A LONGITUDINAL INVESTIGATION OF PSYCHOLOGICAL AND NEURAL MECHANISMS OF WEIGHT LOSS

BY

C2013

TRISHA M. PATRICIAN

Submitted to the graduate degree program in Psychology and the Graduate Faculty of the University of Kansas in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

_____

Nancy Hamilton, Ph.D.

_____

Cary Savage, Ph.D.

_____

Rick Ingram, Ph.D.

_____

Christie Befort, Ph.D.

_____

Laura Martin, Ph.D.

Date Defended: July 22, 2013

The Dissertation Committee for Trisha Patrician

certifies that this is the approved version of the following dissertation:

A LONGITUDINAL INVESTIGATION OF PSYCHOLOGICAL AND NEURAL MECHANISMS OF WEIGHT LOSS

_____

Co-Chairperson: Nancy Hamilton, Ph.D.

_____

Co-Chairperson: Cary Savage, Ph.D.

Date approved: July 22, 2013

Abstract

Although obesity is fundamentally a problem of energy balance wherein calorie intake exceeds calorie output, there is a multitude of psychological and neural factors inherent in eating and overeating. Behavioral and neuroimaging research suggests a relationship between emotion regulation and eating behavior. However, the connections among psychological characteristics, brain function, and weight loss maintenance are poorly understood. Accordingly, in the present study, fMRI was used to examine how two psychological characteristics, emotion amplification and rumination, are related to (a) neural response to food images both before (baseline [BL]) and after (3M) participants undergo a three-month behavioral weight loss intervention, and (b) initial weight loss and weight loss maintenance. Emotion amplification was associated with decreased activation in dorsolateral prefrontal cortex (DLPFC) from BL to 3M. Rumination was associated with decreased activation in DLPFC from BL to 3M and increased activation in lateral prefrontal cortex, anterior cingulate cortex, parahippocampal gyrus, and amygdala from BL to 3M. Rumination was also inversely correlated with BL post-meal activation in DLPFC and caudate; activation in these two regions was prospectively associated with more weight loss from BL to 3M. Findings suggest that emotion amplification and rumination contribute to how food stimuli are processed at a neural level. Potential mechanisms for behavioral regulation and treatment implications are discussed.

Keywords: obesity, weight loss, emotion regulation, fMRI

Acknowledgments

It takes a village to raise a graduate student into a clinical psychologist. This research project and the resulting dissertation would not have been possible without the support of many people. I would like to express my gratitude to my mentors, Dr. Cary R. Savage and Dr. Nancy A. Hamilton, whose expertise, guidance, and encouragement were vital to the completion of this project. Thank you also to my committee members, Dr. Rick Ingram, Dr. Laura Martin, and Dr. Christie Befort, for their assistance and valuable input throughout the various stages of the project.

I am indebted to my colleagues, especially Dr. Rebecca Lepping, Florence Breslin, and Joshua Powell, whose abundant wisdom with data collection, data analysis, and manuscript write-up, as well as emotional and technical support, have been vital to my success.

I would like to express my love and appreciation to my friends and family, near and far, who have supported me in big and small ways over the years. Above all, I owe my deepest gratitude to my husband Stephen, for the countless ways he has shown and continues to show his love for me. Stephen, you finally get to know me as something other than a graduate student!

Table of Contents

List of Figures

List of Tables

A Longitudinal Investigation of Psychological and Neural Mechanisms of Weight Loss

The Problem of Obesity

Obesity (body mass index [BMI] $\geq$ 30 kg/m^2) is a significant public health problem in the United States, with a current prevalence of roughly 35% of American adults (Flegal, Carroll, Kit, & Ogden, 2012). 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 (Kopelman, 2000, 2007). A recent study estimated that obesity-related health care costs account for about 20% of total medical costs in the United States, at approximately $209.7 billion (Cawley & Meyerhoefer, 2011). Clearly obesity places a significant burden on the healthcare system, the economy, and the well-being of the general public.

Although fundamentally a problem of energy balance wherein calorie intake exceeds calorie output, obesity is a complex and heterogeneous medical condition (Brownell & Wadden, 1992). In an effort to better understand this complexity, various lines of research have examined the interactions among the neural, cognitive, and emotional correlates of eating behavior, obesity, and weight loss (e.g., DelParigi et al., 2006; Evers, Stok, & de Ridder, 2010; Macht, 2008; Niemeier, Phelan, Fava, & Wing, 2007; Polivy, Heatherton, & Herman, 1988; Polivy & Herman, 1999; Simmons, Martin, & Barsalou, 2005). From this literature, it is evident that difficulty with emotion regulation is influenced by brain function and negatively impacts the ability to manage eating behavior in the service of losing or maintaining body weight. Accordingly, the present study will examine characteristics of emotional processing such as self-reported ability to amplify emotion and tendency toward rumination, brain activation to food images, weight loss, and weight maintenance in a sample of obese adults entering a diet-based

weight loss program. It is anticipated that a better understanding of these relationships may contribute to individually tailored and more effective obesity treatments, particularly with respect to weight loss maintenance.

Psychological Mechanisms of Eating and Overeating

Eating behavior is driven by a multitude of biological, psychological, and social factors, many of which are beyond the scope of this paper. Importantly, however, eating is inherently an affective experience, a relationship that is reflected both in the psychological motivation to eat and overeat, and in the neural response to food cues as well as to food anticipation and consumption. The current understanding of the psychological theories and neural mechanisms of eating behavior may help to illuminate why losing weight and maintaining weight loss are so difficult for many people.

