental stimuli, then it is not surprising that high amplifiers would activate a reward processing center while viewing images of animals that had been chosen because they were highly interesting.

It was hypothesized that differences in emotion amplification would be associated with activation differences in the dorsomedial PFC (DMPFC). Though not specifically in the DMPFC, there was a pattern of differential responding in the right MPFC. Importantly, high amplifiers compared with low amplifiers showed greater activation to Food images after eating. This finding is similar to that of Martin et al. (2010), who found that obese compared with healthy weight individuals activated the MPFC more (Food versus Nonfood) at both Pre-meal and Post-meal. However, Martin and colleagues found left MPFC activation, whereas the present study found this difference in the right MPFC. Therefore, emotion amplification may be related to right but not left MPFC response to food motivation.

It was not hypothesized that the ACC would show activation differences related to emotion amplification. However, at Post-meal, the left dorsal ACC showed a greater between-group difference in activation to Nonfood compared with Food images, with high amplifiers showing more activation than low amplifiers to the Nonfood images. The dorsal ACC is involved in encoding the reward value of stimuli and modulating attention (Bush et al., 2002). The present finding may have a similar explanation to that of the left OFC: High amplifiers likely experienced the animal images as highly interesting and rewarding.

Despite the fact that no hypotheses were made with regard to the ACC and emotion amplification, it is especially striking that the subgenual (sgACC) was a region in which low and high amplifiers responded differently to food motivation conditions. Abnormalities in sgACC activity have been shown to occur in mood disorders (see Drevets & Savitz, 2008 for a review), and activation differences have also been associated with differential response to both antidepressant pharmacotherapy (Keedwell et al., 2010) and cognitive-behavioral therapy (Siegle et al., 2006) treatment of depression. Furthermore, the sgACC may play a role in reward-related

decision-making (Elliott, Friston, & Dolan, 2000). Elliott and colleagues found that the sgACC was activated in response to high-reward winning streaks during a gambling task, and they suggested that the sgACC was responding to increased reward expectation as well as motivation to continue reward-seeking behavior. Similarly, Rogers and colleagues found that individuals performing a decision-making task showed the most activation in the sgACC in response to high reward. It is possible that low amplifiers encoded high reward value in the sgACC while viewing Food images in a hunger state, whereas high amplifiers encoded high reward value in the sgACC while viewing Food images *after* they had already eaten. This finding suggests food-related disinhibition and enhanced food-seeking behavior, which is consistent with the higher BMI observed among high amplifiers.

It was assumed a priori that high emotion amplification is desirable, as it must represent better emotion regulatory ability. However, results from this study showing that emotion amplification is positively correlated with baseline BMI, coupled with differences in brain activation patterns to food motivation levels between high and low amplifiers, suggest that high amplification may be associated with unhealthy coping strategies such as chronic disinhibited eating, or even binge eating, in response to the hedonic properties of food. It is possible that high amplifiers use food as an energy source for enhancing emotional arousal. Indeed, Hamilton et al. (2009) suggested that individuals may use emotion amplification techniques to combat fatigue or transient mild negative affect.

Limitations and Future Directions

Several limitations related to this study should be acknowledged. One significant limitation to this study was the fact that all participants were obese. Thus, there was no healthy weight comparison group, making it difficult to determine whether group differences were truly

due to emotion regulation ability or were an artifact of using only obese participants. On one hand, the lack of a "lower end" of BMI values may have limited the ability to detect results. On the other hand, within the "obese" category, there was a wide range of BMI measurements, from roughly 30 to over 46. The World Health Organization (2000, p. 9) defines three classes of obesity based on health risk, such that BMI between 30 and 34.9 is considered Class I, BMI 35-39.9 is considered Class II, and BMI 40 or greater is considered Class III. Individuals who fall in Class III obesity are considered to be morbidly obese and have a "very severe" risk of comorbid medical conditions such as hyperlipidemia, hypertension, and diabetes. However, in the present study, this issue was anticipated, and all participants reported the absence of these and other common comorbid conditions. Furthermore, no participants were taking medication for any of these conditions.

While not a limitation, it is important to emphasize that participants in this study comprised obese individuals seeking a diet intervention. Preliminary data analyses (Martin, unpublished data), suggest that on behavioral measures of impulsivity and decision-making, this group of participants differs from a group of obese individuals not seeking a diet. Although it is unclear to what extent this results in brain activation differences pre-diet, the engagement of inhibitory brain regions during viewing of Food images may reflect ambivalent feelings and behavior toward highly appetizing food.

A second limitation was in data analysis procedures. Thresholding criteria of $p<.001$ uncorrected for multiple comparisons, with a minimum cluster size of three contiguous voxels, which were the criteria used in the present study, may inflate Type I error. This is because fMRI data analysis involves statistical tests run on thousands of single voxels simultaneously, leaving open the possibility of up to 100 voxels (in a 100,000 voxel image set) that are in fact false

positives (Lieberman & Cunningham, 2009). Therefore, it has been proposed that researchers use the False Discovery Rate (FDR) of $p < .05$, which includes a stringent correction for multiple comparisons (Genovese, Lazar, & Nichols, 2002). Lieberman and Cunningham (2009) argued that FDR controls Type I error at the expense of inflating Type II error, and they proposed a standard of $p < .005$ with a minimum cluster size of 10 contiguous voxels, for use when analyzing cognitive and affective neuroimaging data. However, the thresholding criteria used in the present study have been acceptable in the past, and have been used in other recent analyses (e.g., Bruce et al., 2010; Martin et al., 2010). Additionally, a cluster size threshold of 10 voxels is likely to preclude the detection of activations in smaller structures. Given the novel and exploratory nature of the investigation, a more lenient threshold seems acceptable for the current analyses. In future analyses, careful consideration should be made to determine which criteria would be most appropriate to use.

The present study included a relatively small sample size after TEARS groups were created (i.e., 9 and 11 individuals per group). In addition to this potential limitation, an additional consideration related to data analysis was the lack of statistical comparison between male and female participants. The low number of men in the study would have yielded underpowered results. Gender differences in brain response to food stimuli have been noted in previous research studies (e.g., Smeets et al., 2006; Wang et al., 2009). From these studies, it may be concluded that men and women process food cues differently, and collapsing males and females into statistical comparisons might limit or bias results of fMRI studies. Future analyses with this dataset should 1) use a larger sample size, 2) include an analysis of females only, and 3) compare activation patterns between males and females.

Conclusion

The results of the present study provide preliminary evidence that differences in emotion regulatory ability are associated with differences in brain activation to images of appetizing food, both when individuals have fasted for several hours and immediately after they have eaten. Group differences between high and low reducers, as well as between high and low amplifiers, emerged in brain regions such as the DLPFC that are known to be involved in self-regulation, as well as in regions such as the OFC and the sgACC that are known to play a role in processing hedonic properties of food and other environmental stimuli. It appears that individual differences in the ability to change the trajectory of felt emotions are related to the ability to "turn up" inhibitory brain mechanisms and "turn down" the reward processing brain regions. These factors likely contribute to overeating and to obesity.

Several questions arise from the present results. First, at a neural level, do changes in activation occur automatically, or as a function of effortful emotion or cognitive regulation? Second, it is important from a clinical perspective to clarify how emotion regulation occurs. For example, food may be the energy source for high amplifiers. If high amplifiers are indeed successful at amplifying emotion, and if they are using food to do so, then it would be useful to investigate other coping strategies and energy sources. Future research should be directed toward answering these and other questions in order to ultimately clarify the role of emotion regulation in eating disorders such as binge eating, as well as in weight loss and weight loss maintenance.

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Table 1

Gender Differences by Participant Characteristic

Characteristic	Number					
	All (n = 35)	Males (n = 7)	Females (n = 28)	χ^2	df	p
Race/Ethnicity						
White, Not Hispanic	21	5	16	1.342	2	.511
Hispanic or Not White	11	1	10			
Unspecified	3	1	2			
	Mean (SD)					
	All (n = 28)	Males (n = 7)	Females (n = 28)	t	df	p
Age	38.83 (8.59)	36.57 (8.14)	39.39 (8.75)	−.773	33	.445
BMI (kg/m^2)	36.09 (4.20)	35.93 (3.29)	36.13 (4.45)	−.113	33	.911
TEARS						
Amplification	22.69 (4.44)	23.43 (3.78)	22.50 (4.64)	.489	33	.628
Reduction	23.00 (4.79)	27.71 (3.20)	21.82 (4.52)	3.234	33	.003
CES-D	7.19 (5.67)	4.43 (3.87)	7.88 (5.89)	−1.463	33	.153
PSQI	10.46 (5.73)	7.29 (4.61)	11.25 (5.77)	−1.682	33	.102
WASI						
Vocabulary		61.86 (5.11)	57.96 (7.47)	1.298	33	.203
Matrix Reasoning		58.00 (6.35)	58.89 (5.95)	−.351	33	.728
Full Scale		117.43 (8.72)	114.82 (10.47)	.607	33	.548

Note. df = degrees of freedom; SD = standard deviation; BMI = body mass index; TEARS = The Emotion Amplification and Reduction Scales; CES-D = Center for Epidemiologic Studies Depression Scale; PSQI = Pittsburgh Sleep Quality Inventory; WASI = Wechsler Abbreviated Scale of Intelligence

Table 2

Correlation Matrix for Behavioral Data

Measure	1	2	3	4	5	6	7	8	9
1. Age	–	−.049	−.190	.005	−.049	.310	.065	.089	.086
2. BMI	−.049	–	.510*	.159	−.032	−.236	.035	.075	.066
3. TEARS-AMP	−.190	.510*	–	.266	.169	−.120	.165	.097	.158
4. TEARS-RED	.005	.159	.266	–	−.392*	−.468*	−.034	−.097	−.013
5. CES-D	−.049	−.032	.169	−.392*	–	.547*	.016	−.044	−.013
6. PSQI	.310	−.236	−.120	−.468*	.547*	–	.012	−.220	−.113
7. VOCAB	.065	.035	.165	−.034	.016	.012	–	.442*	.875*
8. MATREA	.089	.075	.097	−.097	−.044	−.220	.442*	–	.820*
9. WASI	.086	.066	.158	−.013	−.013	−.113	.875*	.820*	–

Note. Correlation matrix for behavioral data. BMI = body mass index (kg/m^2); TEARS-AMP = TEARS Amplification subscale; TEARS-RED = TEARS Reduction subscale; CES-D = Center for Epidemiologic Studies Depression Scale; PSQI = Pittsburgh Sleep Quality Inventory; VOCAB = Vocabulary subtest of WASI; MATREA = Matrix Reasoning subtest of WASI; WASI = Wechsler Abbreviated Scale of Intelligence. *$p < .05$ (2-tailed).