Psychological theories of eating behavior. Notably, psychological theories of eating behavior are predicated on the observation that the reward-related and emotion regulatory properties of food provide powerful motivation to consume and even over-consume highly palatable food, leading to disinhibited eating (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). Disinhibition is especially problematic among individuals attempting to control not only food consumption but also thoughts related to food and eating (Polivy & Herman, 1985; Stunkard & Messick, 1985). This cognitive and behavioral process, known as dietary restraint or restrained eating, was shown in early laboratory studies to be associated with increased ice cream consumption following a pre-load condition (Herman & Mack, 1975). Disinhibited eating may also occur in response to heightened self-referential negative affect, as a method of escaping this discomfort (Heatherton

& Baumeister, 1991; Heatherton, Striepe, & Wittenberg, 1998), or even as a form of distraction from general distress (Macht, Haupt, & Ellgring, 2005). It has also been argued that the energy required for emotion regulation draws from a limited supply of self-regulatory resources, thereby reducing the ability to resist the temptation of tasty food and creating a vulnerability to disinhibited eating (Baumeister & Heatherton, 1996; Muraven et al., 1998; Vohs & Heatherton, 2000). Considered together, research findings indicate that heightened affect coupled with insufficient regulatory or coping ability set the stage for increased eating, particularly of energy dense foods, thereby impeding upon weight management goals.

Brain function and eating behavior. Among the factors contributing to effective management of eating behavior is the reliance on the balance of neural processes that initiate and terminate eating. Hunger is associated with activation of a network of brain regions that includes the amygdala, hypothalamus, insula, orbitofrontal cortex (OFC), hippocampus and parahippocampal gyrus, caudate, and putamen (LaBar et al., 2001; Tataranni et al., 1999). An analysis of multiple positron emission tomography (PET) studies revealed that the insula, OFC, and frontal and parietal operculum are prime regions for processing of gustatory stimuli (Small et al., 1999). Small et al. (1999) contended that the OFC is also involved in cognitive or motivational aspects of gustatory processing. Further, the specific food-related properties of fat and sweet differentially activated the ventral anterior cingulate cortex (ACC), suggesting this region's involvement in motivating consumption of palatable foods (de Araujo & Rolls, 2004). Images of high calorie foods have been shown to elicit increased activation in regions of the brain known to be involved with habit learning and reward processing, such as dorsal and ventral striatum, including nucleus accumbens, and with taste information processing, such as OFC and insula (Rothemund et al., 2007). Additionally, food craving led to increased activation in the

striatum, insula, and hippocampus (Pelchat, Johnson, Chan, Valdez, & Ragland, 2004).

In contrast, the termination of eating is associated with differential activation of dorsal and lateral prefrontal cortex, regions that are involved more generally in the inhibition and regulatory control of emotion and behavior. Studies have shown satiety to be positively associated with activation in the left dorsolateral prefrontal cortex (DLPFC; Pannacciulli et al., 2006), bilateral DLPFC and ventrolateral PFC (VLPFC; Tataranni et al., 1999), and the left lateral PFC (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). Additionally, visual food cues have been shown to be associated with greater activation in areas of the brain typically involved in effortful emotion regulation and cognitive control, including DLPFC, VLPFC, and dorsal ACC (see Carnell, Gibson, Benson, Ochner, & Geliebter, 2012, for a review). With regard to food motivation and eating behavior, the DLPFC has been associated with heightened dietary restraint (Burger & Stice, 2011; Le et al., 2007), weight loss success (McCaffery et al., 2009), and cognitive control of food-related reward (Hollmann et al., 2011). However, research findings also indicate that greater activation in insula and operculum to food anticipation coupled with less activation in striatum to food consumption may lead to overeating due to blunted food-elicited reward response (Stice, Spoor, Ng, & Zald, 2009). The dorsal striatum has been associated with aspects of behavioral self-regulation including stimulus-response learning (Featherstone & McDonald, 2004), rule learning (Seger & Cincotta, 2006), and habit formation (Yin, Knowlton, & Balleine, 2004). There is also some evidence to suggest that dorsal striatum plays an inhibitory role, particularly when co-activated with DLPFC (Hollmann et al., 2011; Marsh et al., 2009).

Collectively, hunger and the motivation to eat are associated with activation of limbic, paralimbic, and striatal areas of the brain, as well as midline areas of the prefrontal cortex such

as OFC and ACC. Thus, the motivation to eat, particularly to eat palatable food, is driven by a network of regions found to be instrumental in processing taste, reward, and affective states. In contrast, satiety and decreased eating are associated primarily with activation of dorsal and lateral aspects of the prefrontal cortex. Therefore, cessation of eating is driven by activation in regions known to be responsible for emotion regulation, cognitive control, and behavioral self-regulation.

Integration of psychological and neural mechanisms in eating behavior. Because eating and emotions are intertwined, it is not surprising that the initiation and cessation of eating are modulated by many of the same brain regions known to facilitate the experience and regulation of emotions. Integration of psychological, behavioral, and neural findings may further clarify the mechanisms behind intentional weight loss and weight loss maintenance. Specifically, the current knowledge of emotions and eating may be applied toward better understanding why many individuals struggle with long-term self-regulation of eating and weight.

Mechanisms of Weight Loss and Maintenance

Despite the popularity of a variety of diets and the increased accessibility of health information, people experience great difficulty achieving and maintaining weight loss over time (Wiess, Galuska, Khan, Gillespie, & Serdula, 2007). The adage "eat less and exercise more" reflects the fact that weight loss can be achieved by decreasing energy intake and increasing energy output. Thus, most weight loss and weight loss maintenance interventions focus on changing diet, increasing physical activity, and engaging in self-monitoring behaviors such as frequent self-weighing. These components are generally supported by literature on successful weight loss and weight maintenance. However, it is also established that psychological

mechanisms including negative affect and disinhibition can hijack attempts to "eat less and exercise more." Additionally, an emerging line of research has revealed that weight loss mechanisms appear at a neural level and that brain function may predict and/or reflect intervention success. Behavioral, psychological, and neural mechanisms of weight loss and maintenance will therefore be examined in more detail below.