Table 3

Behavioral Data Group Differences Between Low TEARS Reduction (LTR) and High TEARS Reduction (HTR) Groups

	Mean (SD)				
	LTR (n = 11)	HTR (n = 9)	t	df	p
Age	38.00 (10.72)	36.44 (6.77)	.377	18	.711
BMI	35.19 (4.92)	36.35 (3.74)	–.581	18	.569
TEARS					
Amplification	22.36 (3.91)	24.67 (3.12)	–1.426	18	.171
Reduction	17.64 (1.50)	29.44 (2.46)	–12.625	12.69	<.001
CES-D	10.73 (6.56)	4.33 (3.00)	2.693	18	.015
PSQI	14.36 (6.71)	7.11 (4.23)	2.810	18	.012
WASI					
Vocabulary	60.82 (6.24)	59.89 (4.89)	.364	18	.720
Matrix Reasoning	59.18 (5.14)	58.11 (6.62)	.408	18	.688
Full Scale	117.64 (8.36)	115.67 (10.22)	.475	18	.641

Note. LTR = Low TEARS Reduction; HTR = High TEARS Reduction

Table 4

Behavioral Data Group Differences Between Low TEARS Amplification (LTA) and High TEARS Amplification (HTA) Groups

	Mean (SD)				
	LTA $n = 11$	HTA $n = 9$	t	df	p
Age	41.00 (11.13)	36.89 (5.80)	1.062	15.59	.304
BMI	34.28 (2.58)	39.29 (4.71)	–2.857	11.82	.015
TEARS					
Amplification	17.64 (1.86)	28.44 (1.51)	–14.044	18	<.001
Reduction	22.64 (3.56)	23.78 (5.56)	–.557	18	.584
CES-D	5.73 (5.62)	10.06 (7.25)	–1.506	18	.150
PSQI	11.18 (6.94)	10.00 (3.43)	.496	15.17	.627
WASI					
Vocabulary	58.91 (6.38)	62.00 (6.29)	–1.085	18	.292
Matrix Reasoning	58.82 (7.17)	62.00 (3.74)	–1.275	17.27	.221
Full Scale	115.64 (10.62)	121.33 (6.93)	–1.383	18	.184

Note. LTA = Low TEARS Amplification; HTA = High TEARS Amplification

Table 5

Regions Reaching Significance During GLM Contrasts of Whole Group Data

Region and contrast	L/R	BA	Talairach Coordinates X	Y	Z	Voxels	*t*
Food vs Nonfood, Pre-meal vs Post-meal							
A priori							
Posterior orbital gyrus	R	47	30	28	−8	5	4.26
Medial frontal gyrus	L	10	−12	56	−5	4	4.06
Middle frontal gyrus	L	10	−45	44	−2	27	4.91
Post hoc							
Precentral gyrus	L	4	−39	−16	37	21	4.43
Superior temporal gyrus	L	22	−57	−7	7	8	4.44
		21	−57	5	−8	12	−4.57
Fusiform gyrus	L	37	−33	−55	−11	35	5.19
Paracentral lobule	L	3	−21	−25	52	19	5.00
Pre-meal Food > Nonfood							
A priori							
Medial frontal gyrus	R	8	9	41	34	7	−3.82
Posterior orbital gyrus	L	–	−24	32	−2	130	4.37
Superior frontal gyrus	R	9	36	47	31	19	−3.80
	L	9	−30	35	31	5	−4.18
Middle frontal gyrus	R	46	42	32	22	41	4.40
	L	46	−45	29	22	22	3.98
Anterior cingulate cortex	R	32	3	44	1	5	−3.80
Post hoc							
Claustrum	R	–	36	−4	7	936	6.38
	L	–	−36	−1	−2	1160	6.84
Thalamus	L	–	−9	−13	7	58	4.56
		–	−24	−22	−2	96	4.96

Hippocampus	L	–	–27	–25	–8	14	3.56
Parahippocampal gyrus	R	36	39	–34	–20	1088	–5.86
	L	36	–39	–31	–20	254	–5.22
Medial frontal gyrus	R	10	6	62	7	6	–3.64
Inferior frontal gyrus	L	9	–57	5	25	118	4.03
Middle frontal gyrus	R	46	42	32	22	40	4.40
		10	33	50	1	12	–4.39
			27	59	13	42	–4.87
Superior frontal gyrus	R	10	18	62	19	24	–3.91
	L	10	–18	53	13	8	–3.81
			–27	50	25	29	–4.42
		8	–33	–19	64	6	3.86
Anterior cingulate	L	32	–12	32	16	7	3.91
Posterior cingulate	R	30	15	–58	16	44	–4.44
		23	12	–52	13	9	–3.56
Precentral gyrus	R	6	24	–13	49	6	3.70
	L	6	–33	–19	64	8	3.62
			–39	–1	28	6	3.91
			–54	–4	34	85	4.22
Postcentral gyrus	R	2	54	–25	37	99	4.39
		40	51	–31	49	6	3.70
	L	2	–60	–19	25	30	4.03
			–63	–19	22	12	3.95
		3	–57	–22	40	48	4.64
Middle temporal gyrus	R	21	60	–34	–8	16	–3.82
	L	39	–48	–64	13	3579	–7.28
Supramarginal gyrus	L	40	–42	–40	37	39	3.57
Middle occipital gyrus	R	37	48	–67	–5	10385	–7.45
Fusiform gyrus	L	37	–42	–52	–5	14	4.04
			–51	–46	–14	446	5.41
		19	–45	–73	–11	453	–5.55

Superior parietal lobule	R	7	15	–64	58	19	4.44
Inferior parietal lobule	R	40	48	–34	46	98	4.63
	L	40	–33	–43	37	38	4.02
			–42	–49	52	100	4.20
			–57	–28	37	87	3.75
			–57	–31	28	31	3.70
Precuneus	R	31	3	–61	28	1724	–5.93
	R	7	3	–58	37	28	–3.78
	L	7	–21	–73	43	4068	6.51
Cuneus	R	19	27	–82	25	46	3.71
	L	19	–15	–85	31	25	4.47
		17	–18	–85	10	7	3.81
Cerebellum	R	–	9	–73	–8	39578	9.78
	L	–	–3	–64	1	18	4.01
Insula	L		–36	–1	–2	1161	6.84
	R		36	–4	7	936	6.38
Hippocampus	L	–	–27	–25	–8	14	3.56
Parahippocampal gyrus	L	36	–39	–31	–20	254	–5.22
	R	36	39	–34	–20	1088	–5.86
Thalamus	L	–	–24	–22	–2	96	4.96
		–	–9	–13	7	58	4.56
Cingulate	L	31	15	–58	16	44	–4.44
Superior frontal gyrus	L	8	–33	23	49	6	3.86
		10	–27	50	25	29	–4.42
	R	10	18	62	19	24	–3.91
Middle frontal gyrus	R	10	27	59	13	42	–4.87
Precentral gyrus	L/R	6	–54	–4	34	85	4.22
Superior temporal gyrus	R	38	48	–10	–11	36	–4.13
Middle temporal gyrus	L	39	–48	–64	13	3582	–7.28
	R	21	60	–34	–8	16	–3.82
Inferior temporal gyrus	L	37	–51	–46	–14	446	5.41

		19	–45	–73	–11	453	–5.55
Fusiform gyrus	L/R	37	39	–34	–20	1088	–5.86
Postcentral gyrus	L/R	2	–57	–22	40	48	4.64
Parietal operculum	L	40	–57	–28	37	87	3.75
Inferior parietal lobule	L	40	–42	–49	52	100	4.20
	R	40	48	–34	46	98	4.63
Superior parietal lobule	R	7	15	–64	58	19	4.44
Precuneus	L	7	–21	–73	43	4070	6.51
	R	18	3	–61	28	1725	–5.93
Cuneus	L	19	–15	–85	31	25	4.47
	R	19	27	–82	25	46	3.71
Lingual gyrus	R	18	9	–73	–8	39591	9.78
Middle occipital gyrus	R	19	48	–67	–5	10387	–7.45
Cerebellum	L		–18	–73	–26	23	3.88
Post-meal: Food > Nonfood							
A priori							
Middle frontal gyrus	R	46	45	20	25	12	–3.95
	L	46	–36	47	7	14	–3.93
Superior frontal gyrus	L	9	–33	38	31	18	–4.12
	R	9	33	44	28	8	–3.73
Medial frontal gyrus	L	10	–9	56	–5	190	–4.67
	R	10	3	56	7	51	–4.75
Medial orbital gyrus	L		–15	23	–8	33	–3.99
Anterior cingulate cortex	L	32	–15	38	10	3	–3.73
			–3	38	–5	46	–4.61
	R	32	15	38	13	15	–3.62
Post hoc							
Parahippocampal gyrus	R		27	–22	–20	6	4.02
Insula	L		–39	–7	7	346	5.30
	R		39	–4	4	375	6.17
Thalamus	R	–	18	–34	10	44	–4.65

Claustrum	R	–	36	–16	–2	34	–3.92
Caudate	R	–	15	23	7	6	–3.70
		–	9	20	4	62	–4.74
		–	6	11	–2	18	–3.91
Superior frontal gyrus	L/R	10	–27	53	–2	163	–5.05
Middle frontal gyrus	R	6	33	–4	52	151	–5.10
		8	24	14	40	35	–4.95
		10	33	47	22	120	–4.20
	L	6	–39	–1	49	153	–4.03
		10	–42	50	–2	138	–4.71
Precentral gyrus	L	6	–42	–4	31	189	5.46
Superior temporal gyrus	L/R	22	48	–7	–8	202	–6.33
Middle temporal gyrus	L/R	39	42	–64	19	14948	–7.96
Inferior temporal gyrus	R	20	51	–46	–14	11	3.77
Fusiform gyrus	L	37	–39	–52	–14	15	–4.07
Postcentral gyrus	L	3	–36	–31	52	11	–4.17
Precuneus	L	7	–12	–43	31	8	–4.04
		19	–18	–82	40	19	3.65
	R	7	24	–67	49	1565	6.58
Superior parietal lobule	L/R	7	12	–67	55	61	4.69
Supramarginal gyrus	R	40	57	–43	34	14	–4.22
Angular gyrus	L	39	–42	–55	34	62	–4.44
Inferior parietal lobule	L	40	–42	–37	40	82	5.08
	R	40	42	–52	37	56	–4.40
Cuneus	L/R	19	–24	–88	22	18	4.14
Cerebellum	L	–	–24	–46	–14	30591	14.08
Pons	R	–	6	–37	–41	8	–3.99

Note. L = left; R = right; L/R = both left and right (bilateral); BA = Brodmann's Area

Table 6

Regions of Significant Activation for Random Effects GLM Contrasts of Food versus Nonfood, Pre-meal versus Post-meal, and LTR versus HTR

Region	L/R/B	BA	Talairach Coordinates X	Y	Z	Voxels	*t*
Pre-Meal vs Post-Meal, Food vs Nonfood							
A priori							
Middle frontal gyrus	L	9	–36	20	37	14	–5.38
	R	9	51	38	1	4	–5.01
			51	17	28	16	–5.11
Straight gyrus	R	–	12	41	–5	7	–5.50
Post hoc							
Insula	R	–	24	20	–2	6	4.44
Uncus	L/R	28	21	8	–26	12	5.05
Globus pallidus	R	–	15	–1	4	6	–4.93
Thalamus	L	–	–12	–10	1	9	–4.75
Cerebellum	L	–	–27	–70	–23	13	4.60
Pre-Meal Food > Nonfood							
A priori							
Middle frontal gyrus	L	9	–33	17	37	3	–4.24
Inferior frontal gyrus, orbital part	L	47	–33	20	22	21	5.33
Post hoc							
Uncus	R	28	21	8	–25	8	4.34
Precuneus	L/R	7	–21	–67	46	37	5.17
Cerebellum	R	–	21	–46	–35	6	4.57
Post-Meal Food > Nonfood							
A priori							
Middle frontal gyrus	R	9	27	23	34	42	6.92
		11	25	32	–11	5	3.93

Post hoc							
Middle frontal gyrus	R	8	30	41	28	14	4.60
Fusiform gyrus	L	37	–30	–43	–11	14	4.72

Note. L = left; R = right; L/R = both left and right (bilateral); BA = Brodmann's Area

Table 7

Regions of Significant Activation for Random Effects GLM Contrasts of Food versus Nonfood, Pre-meal versus Post-meal, and LTA versus HTA

Region	L/R/B	BA	Coordinates			Voxels	*t*
			X	Y	Z		
Food versus Nonfood, Pre-meal versus Post-meal, LTA versus HTA							
A priori							
Subgenual anterior cingulate gyrus	L	25	–9	8	–11	6	5.04
Medial frontal gyrus	R	8/9	9	44	37	3	–4.88
Post hoc							
Amygdala	L	–	–27	–4	–14	6	–4.36
Parahippocampal gyrus	L	28	–24	–22	–8	6	–4.70
Cingulate gyrus	R	31	15	–31	43	22	–5.93
Precentral gyrus	L	4	–39	–16	56	12	–4.53
Superior temporal gyrus	L/R	22	–57	–16	1	18	–4.89
	L	42	–63	–31	19	21	–5.14
Postcentral gyrus	R	3	24	–28	68	16	–4.51
	L	2	–42	–25	37	45	–5.04
Paracentral gyrus	L/R	6	–9	–40	71	40	–5.75
Superior parietal lobule	L	7	–27	–49	62	199	–6.87
Pre-Meal Food > Nonfood							
A priori							
Gyrus rectus	L	10	–9	41	–8	4	3.94
Post hoc							
Globus pallidus	R	–	12	–1	1	7	5.75
Superior temporal gyrus	L	42	–63	–31	19	10	–4.08
Middle temporal gyrus	R	39	51	–58	23	14	4.91
Postcentral gyrus	L	2	–54	–25	44	6	–4.32
		40	–60	–25	19	15	–4.45

		5	–36	–46	59	7	–4.01
Cuneus	L	18	–21	–73	19	51	5.01
Cerebellum	R	–	27	–73	–14	37	5.95
	L	–	–24	–49	–38	6	5.07
Post-Meal Food > Nonfood							
A priori							
Inferior frontal gyrus	R	44	45	8	22	58	5.34
	L	46	–45	32	4	14	4.50
		46	–39	35	13	8	4.60
Dorsal anterior cingulate gyrus	L	32	–3	35	–8	5	4.71
			–12	17	40	3	4.66
Post hoc							
Superior frontal gyrus	R	6	27	5	58	12	4.79
		6	6	17	58	386	6.24
			–15	17	49	32	4.80
			–21	23	52	19	4.62
Precentral gyrus	L	4	–42	–7	49	9	5.09
Superior temporal gyrus	R	22	39	–40	19	128	7.00
	L	22	–57	–19	1	82	6.71
Middle temporal gyrus	R	21	52	–52	7	9	4.71
Angular gyrus	R	39	39	–52	19	17	4.81
Precuneus	R	7	9	–37	46	11	4.65
	L	7	–15	–49	68	20	5.28
	L	7	–9	–70	7	10	5.34
Cerebellum		–	–9	–43	–41	14	4.83

Note. *L = left; R = right; L/R = both left and right (bilateral); BA = Brodmann's Area*

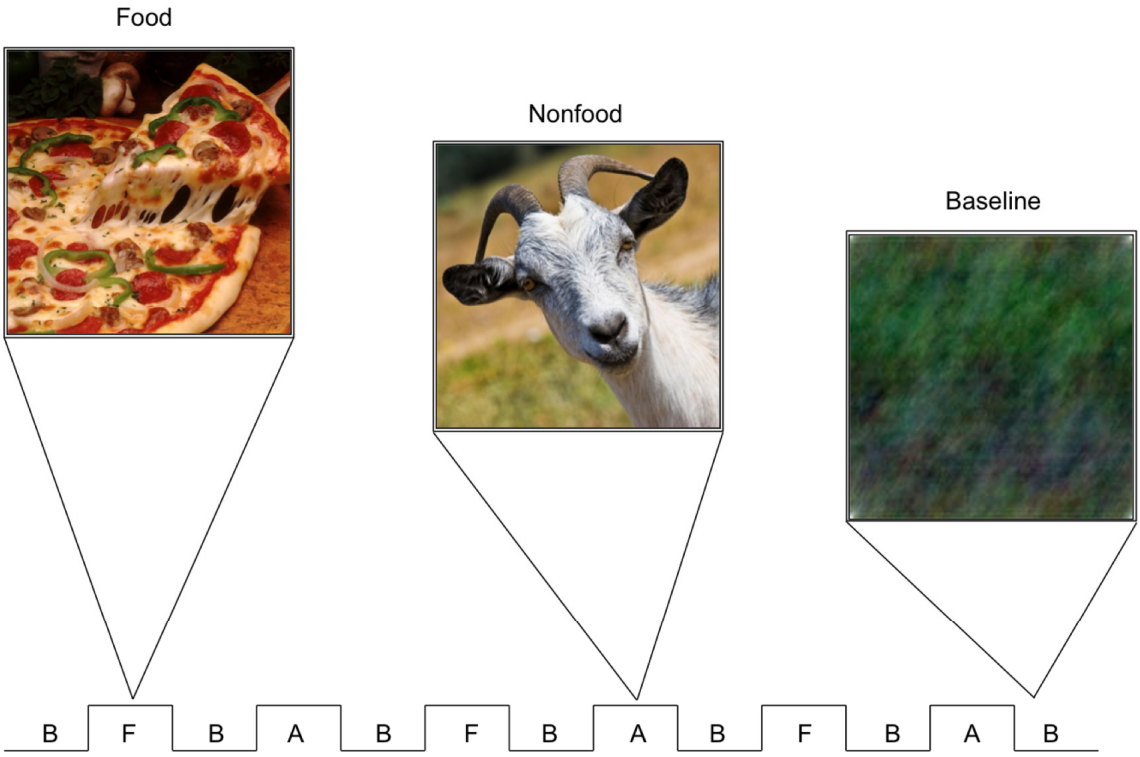

Figure 1. Food motivation paradigm: Participants viewed pictures of Food (F) and animals (A; Nonfood), as well as blurry images (B) created from the food and animal images. This was a block design, with 13 blocks containing 10 images each. Food and animal images alternated with blurry images. Order was counterbalanced so that some scanning sessions began with blurry and food images, and other sessions began with blurry and animal images. Participants were shown each image only one time.