Behavioral mechanisms of weight loss and maintenance. A large amount of empirical research has emerged from the National Weight Control Registry (NWCR), an ongoing registry composed of individuals who have lost at least 30 pounds (13.6 kg) and kept that weight off for at least one year (Klem, Wing, McGuire, Seagle, & Hill, 1997). Studies using the NWCR have shown that successful weight loss maintenance is associated with consumption of less than 25% of calories from fat, consumption of breakfast, eating five times per day (three meals and two snacks), engaging in greater than 60 minutes of physical activity per day, and consistent daily self-weighing (Bachman, Phelan, Wing, & Raynor, 2011; Butryn, Phelan, Hill, & Wing, 2007; McGuire, Wing, Klem, Lang, & Hill, 1999; Phelan, Roberts, Lang, & Wing, 2007; Schick et al., 1998; Wyatt et al., 2002). Other studies not using the NWCR that have examined behavioral predictors of weight maintenance have shown that successful weight maintenance is associated with weighing oneself weekly (VanWormer et al., 2009), frequently exercising at least 30-60 minutes per day (Befort et al., 2007; Kruger, Blanck, & Gillespie, 2006), eating five or more fruits and vegetables per day (Befort et al., 2007; Champagne et al., 2011), and practicing portion control (Befort et al., 2007; Kruger et al., 2006; Reyes et al., 2012).

Psychological mechanisms of weight loss and maintenance. Studies have found that the ability to engage in the aforementioned behavioral strategies in order to lose weight or maintain weight loss can be hindered by the presence of negative affect, especially when

emotion regulatory efforts are not successful. In a two-year prospective analysis of a large sample of individuals from the NWCR, Phelan et al. (2003) found that those who were unable to recover from relapse had higher scores on the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977) compared with those who were able to lose weight they had regained. Similarly, a qualitative investigation of 15 men and women in a behavioral weight loss program revealed that participants attributed difficulty with weight loss to emotion-related factors such as stress, depression, and fatigue (Adolfsson, Carlson, Undén, & Rössner, 2002). An additional qualitative study showed that poor adherence to self-monitoring through food diary use was associated with a tendency toward emotional eating, as well as with poor weight loss outcome (Burke, Swigart, Turk, Derro, & Ewing, 2009). Weight regain has been associated with a number of factors, including greater depression, dietary disinhibition, and binge eating (McGuire et al., 1999; Niemeier et al., 2007; Wing et al., 2008).

In contrast, the ability to cope effectively with temptation as well as the ability to generate and use emotion regulation strategies appears to positively influence eating behavior and weight management. A qualitative comparison of successful weight maintainers versus regainers indicated that positive self-talk and productive problem-solving were related to successful maintenance (Reyes et al., 2012). A cross-sectional study of obese women showed that relapsers (individuals who had lost weight but gained it back) tended to cope with problems by using avoidance methods such as eating, whereas maintainers tended to cope with problems by confronting and working to solve them (Kayman, Bruvold, & Stern, 1990). Drapkin, Wing, and Shiffman (1995) investigated coping responses to a set of hypothetical high-risk tasks in a sample of obese individuals with diabetes. They found that the number of hypothetical situations to which individuals who were able to generate any coping strategy was positively associated

with weight loss during a 12-month weight loss intervention. Similarly, Grilo, Shiffman, and Wing (1989) found that individuals who were able to generate either a cognitive or a behavioral coping strategy in response to an actual temptation situation were less likely to give in to the temptation and overeat. Elfhag and Rössner (2005) suggested that the successful weight loss maintainer establishes flexible control over eating and exhibits creativity in problem-solving and coping. Taken together, difficulty regulating negative affect may contribute to difficulty losing weight and maintaining weight loss, whereas ability to engage in adaptive emotion regulation strategies appears to facilitate weight loss outcomes.

Neural mechanisms of weight loss and maintenance. In the past two decades, various research groups have used brain imaging research using techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) in order to examine the neural correlates of obesity (e.g., Dimitropoulos, Tkach, Ho, & Kennedy, 2012; Gautier et al., 2001; Le et al., 2006, 2007; Martin et al., 2009; Rothemund et al., 2007; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008; Stoeckel et al., 2008). An in-depth discussion of these studies is beyond the scope of this paper (see Carnell, Gibson, Benson, Ochner, & Geliebter, 2012, for a review). However, in general, findings from imaging studies examining brain activity to images of appetizing food show that obesity is characterized by *hyper*-responsiveness in regions typically associated with reward, emotion, and taste processing (e.g., OFC, MPFC, rostral and subgenual anterior cingulate cortex, ventral striatum, insula, and hippocampus), as well as *hypo*-responsiveness in regions typically associated with inhibitory control and executive function (e.g., lateral PFC, dorsal ACC). It is noteworthy that obesity is effectively associated with hyper-responsiveness in regions of the brain that drive food motivation and hypo-responsiveness in those regions that modulate termination of eating.

Emerging research has also begun to characterize the neural correlates of weight loss and weight loss maintenance. At the present time, longitudinal neuroimaging studies comparing pre- and post-treatment activation are scarce. Two fMRI studies have investigated obese participants before and after bariatric surgery, and one fMRI study investigated obese participants before and after a non-surgical behavioral weight loss intervention. In order to investigate food cue-elicited activation, two studies presented stimuli both before and following a period of fasting, whereas the third study presented stimuli only following the fasting period. Across all three studies, one consistent finding was that post-intervention success was associated with decreased brain activity in regions typically involved in taste and reward processing. These studies will be described in greater detail below.

Obese participants undergoing bariatric surgery. To date, two studies have investigated individuals' neural responses to food cues before and following bariatric surgery for weight loss. Ochner et al. (2011) scanned obese women before and after they received Roux-en-Y gastric bypass surgery. Participants were exposed to both visual and auditory stimuli of both high and low energy dense foods, one hour after consuming 250 Kcal of liquid nutrition. From pre- to post-surgery, participants showed decreased activation to high versus low energy dense food stimuli in right DLPFC, dorsal cingulate, lentiform nucleus, and ventral striatum. These changes were accompanied by decreased desire to consume the high energy dense foods. In the second study, Bruce et al. (2011) scanned obese men and women before and after they received adjustable gastric banding surgery. Participants were shown food and animal (nonfood) images before and after eating a standardized 500 Kcal meal. Brain response changes were observed when comparing pre- to post-surgery. At pre-meal, participants showed increased activation (post > pre-surgery) to food versus nonfood images in right middle and superior frontal gyrus

(Brodmann Area 10). At post-meal, participants showed decreased activation (pre > post-surgery) to food versus nonfood images in middle and inferior frontal gyri and in insula/operculum. Further, lower post-surgery activation in inhibitory control regions at post-meal was associated with increased cognitive restraint, decreased hunger, and decreased disinhibited eating. Thus, in both studies, it appears that post-meal response to food pictures was characterized by a decrease from pre to post-surgery in both cognitive control regions (middle and inferior frontal gyri, DLPFC, dorsal ACC) and taste and reward processing regions (insula/operculum, lentiform nucleus, ventral striatum). Bruce et al. (2011) also demonstrated that pre-meal response to food pictures was characterized by an increase from pre to post-surgery in a cognitive control region often implicated in goal maintenance and pursuit (Ramnani & Owen, 2004). Additionally, in both studies, these findings were associated with decreased self-reported desire to eat. It should be noted that male and female participants in Bruce et al.'s (2011) study lost ~25% of initial body weight compared with only female participants in Ochner et al.'s (2011) study who lost ~12% of initial body weight. However, despite study design and weight loss differences, commonalities in neural mechanisms were observed.

Obese participants undergoing behavioral intervention. Although fMRI studies investigating brain changes associated with weight loss following bariatric surgery are rare at the present time, there are more studies investigating neural correlates of weight loss and/or weight loss maintenance achieved through only behavioral methods. The majority of these studies are cross-sectional in design, comparing formerly obese with healthy weight (never obese) individuals. Results indicate that successful weight loss is associated with increased activation in dorsal PFC and DLPFC in response to food stimuli, particularly after eating, as well as with heightened dietary restraint, which is positively correlated with activation in dorsal and DLPFC

(DelParigi et al., 2007; Le et al., 2007; McCaffery et al., 2009). Collectively, these findings indicate that engagement of dorsal and lateral PFC is critical for controlling eating behavior over both the short-term and the long-term.

To date, there is one longitudinal fMRI study examining neural mechanisms of weight loss. Murdaugh et al. (2012) compared a group of overweight and obese men and women enrolled in a behavioral weight loss program with a group of healthy weight control participants. All participants were scanned following an eight hour fast, at the beginning (pre-treatment) and end (post-treatment) of the 12 week weight loss period (controls were scanned 12 weeks after their baseline scanning session). Pre-treatment, the obese group showed greater activation to high calorie food images compared with images of cars in several emotion and reward processing areas, including insula, amygdala, and ACC. Twelve-week percent weight change among obese participants was positively correlated with pre-treatment high calorie food versus control image activation in multiple reward, emotion, and food processing regions, including the nucleus accumbens, insula, and frontal operculum, as well as cognitive control regions including ACC and middle frontal gyrus. In other words, greater baseline activation in these areas predicted weight gain during the twelve-week intervention period. Weight maintenance as measured nine months post-treatment was predicted by a greater decrease in activation in insula and putamen from pre- to post-treatment.

Summary of longitudinal studies. It is striking that both Ochner et al. (2011) and Bruce et al. (2011) reported finding that, from pre- to post-surgery, participants showed decreased activation *after eating* in both cognitive control and reward processing regions, and that these decreases were associated with behavioral indicators of lower food motivation. Additionally, pre-meal activation of Brodmann Area 10 to food versus nonfood images was negatively

correlated with pre-surgery BMI, suggesting that individuals who have higher BMI exhibit lower activation in this cognitive control region in response to visual food cues (Bruce et al., 2011). Similarly, Murdaugh et al. (2012) showed that successful weight loss and maintenance were associated with diminished activation in areas of the brain that process reward, emotion, and taste, suggesting that difficulty with weight loss and maintenance is associated with *hyper*-responsiveness to food cues in these same regions, both before and following behavioral treatment. One question that arises from these findings is whether a better understanding of some of the cognitive and emotional processes involved with eating might help to clarify the relationship between brain function and weight loss outcomes.

Characteristics of Emotional Processing and Differential Effects on Eating Behavior

Findings from the aforementioned longitudinal fMRI studies of weight loss, considered with psychological theories of eating behavior, suggest that the ability to efficiently regulate emotions might be associated with the ability to manage eating behavior, with the end result of successful weight loss and maintenance. Conversely, heightened affect coupled with insufficient regulatory or coping ability represents a liability toward achieving weight management goals. For example, individuals who report difficulty coping with negative life situations, perhaps due to having limited effective emotion regulation skills, are more likely to regain weight that they have successfully lost (Byrne, Cooper, & Fairburn, 2003). Control of eating behavior therefore relies on the efficient regulation of affective states, including specific emotions such as sadness and anger, general mood states such as dysphoria or anhedonia, and other valenced states such as reward anticipation. Thus, further examination of emotion regulation and its relation with eating behavior is warranted.