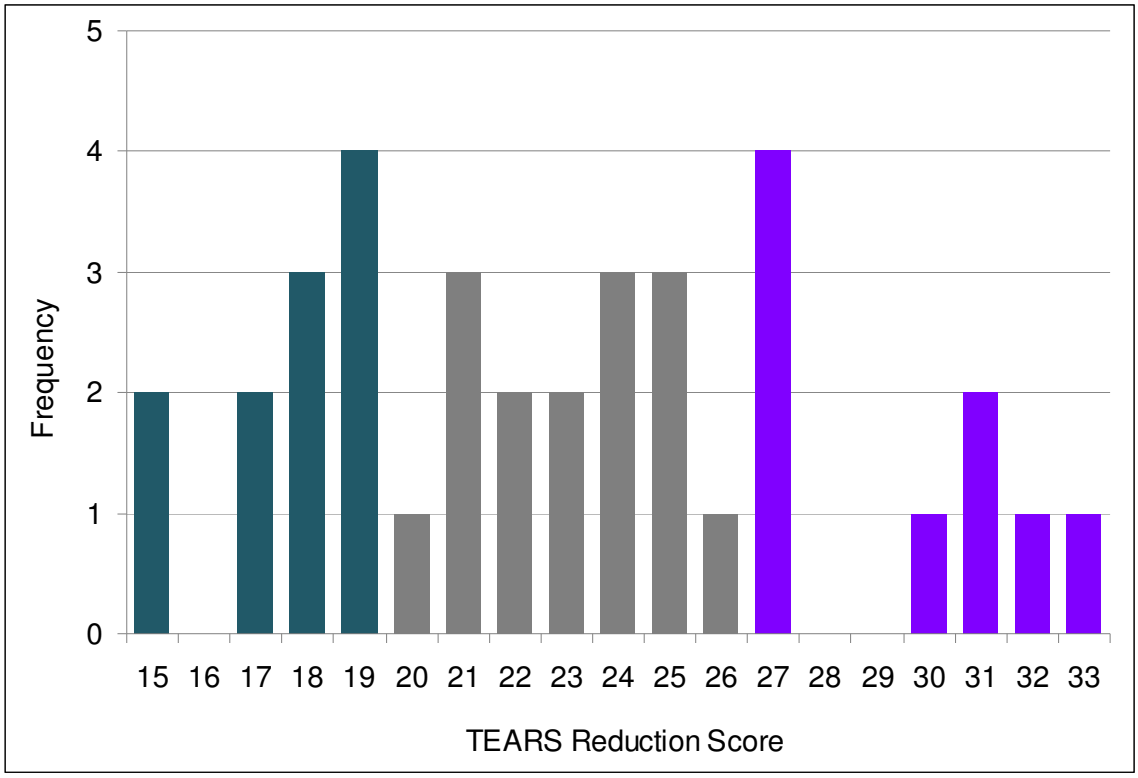

Figure 2. Frequency histogram for TEARS Reduction scores. Scores were ranked among participants and split into quartiles, with the lowest and highest quartiles of scores selected for analysis. Scores falling on the cut points were included. The Low TEARS Reduction (LTR; n = 11) comprised individuals who scored between 15 and 19, and the High TEARS Reduction group (HTR; n = 9) comprised individuals who scored between 27 and 33.

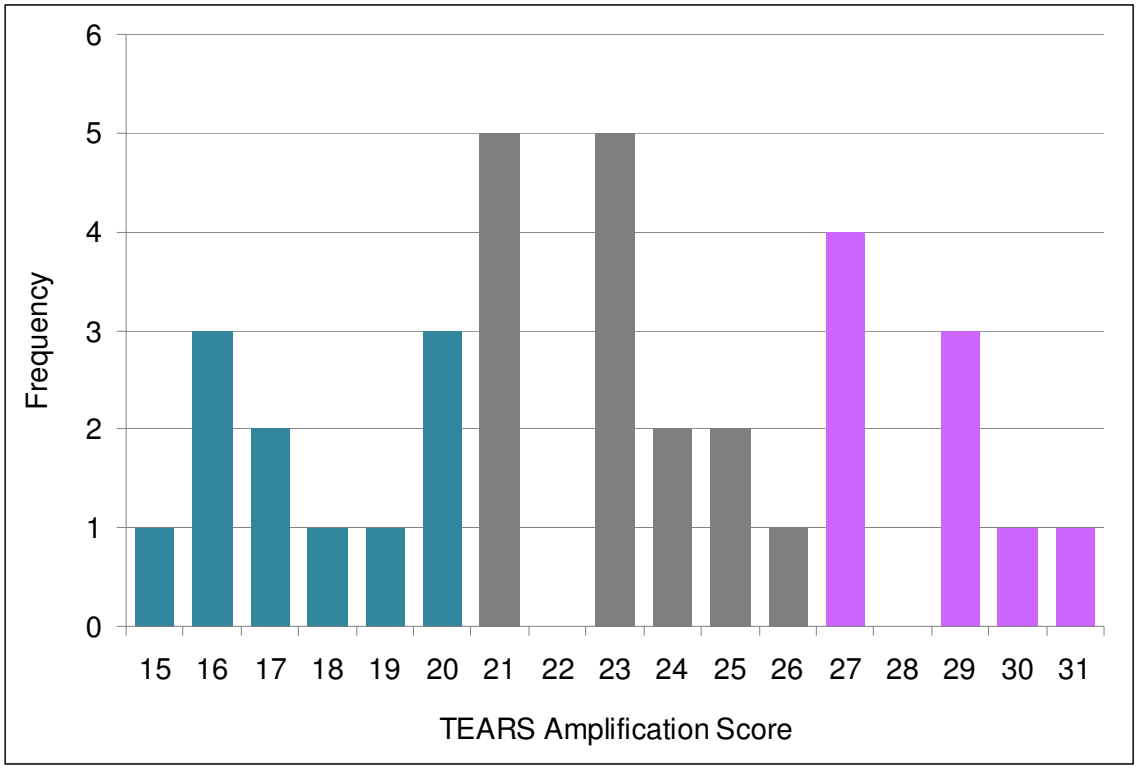

Figure 3. Frequency histogram for TEARS Amplification scores. Scores were ranked among participants and split into quartiles, with the lowest and highest quartiles of scores selected for analysis. Scores falling on the cut points were included. The Low TEARS Amplification (LTA; n = 11) comprised individuals who scored between 15 and 19, and the High TEARS Amplification group (HTA; n = 9) comprised individuals who scored between 27 and 31.

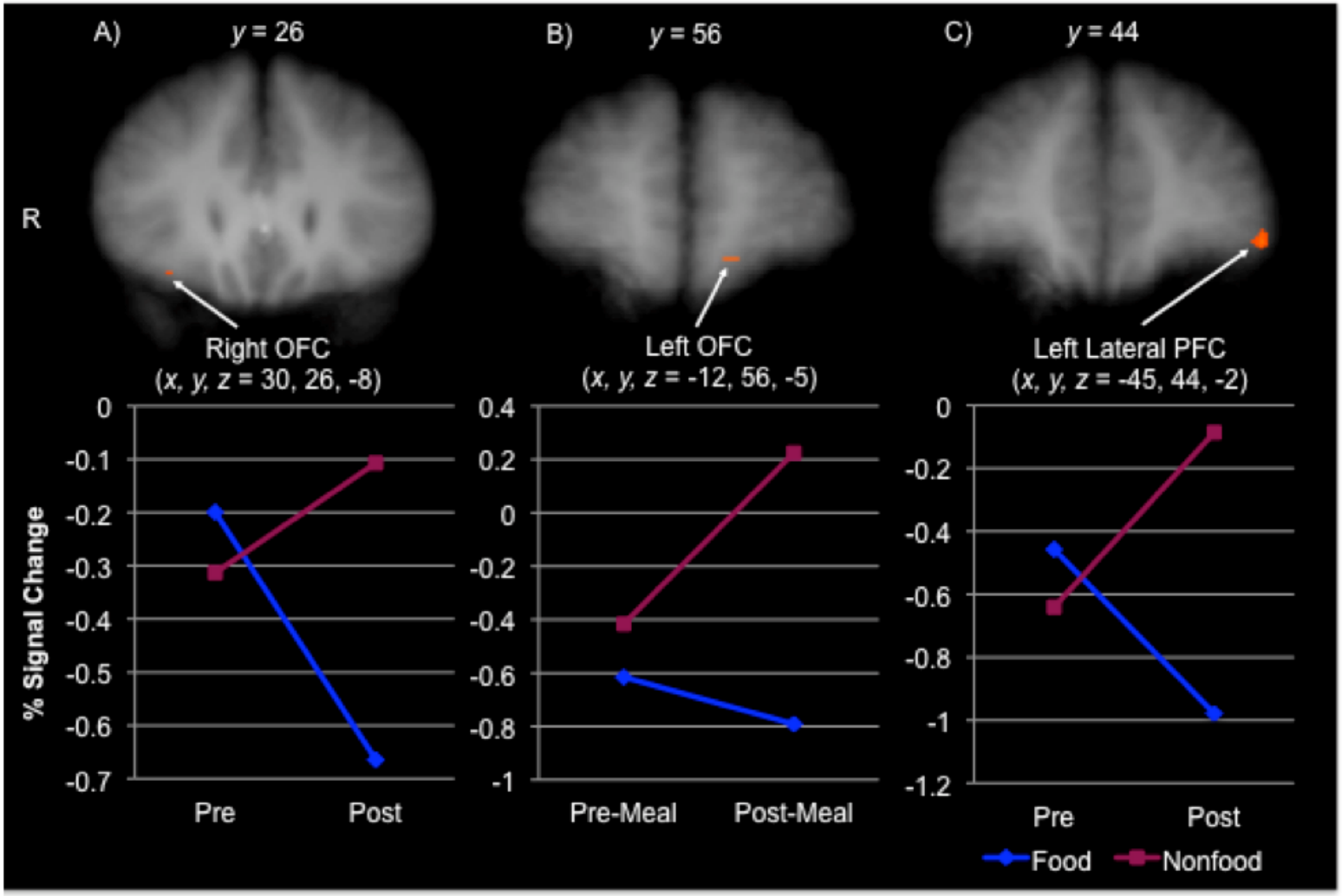

Figure 4. Areas of significant activation difference in the Food versus Nonfood and Pre-meal versus Post-meal contrast using whole-group data. Mean percent signal change in the maximally activated voxel of each cluster was extracted and calculated

for each condition. Two clusters of activation were found in a priori regions, A) the right OFC (*x, y, z* = 30, 26, –8), B) the left OFC (*x, y, z* = –8, 56, –5), and C) the left lateral PFC (*x, y, z* = –45, 44, –2). In all three clusters, there was a greater difference in mean percent signal change at Post-meal than at Pre-meal, such that activation to Food was greater than activation to Nonfood.