In order to understand why specific emotion regulation strategies have differential effects

on eating behavior, it is necessary to conceptualize emotion regulation as a process rather than a discrete event. The term *emotion regulation* encompasses a multitude of both automatic and purposeful processes that govern the selection, experience, and expression of emotions, including the ability to maintain or change the trajectory of emotions (Gross, 1998). Emotion regulation is therefore one specific type of self-regulation, which refers to 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). Together, these processes are integral to the selection of long-term goals over short-term gratification, such as in the case of regulating eating behavior in the service of losing or maintaining body weight (Karoly, 1999).

Under some circumstances and for some individuals, processes and strategies of emotion regulation might negatively impact weight loss efforts. According to psychological theories of eating and overeating, heightened levels of negative affect and/or demands on self-regulatory resources leave an individual vulnerable to disinhibited eating (Baumeister & Heatherton, 1996; Heatherton & Baumeister, 1991; Heatherton, Striepe, & Wittenberg, 1998; Muraven et al., 1998; Vohs & Heatherton, 2000). When experiencing negative affect, many individuals engage in rumination, a cognitive process in which thoughts occur repetitively, automatically, and without leading to active problem-solving. In fact, rumination often results in amplification of negative (but not positive) emotions, and is thus indicative of self-regulatory failure. Similarly, although the ability to maintain or enhance positive affect is at first glance an adaptive form of emotional self-regulation (Tugade & Fredrickson, 2007), Yeomans and Coughlin (2009) found that high disinhibition was associated with greater snacking when in a state of induced positive

affect. In sum, both rumination and emotion amplification may contribute to overeating in the short-term and difficulty losing weight in the long-term.

Rumination: A costly emotion regulation strategy. When control of eating behavior is the goal, there are some emotion regulation strategies that are more psychologically costly than others. One costly strategy is rumination, during which an individual focuses attention on negative feelings in the absence of actually changing the situation (Nolen-Hoeksema, 1991). According to the goal progress theory of rumination, an individual engages in rumination when there is a disconnect between the present and desired reality; from this stance, rumination is meant to be adaptive by keeping goal pursuit information readily accessible (Martin, Shrira, & Startup, 2004). However, if the goal is perceived to be unattainable, and if the individual lacks the cognitive flexibility to switch to a different goal, the result is depressive rumination that is counterproductive. As such, rumination could be considered either a poor strategy for emotion regulation or an indication that emotion regulation efforts have failed. In this sense, rumination tends to aggravate and prolong negative affect by contributing to avoidance and impairing problem-solving (Lyubomirsky, Tucker, Caldwell, & Berg, 1999; Nolen-Hoeksema, 1991). Thus, an unintentional effect of rumination might be perpetuation or even amplification of the very negative affect the individual is attempting to decrease.

Naturally, rumination and a ruminative response style have been associated with negative affect (e.g., depression, anxiety), cognitive inflexibility, and eating pathology (Aldao & Nolen-Hoeksema, 2010; Cropley, Michalianou, Pravettoni, & Millward, 2012; Davis & Nolen-Hoeksema, 2000; Kubiak, Vögele, Siering, Schiel, & Weber, 2008; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Additionally, rumination appears to impact particular aspects of eating. For example, Cropley et al. (2012) found that individuals who tended to ruminate over work-

related issues after leaving work also tended to consume more unhealthy foods compared with individuals who did not ruminate about work. Rumination about food, especially when it is triggered by food advertisement, is associated with increased emotional eating (Kemp, Bui, & Grier, 2011). Given the prolific nature of advertisements for unhealthy foods, it is likely that individuals who tend to ruminate are particularly susceptible to consumption of these foods, which interferes with efforts to maintain a healthy weight.

Emotion amplification. Although a significant amount of literature is dedicated to understanding how individuals reduce negative emotion, relatively less has been devoted to examining how individuals increase emotions, including positive ones. Emotion amplification involves processes related to maintaining, intensifying, enhancing, and prolonging emotional experience (Gross, 1998; Hamilton, Karoly, Gallagher, McCurdy, & Karlson, 2009). Amplification may be either explicit or implicit; that is, it may occur voluntarily through effortful behavior or automatically without forethought. Furthermore, amplification may be proactive (e.g., gathering self-regulatory resources to increase positive affect and amp oneself up before an event) or reactive (e.g., using stimuli in the environment to maintain, intensify, enhance, or prolong an emotion experience already in progress).

Laboratory studies in which participants are instructed to up-regulate emotional intensity have yielded results suggesting that intensifying or upregulating emotion is an effortful process. In a test of the self-regulatory strength and depletion model, Muraven, Tice, and Baumeister (1998) found that upregulation and downregulation of emotional responses to a negative film both resulted in increased subjective fatigue as well as poorer performance on a handgrip physical exertion task, in comparison with a no-regulate control group. McRae, Ciesielski, and Gross (2012) instructed participants to use cognitive reappraisal strategies in the service of either

increasing positive affect or decreasing negative affect. They found that increasing positive affect was associated with both greater positive affect but lower decrease in physiological arousal compared with decreasing negative affect. Importantly, results from these two studies suggest that upregulation of emotions is an effortful process that may deplete an individual of energy necessary to engage in subsequent self-regulatory tasks. This might be further compounded if food is the source of energy for emotion amplification.

The Emotion Amplification and Reduction Scales (TEARS). Historically the affect regulation process has been difficult to measure. Thus, TEARS (Hamilton et al., 2009) were designed to capture the fundamental processes of emotional self-regulation and are intended to be valence nonspecific. The purpose of the scales is to measure the extent to which individuals feel they are able to change the trajectory of an emotional response by up-regulating and down-regulating emotion states, regardless of whether these states are positively or negatively valenced. The emotion amplification subscale is characterized by a person's ability to intensify, harness, or prolong emotions or the effects of an emotion; this subscale covaried with positive affect and fatigue and inversely with negative affect and was uncorrelated with depressive symptoms (Hamilton et al., 2009). Emotion reduction is characterized by a person's ability to soften, shorten, stop, or prevent emotions or their effects; this subscale covaried inversely with depressive symptoms, fatigue, and negative affect and was uncorrelated with positive affect (Hamilton et al., 2009). These data indicate that emotion amplification and reduction are related but discriminable processes that should be investigated as such.

Summary

Although many individuals use food to regulate affective states (Antin & Hunt, 2012; Macht & Simons, 2011; Thayer, Newman, & McClain, 1994), this behavior appears to be

particularly deleterious when obese individuals are attempting to losing weight and maintain weight loss (Lowe & Fisher, 1983; Ozier et al., 2008; Thayer et al., 1994). Studies show that negative and positive affect states might be associated with disinhibited eating when they are achieved through psychologically taxing processes such as effortful emotion amplification and/or rumination. Further, there is significant overlap between regions of the brain that process and regulate emotions and those that manage the initiation and termination of eating. Food is an affective stimulus to which the brain responds accordingly; however, brain networks may become hijacked in the service of effortful emotion regulation, leaving limited neural resource available for termination of eating. It is evident that the relations among ability to amplify emotion and tendency toward rumination, brain response to food cues, and weight loss maintenance are poorly understood. Therefore, understanding rumination and emotion amplification at a neural level may help to clarify the mechanisms behind their effect on weight loss outcome.

The Present Study

Significant attention in the literature as well as in popular media has been paid to how people regulate negative emotions, and in particular how people down-regulate negative affect. However, relatively little is known about how people up-regulate emotions, including positive ones. Given the limited understanding of emotion amplification, further investigation of this process, its neural correlates, and its association with weight loss outcomes is a novel area of exploration. Additionally, the effect of rumination on weight loss outcomes has not been explored but nevertheless represents a distinct target for intervention. Preliminary data (manuscript in progress) indicate that both emotion amplification and rumination are negatively correlated with percent weight loss during a diet-based weight loss intervention. Additionally,

preliminary analyses have revealed that emotion amplification is negatively correlated with activation in a prefrontal-striatal network in response to food versus nonfood images after consuming a small meal. Furthermore, percent signal change in these regions positively correlated with percent weight loss after three months, such that greater activation in this prefrontal-striatal network was associated with better weight loss outcome.

The intent of the present study was to build on these findings and address the links among brain function, psychological factors, and weight-related outcomes (see Figure 1). Specifically, fMRI data were acquired both before (BL) and after (3M) participants underwent a three-month behavioral weight loss intervention. Images of appetizing food were presented to participants both before and after eating a small meal, and rumination and emotion amplification were correlated with preprandial and postprandial brain activation. Clusters of activation were then analyzed in follow-up analyses to determine their relations with weight loss outcome at 3M and at the conclusion of the weight maintenance period of the intervention (9M).

Research Aims and Hypotheses

Although it is possible that rumination and emotion amplification are related processes, they were examined separately in the present study. Specifically, for each process, the neural correlates and weight loss outcomes were investigated. It was hypothesized that rumination and emotion amplification would show both common and distinct neural correlates at BL and at 3M. Consistent with the extant literature, food images were hypothesized to elicit response in a prefrontal-striatal-limbic network that included lateral PFC, ACC, MPFC, OFC, insula/operculum, striatum, and hippocampus/parahippocampal gyrus. It was predicted that, in the pre-diet (BL) analysis, no clusters in *a priori* regions would significantly correlate with TEARS Amplification scores. It was also predicted that emotion amplification would be

negatively correlated with activation in the DLPFC and dorsal striatum at the post-meal session. For the pre- to post-diet change (BL to 3M) analysis, it was hypothesized that high emotion amplification would be associated with less post-diet increase in inhibitory regions (lateral PFC, dorsal striatum, dorsal ACC) and with less post-diet decrease in taste and reward processing regions (OFC, MPFC, insula). In the pre-diet (BL) analysis, it was hypothesized that at both pre-meal and post-meal, rumination would be positively correlated with activation in regions responsible for cognitive activity, self-related processing, and emotional processing (lateral PFC, ACC, MPFC, insula, and hippocampal formation including parahippocampal gyrus). For the pre- to post-diet change (BL to 3M) analysis, it was hypothesized that that high rumination would be associated with less post-diet increase in cognitive control regions (lateral PFC, dorsal ACC) and with less post-diet decrease in self and emotion processing regions (OFC, MPFC, insula). Additionally, preliminary findings that both rumination and emotion amplification are inversely correlated with weight loss were expected to be confirmed.

Method

The present study was part of a longitudinal fMRI and diet intervention study investigating brain function predictors of weight loss and weight loss maintenance (NIH R01DK080090). In the longitudinal study, 80 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. Additional height and weight measurements were acquired six and nine months after the baseline appointment.

Participants

Complete datasets that included usable BL and 3M fMRI, BL TEARS Amplification and BL Rumination, and BL, 3M, and 9M weight data were collected from 50 young to middle-aged adults (age range 24−55 years) who were obese (BMI $\geq$ 30 kg/m^2) but otherwise healthy at the start of the study. 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, neurological and 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

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 (Hamilton et al., 2009). Responses are on a 4-point rating scale, ranging from 1 (*not at all true of me*) to 4 (*very true for me*). For this investigation, only the 9 items from the TEARS Amplification subscale were used and the modified scale showed good internal consistency (α=.87).