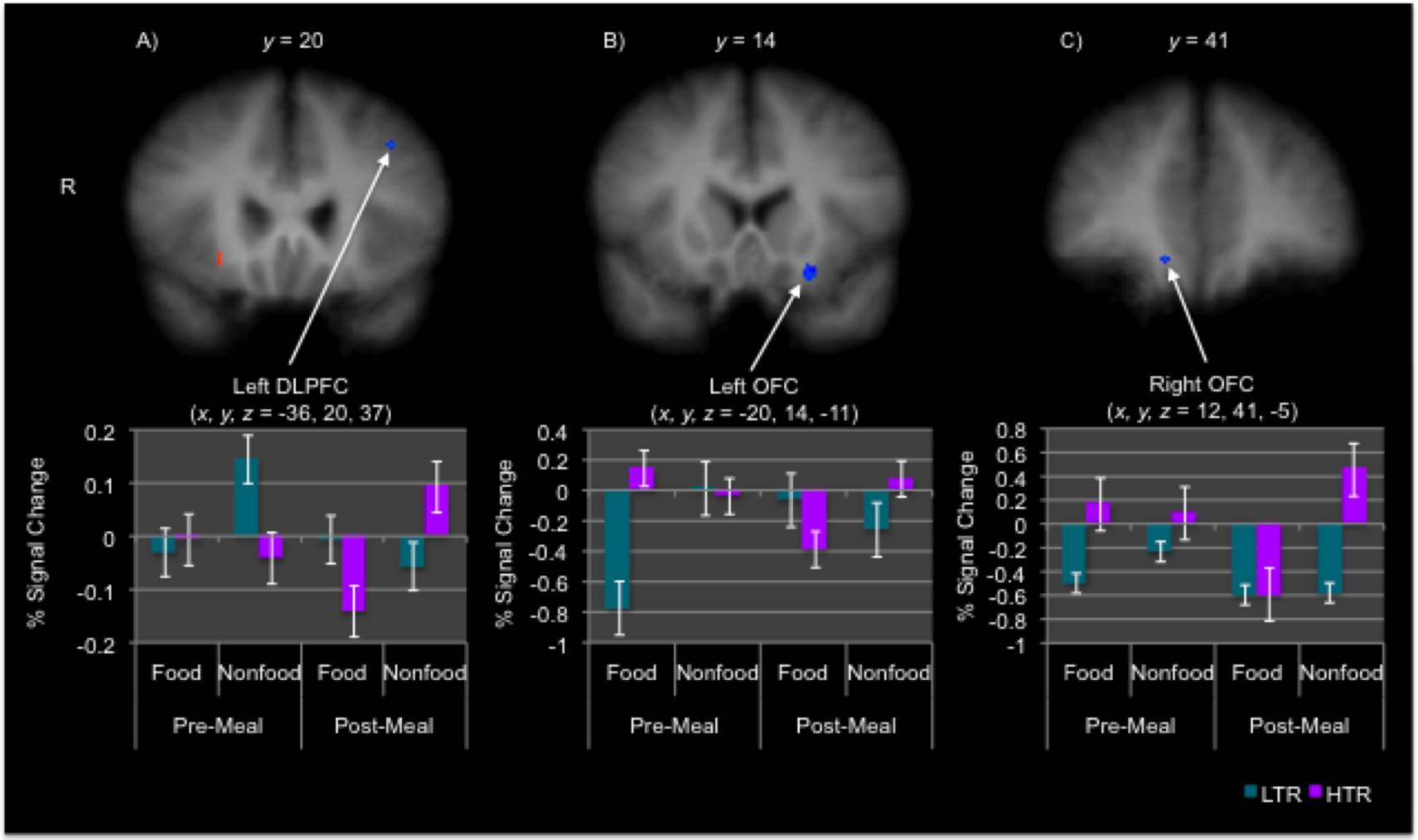

Figure 5. Comparison of Food versus Nonfood images at Pre-meal and Post-meal by LTR versus HTR groups. Mean percent signal change in the maximally activated voxel of each cluster was extracted and calculated for each condition. Three clusters of

activation were found. A) In the left DLPFC (*x, y, z* = –36, 20, 37), the LTR group showed a greater difference in activation (Nonfood greater than Food) at Pre-meal, whereas the HTR group showed a greater difference in activation (Nonfood greater than Food) at Post-meal. B) In the left OFC (*x, y, z* = –20, 14, –11), the LTR group showed a greater difference in activation (Nonfood greater than Food) at Pre-meal, whereas the HTR group showed a greater difference in activation (Nonfood greater than Food) at Post-meal. C) In the right OFC (*x, y, z* = 12, 41, –11), the HTR group showed a greater difference in activation (Nonfood greater than Food) at Post-meal than did the LTR group.

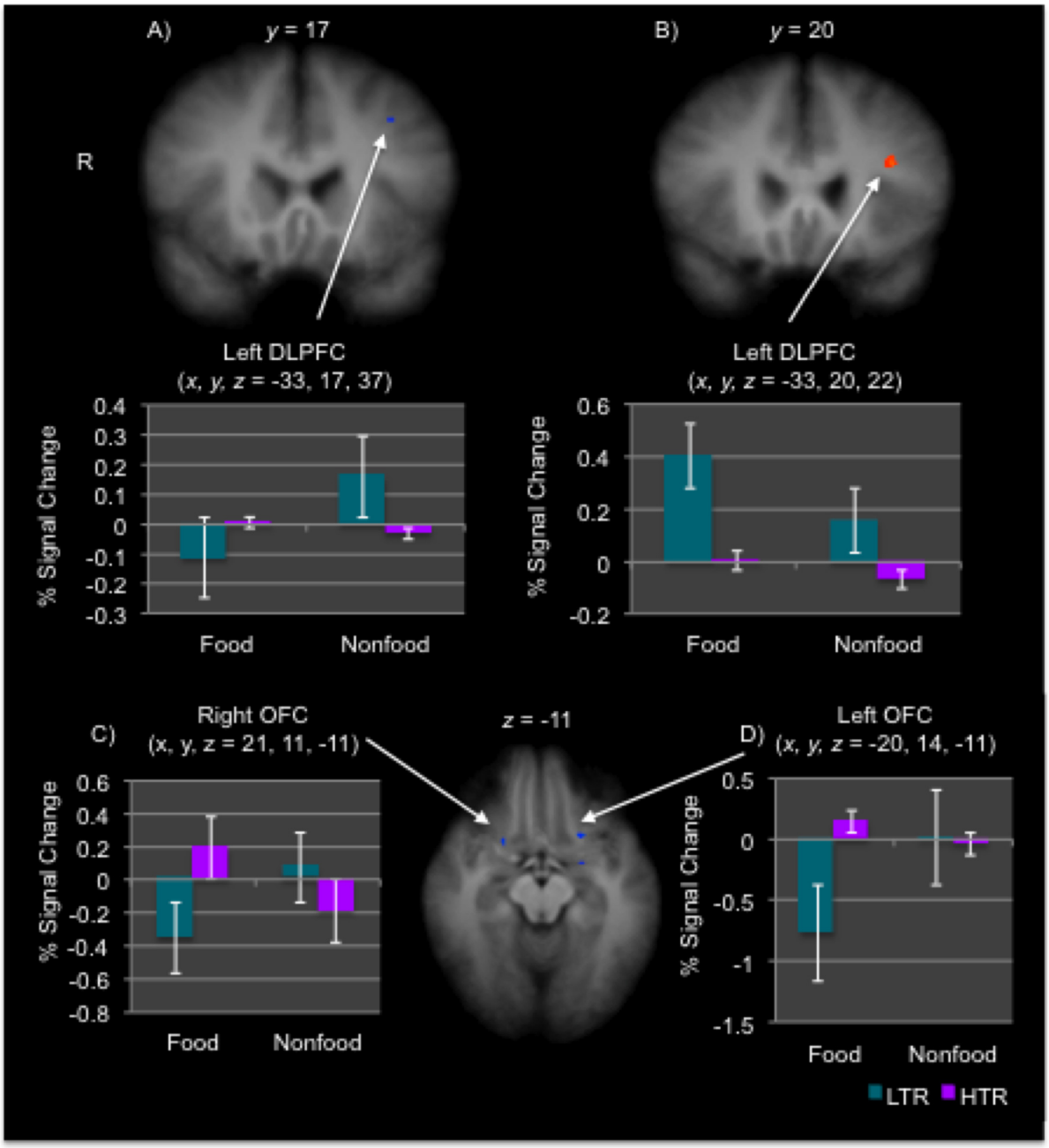

Figure 6. Comparison of Food versus Nonfood images at Pre-meal by LTR versus HTR groups. Mean percent signal change in the maximally activated voxel of each cluster was extracted and calculated for each condition. Four clusters of activation were found in *a priori* regions, A) and B) left DLPFC (*x, y, z* = –33, 17, 37; –33, 20, 22), and C) right OFC

(*x, y, z* = 21, 11, –11) and D) left OFC (*x, y, z* = –20, 14, –11). In all four regions, the LTR group showed greater difference in activation between Food and Nonfood images compared with the HTR group.