Response Styles Questionnaire (RSQ). The tendency to engage in rumination was measured by the RSQ (Nolen-Hoeksema, Morrow, & Fredrickson, 1993), a 32-item self-report assessment of people's cognitive and behavioral responses to sad or depressed mood. Participants are instructed to select from a list of thoughts and behaviors those that generally apply to them when they are in a sad or depressed mood. Thoughts and behaviors are classified as either ruminative responses or distracting responses. Although reliability over time varied depending on the length of the follow-up period (Kasch, Klein, & Lara, 2001; Nolen-Hoeksema, Morrow, & Fredrickson, 1993), the RSQ has nevertheless shown good construct validity through significant correlation with broad dimensions of negative affect (depression, trait anxiety, and neuroticism), as well as discriminant validity from specific (spider) phobia and aggression (Roelefs, Muris, Huibers, Peeters, & Arntz, 2006). For this investigation, only the 16 thoughts and behaviors classified as ruminative responses were used. In the current sample, the Rumination subscale of the RSQ showed acceptable internal reliability (α=.75).

Study 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 of which 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 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 capitalize on 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 took 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 contribution to the study.

fMRI Methods

fMRI cognitive activation paradigm. The experimental paradigm has been used in research studies with children (Bruce et al., 2010; Holsen et al., 2005) and adults (Bruce et al., 2011; Martin et al., 2009). Participants viewed food, animal (nonfood), and blurry images during two scanning sessions: (a) after fasting for four hours (pre-meal) and (b) 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 an 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 ($p = 0.55$) or arousal ($p = 0.96$). 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 2. Each functional scan involved three repetitions of each block of each stimulus condition type (i.e., food, animal), which alternated between blocks of blurred images. Visual stimuli were generated with Presentation (Neurobehavioral Systems, Inc., Albany, CA) on a 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. Participants were instructed to remember as many food and animal images as they could while in the scanner. 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 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 × 100 mm, matrix = 192 × 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 40° angle (repetition time/echo time [TR/TE] = 3000/30 ms, flip angle = 90°, FOV = 220 mm, matrix = 64 × 64, slice thickness = 3 mm, 0.5 mm skip, in-plane resolution = 3 × 3 mm, 130 data points).

Method to minimize susceptibility artifact. To optimize signal in ventromedial prefrontal regions by minimizing susceptibility artifact from air sinuses and to standardize head positioning across individuals, BOLD sequences were acquired in oblique slices at a 40° angle. Additionally, 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.

Data Analysis

Functional MRI data were 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 3 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.

Whole brain voxelwise 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 random effects general linear model (GLM), allowing for multiple predictors (e.g., food versus nonfood, pre- versus post-diet) 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. Motion correction parameters were included in the GLM as regressors of no interest. In order to minimize voxels and therefore multiple comparisons, a brain mask was applied to the GLM analysis. The resulting GLM was overlaid with a contrast of food greater than nonfood, and separate beta maps were created and saved for each participant. This multi-subject beta map set containing beta values for the difference between activation to food and nonfood images (F−NF) was then entered into BrainVoyager's ANCOVA function. TEARS Amplification and Rumination scores were entered separately as the covariates, with no between or within-subjects factors. Resulting correlation maps were overlaid on a three-dimensional rendering of a skull-stripped group average brain. To correct for multiple comparisons, a cluster-level threshold was applied (Forman et al., 1995). Based on BrainVoyager's cluster level threshold estimator plugin, voxel values were considered significant if the activation survived a statistical threshold of $\alpha<.05$, corrected for whole brain. This procedure was performed separately for pre-meal and post-meal analyses, as well as for

Amplification and Rumination. Clusters of activation were identified and verified by mapping Talairach coordinates onto two atlases (Mai, Paxinos, & Voss, 2008; Talairach & Tournoux, 1988).

Exploratory longitudinal analyses. In order to determine whether emotion amplification or rumination is associated with changes in brain function over the course of a behavioral weight loss intervention, exploratory analyses were conducted. GLM files were created for pre-meal and post-meal and overlaid with the contrast (Food BL − Nonfood BL) − (Food 3M − Nonfood 3M), representing the interaction of timepoint (BL, 3M) and stimulus type (F, NF). Separate subject beta maps of these contrast values were then created, saved, and entered into BrainVoyager's ANCOVA function. The rest of the analysis procedure matched those conducted for single timepoint analyses.

It was hypothesized that emotion amplification would be associated with less post-diet increase in inhibitory regions (lateral PFC, dorsal striatum, dorsal ACC) and with less post-diet decrease in taste and reward processing regions (OFC, MPFC, insula). Additionally, it was hypothesized that rumination would be associated with less post-diet increase in cognitive control regions (lateral PFC, dorsal ACC) and with less post-diet decrease in self and emotion processing regions (OFC, MPFC, insula).

Follow-up region of interest (ROI) analyses. Based on studies of emotion regulation, as well as those investigating food motivation (e.g., Bruce et al., 2010; Martin et al., 2009), several regions were considered *a priori* regions of interest, including lateral PFC, ACC, MPFC, OFC, insula/operculum, striatum, and hippocampus/parahippocampal gyrus. For *a priori* regions that showed a significant correlation between Amplification and F−NF beta value, or between Rumination and F−NF beta value, the mean percent signal change from all voxels within

activated clusters was extracted. For quality control purposes and to check for the presence of outliers, scatterplots were created depicting the extracted percent signal change values and Amplification/Rumination scores. Additionally, percent signal change values in *a priori* regions of interest were correlated with percent weight loss (BL to 3M and 3M to 9M separately) in follow-up analyses.