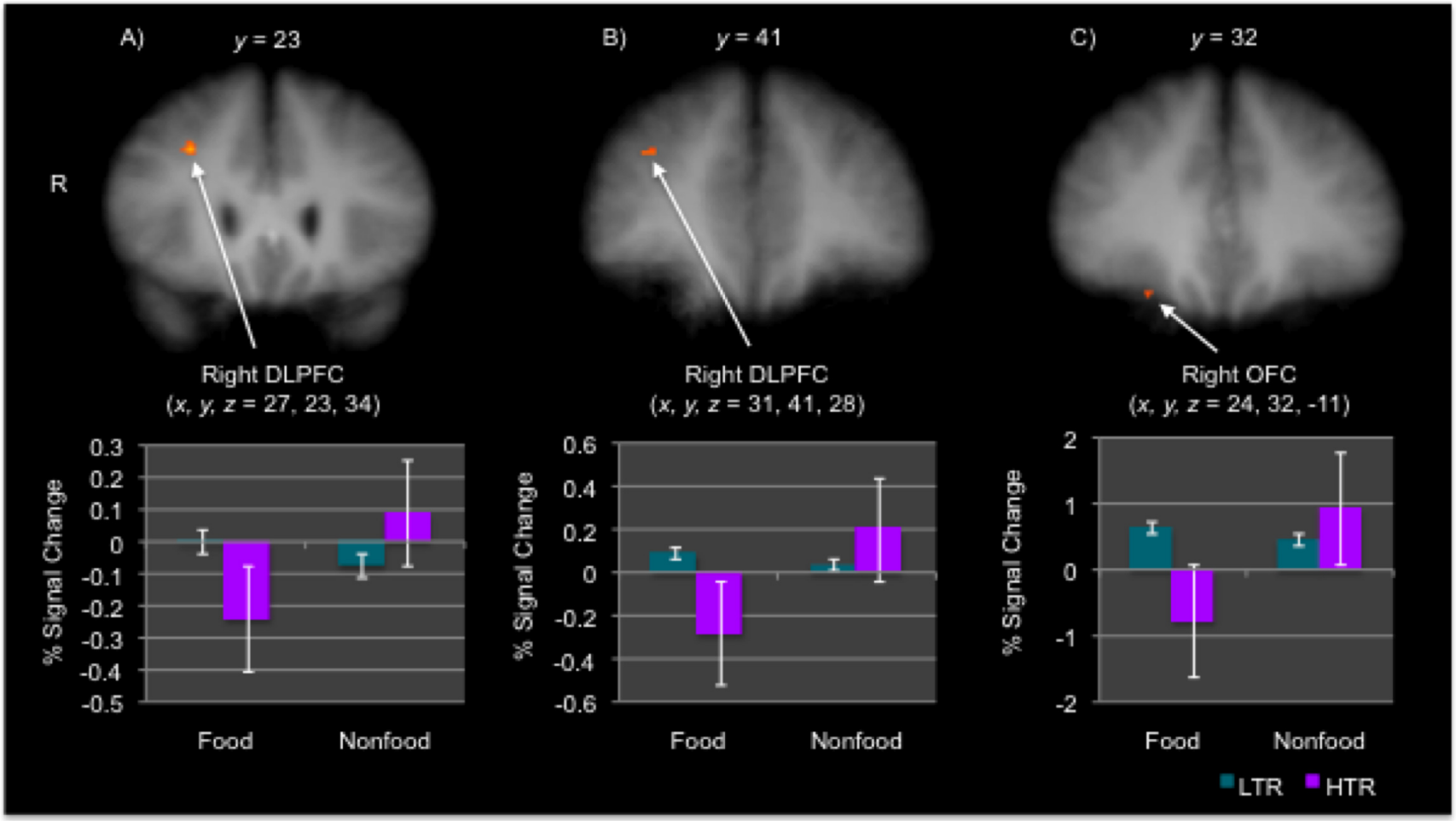

Figure 7. Comparison of Food versus Nonfood images at Post-meal by LTR versus HTR groups. Mean percent signal change in the maximally activated voxel of each cluster was extracted and calculated for each condition. Three clusters of activation were found in a priori regions, A) and B) right DLPFC (*x, y, z* = 27, 23, 34; 31, 41, 28), and C) right OFC (*x, y, z* = 24, 32, –11). In all

three regions, the HTR group displayed greater difference in activation between Food and Nonfood images compared with the LTR group.

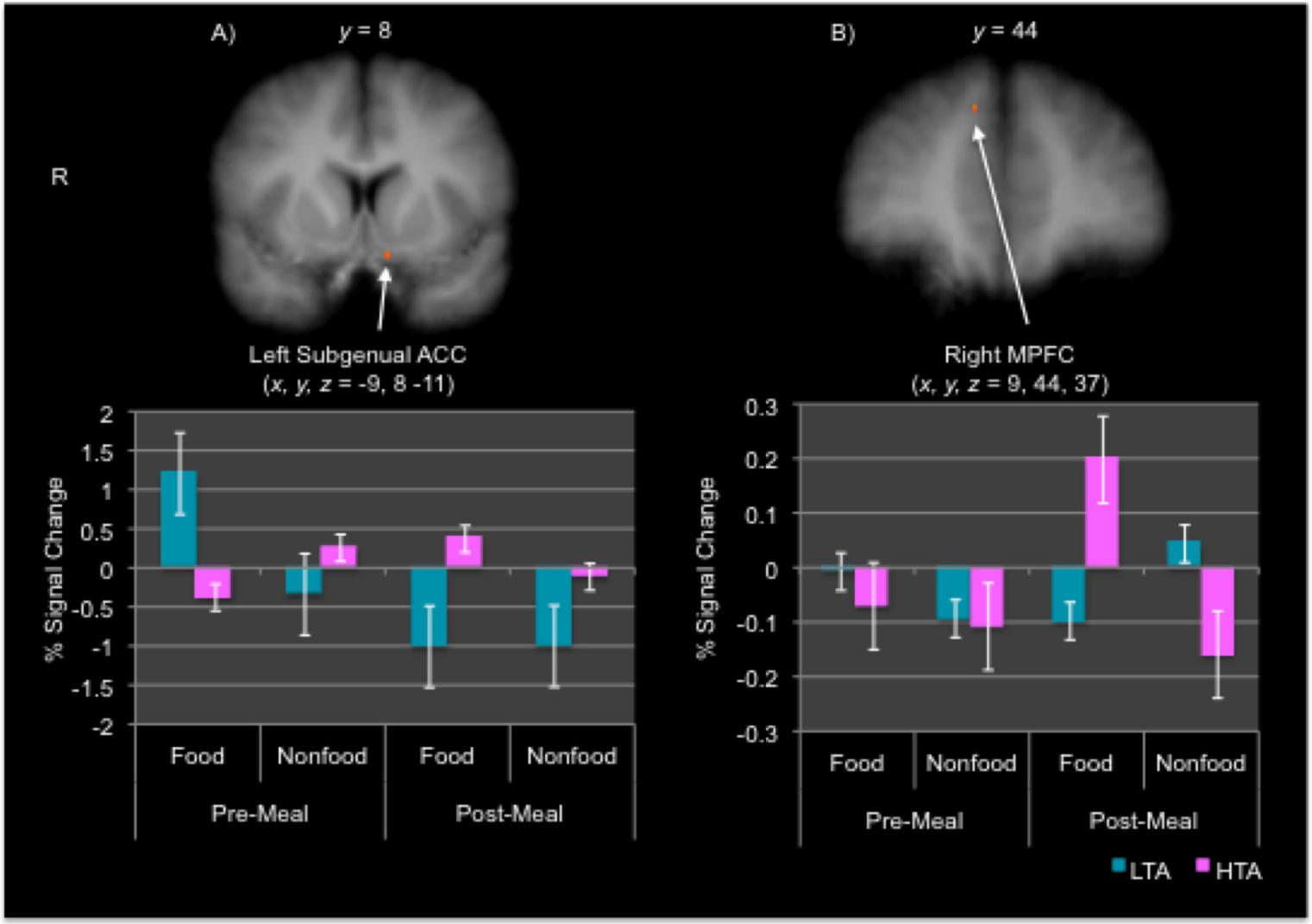

Figure 8. Comparison of Food versus Nonfood images at Pre-meal and Post-meal by LTA versus HTA groups. Mean percent signal change in the maximally activated voxel of each cluster was extracted and calculated for each condition. Two clusters of

activation in *a priori* regions were found. A) In the left sgACC (*x, y, z* = –9, 8, –11), the LTA group showed a greater difference (Food greater than Nonfood) at Pre-meal than did the HTA group. The LTA group also showed a greater change (Pre-meal greater than Post-meal) in activation to Food images than did the HTA group. B) In the right MPFC (*x, y, z* = 9, 44, 37), the HTA group showed a greater difference (Food greater than Nonfood) at Post-meal than did the LTA group. The HTA group also showed a greater change (Post-meal greater than Pre-meal) to Food images than did the LTA group.

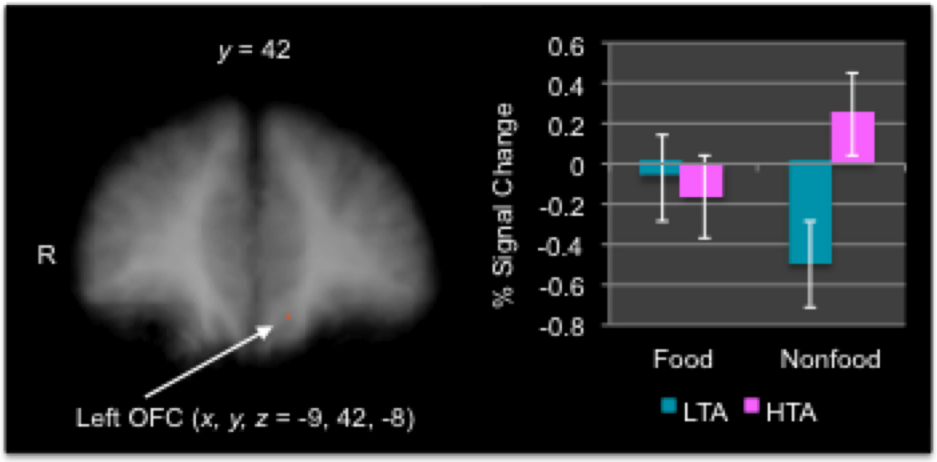

Figure 9. Comparison of Food versus Nonfood images at Pre-meal by LTA versus HTA groups. A cluster of activation was found in the *a priori* region of left OFC (*x, y, z* = –9, 42, –8). There was a greater between-group difference in activation to Nonfood compared with Food images at Pre-meal.

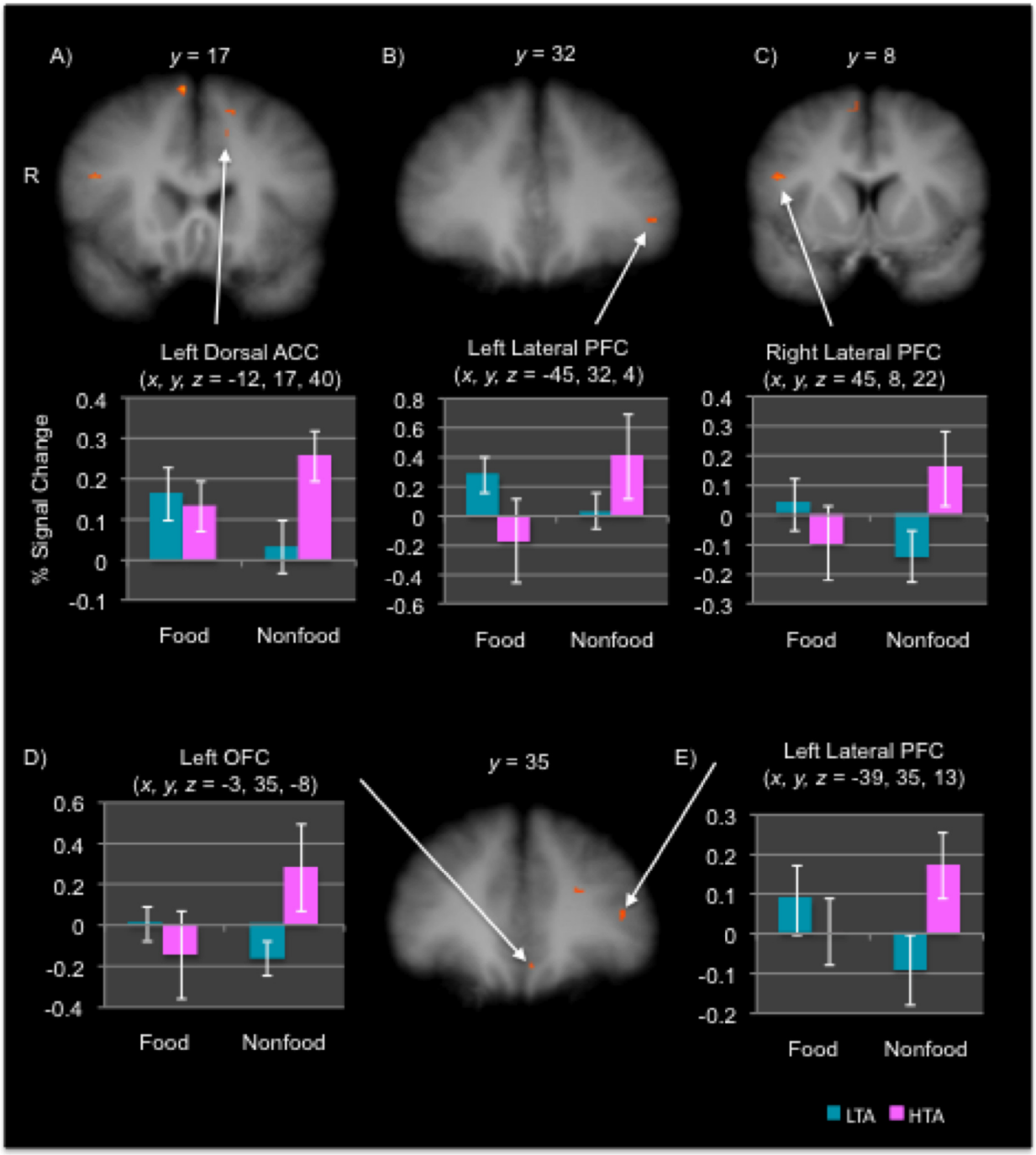

Figure 10. Comparison of Food versus Nonfood images at Post-meal by LTA versus HTA groups. Five clusters of activation were found in a priori regions, A) left dorsal ACC (*x, y, z* = –12, 17, 40), B) and E) left lateral PFC (*x, y, z* = –45, 32, 4; –39, 35, 13), C) right lateral PFC (*x, y, z* = 45, 8, 22), and D) left OFC (*x, y, z* = –3, 35, –8). In the left dACC, there was

a greater between-group difference in activation to Nonfood than Food images (HTA greater than LTA). In the left and right lateral PFC, the LTA group showed greater activation to Food than Nonfood images, and the HTA group showed the opposite pattern. In the left OFC, the HTA group showed greater difference in activation (Food versus Nonfood) than the LTA group.

Appendix

Questionnaires Administered

University of Kansas
Weight Control Research Project
Health History: Weight Management and Brain Function

Today's Date: _____

_____

Last Name First Name Middle Initial

_____

Address

_____

City State Zip Code

Age: _____ Date of Birth: _____ Gender: M F

Which of the following would you say best represents your race?

_____ White

_____ Black or African American

_____ Asian

_____ Native Hawaiian or Pacific Islander

_____ Native American or Alaska Native

_____ Other or Unknown

Which of the following would you say best represents your ethnicity?

_____ Hispanic or Latino

_____ Not Hispanic or Latino

_____ Other or Unknown

The above information is collected for aggregate descriptive purposes only

Home Phone: _____ Cell Phone: _____

Work Phone: _____ Email: _____

Name of Primary Care Physician: _____

Office location:_____

Phone #: _____

Fax #:_____

GENERAL HEALTH HISTORY

Please provide your estimated height and weight.
Ht:_____ inches Wt:_____ lbs

For office use only: BMI: _____ Eligible? □yes □no

1. Do you have or have you ever had any of the following medical conditions?

				Approximate Date/Year of Diagnosis	Describe the Problem
a.	Heart Attack	□yes	□no	_____	_____
b.	Angina (chest pain on exertion)	□yes	□no	_____	_____
c.	Irregular Heart Problems	□yes	□no	_____	_____
d.	Other Heart Problems	□yes	□no	_____	_____
e.	Stroke	□yes	□no	_____	_____
f.	Fainting Spells	□yes	□no	_____	_____
g.	High Blood Pressure	□yes	□no	_____	_____
h.	High Cholesterol	□yes	□no	_____	_____
i.	Thyroid Problems	□yes	□no	_____	_____
j.	Cancer	□yes	□no	_____	_____
k.	Kidney Problems	□yes	□no	_____	_____
l.	Liver Problems	□yes	□no	_____	_____
m.	Gout	□yes	□no	_____	_____
n.	Diabetes	□yes	□no	_____	_____
o.	Emotional/Psychiatric Problems	□yes	□no	_____	_____
p.	Drug/Alcohol Problems	□yes	□no	_____	_____
q.	Claustrophobia	□yes	□no	_____	_____
r.	Osteoarthritis	□yes	□no	_____	_____
s.	Sleep apnea or Snoring	□yes	□no	_____	_____
t.	Excessive daytime sleepiness	□yes	□no	_____	_____
u.	Seizure disorder (epilepsy)	□yes	□no	_____	_____

2. Do you have any medical problems that would prevent you from participating in a regular walking program? □yes □no
   If yes, please describe the problem:_____
   _____

3. Have you had any surgery in the past 12 months? □yes □no
   If yes, please describe the surgery:_____
   _____

4. Have you participated in a regular exercise program over the past 6 months which consists of at least 20 minutes of activity, 3 days per week? □yes □no
   Please describe:_____
   _____

5. Do you have to sleep with extra pillows or have to sit up in the middle of the night because of shortness of breath? □yes □no

6. Please list <u>all</u> medications that you are currently taking on a regular basis (make sure to indicate if you are taking medication for diabetes, high blood pressure or cholesterol):

MEDICATION	REASON FOR TAKING
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

7. How often did you have a drink containing alcohol in the past 6 months?
   Never (skip to question 10)
   Monthly or less
   2 or 4 times a month
   2 or 3 times a week
   4 to 5 times a week
   6 or more times a week

8. How many drinks did you have on a typical day when you were drinking in the past 6 months?
   0 drinks
   1 to 2 drinks
   3 to 4 drinks
   5 to 6 drinks
   7 to 9 drinks
   10 or more drinks

9. How often did you have 6 or more drinks on one occasion in the past 6 months?
   Never
   Less than monthly
   Monthly
   Weekly
   Daily or almost daily

10. What type of drinks did you consume (e.g. beer, hard liquor, etc.)?
_____

11. In the past year, have you regularly smoked cigarettes, pipes, cigars, or used chewing tobacco?

			Please describe daily habit (e.g. 1 pack a day, etc.)
Cigarettes	□yes	□no	_____
Pipe	□yes	□no	_____
Cigars	□yes	□no	_____
Chewing Tobacco	□yes	□no	_____

12. Are you Right or Left handed (dominant)? RIGHT LEFT

13. Do you wear glasses, contacts, or both? GLASSES CONTACTS BOTH
    A. Are you near or far sighted? NEAR FAR

    B. What is the prescription of your glasses? Right: _____ Left: _____

    C. What is the prescription of your contacts? Right: _____ Left: _____

14. What is the highest grade or year of school you completed?
    Never attended school or Kindergarten only
    Grades school
    High school graduate or GED
    College 1-3 years (some college or technical school)
    College or Technical associates or certificate
    College graduate (4 year degree)
    Graduate degree

15. Were you ever held back a grade or advanced a grade in school? YES NO

16. Have you smoked marijuana or taken other drugs within the past 30 days? YES NO

17. Do you have a fear or phobia of any animals? YES NO
    Describe: _____

MRI Patient Safety

18. Have you ever had an MRI? YES NO Describe: _____

19. Are you able to lie flat? YES NO

20. Have you ever had a kidney/liver transplant or have kidney or liver disease? YES NO Describe: _____

21. Have you ever done welding or metal work? YES NO

22. Have you ever had any type of injury involving bullets, metal, or shrapnel being imbedded or implanted in your body or eyes, or do you have a body piercing? YES NO

23. Do you have permanent eyeliner or tattoos? YES NO

24. Do you wear a hearing aide or have cochlear implants? YES NO

25. Do you have an eyelid spring or wire? YES NO

26. Do you have dentures or removable dental work? Do you have metal crowns, fillings, or braces? YES NO Describe: _____

27. Do you have an artificial limb or joint? YES NO

28. Do you have an insulin pump? YES NO

29. Do you have any medication skin patches? YES NO

30. Do you have vascular IV access (e.g. IV port)? YES NO

31. Do you have any type of equipment attached for pain control or stimulation? YES NO

32. Do you have a cardiac or gastric pacemaker or defibrillator? YES NO

33. Do you have aneurysm clips? YES NO

34. Do you have a vagus nerve stimulator? YES NO

35. Do you have a hydrocephalus shunt tube? YES NO

36. Do you have any intravascular stents, filters, or coils? YES NO

37. Do you have a spinal shunt? YES NO

38. Do you have any Harrington rods? YES NO

WOMEN ONLY ANSWER THE FOLLOWING QUESTIONS

39. Are you currently pregnant? □yes □no

40. Were you pregnant within the past 6 months? □yes □no

41. Do you plan to become pregnant in the next 18 months? □yes □no

42. Do you have an intrauterine device (IUD)? □yes □no

43. Have you gone through menopause or the change of life? □yes □no

44. Have you had a hysterectomy? □yes □no

45. When was your last menstrual period? DATE: _____/_____/_____

46. Do you take :
    - Birth Control Pills? □yes □no
    - Estrogens (i.e. Premarin)? □yes □no
    - Progesterone (i.e. Provera)? □yes □no

THANK YOU!

PHYSICIAN CONSENT TO PARTICIPATE IN A DIET AND PHYSICAL ACTIVITY PROGRAM AT THE UNIVERSITY OF KANSAS

TO: _____ **Physician's Name**	**PLEASE FAX TO:** **Tony Lynch** **University of Kansas** **Schiefelbusch Institute for Life Span Studies**
Address	**Center for Physical Activity and Weight Management**
	1301 Sunnyside, Robinson RM 100
City State Zip	**Lawrence, KS 66045**
( )	**Telephone:** **FAX: (785) 864-2009**
Telephone Number	

Your patient _____ has asked to participate in a diet and exercise program at the University of Kansas. This is a 9-month research study designed to help individuals to change their eating and exercise habits and to examine the impact that this will have on long-term weight loss and increases in physical fitness. This will involve the following:

1. A walking program that will be primarily home-based. The exercise will gradually be progressed from 15 minutes per day to as much as 60 minutes per day, 5 days per week for a total of 2,000 kcal energy expenditure per week. Exercise intensity will be set at 50-70% of the individual's maximal heart rate.

2. A diet program that will reduce energy intake to 1200-1500 calories per day, with dietary fat reduced to 20-30% of total energy intake. The diet will use meal replacements for entrees and will be combined with fruits and vegetables, grains, and beverages.

3. Magnetic Resonance Imaging (MRI).

4. Behavioral modification techniques for changing diet and exercise behaviors.

5. Additional factors that are exclusionary criteria for this study that you should consider are listed on the attached sheet.

****

Please indicate below if this program seems appropriate for your patient or if you see any contraindications for her participation (*please check the appropriate box below)*.

I know of no contraindications to this patient participating in any of the above components of the program.

I feel that this program would not be appropriate for this patient for the following reason(s):

_____

_____ _____

Signature of Physician **Date**

Please consider the following Inclusion and Exclusion Criteria as you evaluate whether your patient is capable of safely participating in the weight loss and exercise research study at the University of Kansas.

Inclusion Criteria:	**Exclusion Criteria:**
• Female or Male • 21 - 55 years of age · BMI = 30 - 39.9 kg/m^2 • Ability to provide informed consent. • Ability to provide consent from their personal physician to participate in this study. • No plans to move out of the Kansas City area for at least 1 year.	• Reporting regular exercise participation of at least 20 minutes per day on at least 3 days per week or greater than 500 kcal per week during the previous six months. (*This study is designed to recruit relatively sedentary adults.)* • Insulin dependent diabetes, heart disease, cancer, thyroid condition, high blood pressure, stroke, seizure disorder, current or history of psychological disorder including but not limited to depression, anxiety, chemical dependency, anorexia, bulimia, history of bariatric surgery, or other medical conditions which would affect energy metabolism or your patient's ability to safely participate in this study. • Women who are currently pregnant, pregnant within the previous six months, or planning on becoming pregnant within the next year. (Pregnancy during initial screening will be based on self-report and will be included on the detailed medical history that is completed by subjects. Pregnancy will be ruled out via urine Beta HcG testing prior to MRI) • Taking any medication that would severely diminish exercise capacity (e.g., beta blockade) or alter metabolism (e.g., steroids, metabolife, dexatrim, etc...) • Arrhythmia on electrocardiogram that would indicate that moderate exercise was contraindicated. • History of orthopedic complications that would prevent optimal participation in the exercise component (e.g., heel spurs, severe arthritis).

Energy Balance Laboratory & Center for Physical Activity and Weight Management
1301Sunnyside Ave., Robinson Center, Room 100, Lawrence, KS 66045-7555.
Phone: (785) 331-4681 Fax: (785) 864-2009. wcrp@ku.edu

The Emotion Amplification and Reduction Scales

Instructions: Please respond to each of the following statements as they apply to you and your experience with your feelings and emotions. Use the following response scale:

	1= Not at all true for me 2= Somewhat true for me 3= Moderately true for me 4= Very true for me
1. I can use my emotions or feelings to my advantage.	1 2 3 4
2. I can control my emotional reaction to events or situations.	1 2 3 4
3. When the need arises, I can cut short an emotional response.	1 2 3 4
4. I can stop an emotion before it overwhelms me.	1 2 3 4
5. Prior to a stressful situation, I can get myself into a calm state that actually prevents me from feeling bad when the stressful event happens.	1 2 3 4
6. If I want to, I can get myself emotionally "charged up".	1 2 3 4
7. I can get emotionally "revved up" to enhance my performance.	1 2 3 4
8. If I wanted to, I could turn UP the intensity level of whatever emotion I may be feeling.	1 2 3 4
9. I can do things that will enrich my emotional experience.	1 2 3 4
10. When I know in advance that I will be faced with an exciting or stressful situation, I could (if I wanted to) remain calm.	1 2 3 4
11. I can choose to remain calm in almost any situation.	1 2 3 4
12. I can readily make myself tone down the intensity of any emotion that I might be feeling.	1 2 3 4
13. I can deepen the feeling of an existing emotion.	1 2 3 4
14. When I know in advance that an upcoming situation is going to make me feel a particular emotion (such as sadness or anger), I am able to do things that prevent the feelings from occurring when that situation arises.	1 2 3 4
15. I can do things that will deepen my emotional experience.	1 2 3 4

16. I can harness the energy of my emotions to enhance my performance.	1	2	3	4
17. No matter how intensely I may be feeling a particular emotion, I can almost always make myself calm down.	1	2	3	4
18. I can hold on to a feeling or emotion.	1	2	3	4

Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions related to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get during the night? (This may be different than the number of hours you spend in bed) _____

5. During the past month, how often have you had trouble sleeping because you. . .	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times a week (3)
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason.				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
9. During the past month, how would you rate your sleep quality overall?				

The University of Kansas Weight Management Programs
CES-D: Feelings, Attitudes and Behaviors

Below is a list of feelings, attitudes, and behaviors that you may have experienced **DURING THE PAST WEEK.** Please use the following scale and circle the one response that best describes how often you have had these experiences **DURING THE PAST WEEK. Rarely or none of the time = less than one day; Some or a little of the time = 1 or 2 days; Moderately = 3 or 4 days; Most or all of the time = 5 to 7 days.**

(Circle one number on each line)

During the past week…	Rarely	Some of the time	Moderately	Most of the time
1) I was bothered by things that usually don't bother me. . .	0	1	2	3
2) I did not feel like eating; my appetite was poor	0	1	2	3
3) I felt that I could not shake off the blues even with help from family and friends	0	1	2	3
4) I felt that I was just as good as other people	0	1	2	3
5) I had trouble keeping my mind on what I was doing	0	1	2	3
6) I felt depressed (blue or down)	0	1	2	3
7) I felt that everything I did was an effort	0	1	2	3
8) I felt hopeful about the future	0	1	2	3
9) I thought my life had been a failure	0	1	2	3
10) I felt fearful	0	1	2	3
11) My sleep was restless	0	1	2	3
12) I was happy	0	1	2	3
13) I talked less than usual	0	1	2	3
14) I felt lonely	0	1	2	3
15) People were unfriendly	0	1	2	3
16) I enjoyed life	0	1	2	3
17) I had crying spells	0	1	2	3
18) I felt sad	0	1	2	3
19) I felt that people disliked me	0	1	2	3
20) I could not "get going"	0	1	2	3