Results

Participant demographics and other characteristics are summarized in Table 1. Out of the 50 participants, 34 (68%) were female, 38 (76%) were Caucasian, and 47 (94%) had attended at least some college. Participants' age range was 24 to 55 years (M = 39.02, SD = 7.99). BMI range was 30.45 to 43.58 kg/m^2 (M = 35.16, SD = 3.37). During the weight loss phase of the study (BL to 3M), participants lost 2.81 to 23.72% of baseline body weight (M = 11.09, SD = 4.83). During the weight maintenance phase of the study (3M to 9M), participants' results ranged from gaining 11.04% to losing 16.80% of 3M body weight (M = 1.04% weight gain, SD = 5.22% weight gain). Neither TEARS Amplification nor Rumination scores were significantly correlated with either percent weight change variable, nor were they significantly correlated with each other (see Table 2). As shown in Table 3, women compared with men had higher scores on the WASI (M = 115.21 vs 108.94, p=.034), lost less weight from BL to 3M (M = 9.56% weight loss vs 14.36% weight loss, p=.006), and endorsed more tendency to ruminate (M = 3.56 vs 1.38, p=.006).

Cross-Sectional Neuroimaging Results

Neural correlates of emotion amplification. The clusters found to represent a significant correlation of TEARS Amplification score with the difference in activation to food versus nonfood images (F−NF beta value difference) are listed in Table 4.

Pre-meal BL. At pre-meal at baseline, Amplification score was not significantly correlated with the difference in activation to food versus nonfood images in any of the *a priori* regions. For *post hoc* regions, the difference between food and nonfood activation was inversely correlated with Amplification score in bilateral BA 39, specifically the right angular gyrus and the left middle temporal gyrus.

Post-meal BL. At post-meal at baseline, Amplification score was not significantly correlated with the difference in activation to food versus nonfood images in any of the *a priori* regions. For *post hoc* regions, the difference between food and nonfood activation was inversely correlated with Amplification score in right superior and middle frontal gyrus (both BA 6), as well as in right inferior parietal lobule.

Pre-meal 3M. At pre-meal at 3M follow-up, Amplification score was not significantly correlated with the difference in activation to food versus nonfood images in any of the *a priori* regions. For *post hoc* regions, Amplification score was inversely correlated with activation in left inferior occipital gyrus.

Post-meal 3M. At post-meal at 3M follow-up, Amplification score was not significantly correlated with the difference in activation to food versus nonfood images in any of the *a priori* regions. For *post hoc* regions, Amplification score was inversely correlated with activation in the right inferior occipital gyrus.

Neural correlates of rumination. Visual inspection of the scatterplots created from the correlation of percent signal change and Rumination score revealed that one participant had a score of 15, which was more than four standard deviations above the mean score. Therefore, clusters of activation that were clearly driven (and not simply enhanced) by the presence of the outlier were discarded. The remaining clusters found to represent a significant correlation of

Rumination score with the difference in activation to food versus nonfood images (F−NF beta value difference) are listed in Table 5.

Pre-meal BL. At pre-meal at baseline, Rumination score was positively correlated with activation in the *a priori* region of left middle frontal gyrus (BA 9; *x, y, z* = –48, 26, 31) and right superior frontal gyrus (BA 11; *x, y, z* = 18, 62, –15). For *post hoc* regions, positive correlations were found in left precentral gyrus, superior and inferior parietal lobule, and cerebellum, right superior frontal gyrus and cuneus, and bilateral cerebellum. Inverse correlations were observed in right inferior parietal lobule and fusiform gyrus.

Post-meal BL. At post-meal at baseline, Rumination score was inversely correlated with activation in *a priori* regions of left inferior frontal gyrus (BA 47; *x, y, z* = –39, 26, –8; BA 45; *x, y, z* = –48, 17, 4), left superior frontal gyrus (BA 10; *x, y, z* = −27, 50, 22), and right caudate nucleus (*x, y, z* = 9, −1, 16). For *post hoc* regions, inverse correlations were found in left superior and middle frontal gyrus, right precuneus, inferior parietal lobule, and middle/inferior temporal gyrus, left occipital gyrus, and right cerebellum and pons.

Pre-meal 3M. At pre-meal at 3M follow-up, Rumination score was significantly correlated with activation to food versus nonfood images in the *a priori* region of left middle frontal gyrus (BA 9; *x, y, z* = –27, 38, 28). For *post hoc* regions, inverse correlations were found in left precuneus and superior temporal gyrus, right middle temporal gyrus, right cerebellum, and left brainstem.

Post-meal 3M. At post-meal at 3M follow-up, Rumination score was positively correlated with activation in *a priori* regions of bilateral middle frontal gyrus (BA 9; *x, y, z* = −33, 20, 28; BA 10; *x, y, z* = 33, 47, 19 and –36, 44, 10), right inferior frontal gyrus (BA 45; *x, y, z* = 51, 23, 10; BA 44; *x, y, z* = 42, 8, 22), bilateral insula (*x, y, z* = 33, 14, −5; −39, 17, 4; −39,

−1, 10), right parahippocampal gyrus (BA 35; *x, y, z* = 24, −25, −17), and left hippocampus (*x, y, z* = −33, −19, −11). For *post hoc* regions, positive correlations were found in right superior frontal gyrus, precuneus, and middle temporal gyrus, as well as bilateral superior temporal gyrus and left cerebellum.

Exploratory Longitudinal Analyses

Exploratory analyses were conducted to correlate Amplification and Rumination scores with the contrast value of the interaction of timepoint (BL, 3M) and stimulus type (food, nonfood). In this analysis, a positive contrast value indicates BL>3M (decreased activation) for the food versus nonfood beta difference, whereas a negative contrast value indicates 3M>BL (increased activation) for the food versus nonfood beta difference. For example, a *positive* correlation with Amplification indicates that higher Amplification score was associated with *decreased* activation from pre- to post-diet, and an *inverse* correlation indicates that higher Amplification score was associated with *increased* activation from pre- to post-diet.

Correlation with contrast values – Amplification. The clusters found to represent a significant correlation between Amplification score and the contrast value from the interaction of timepoint (BL, 3M) and stimulus type (food, nonfood) are listed in Table 6. At pre-meal, a positive correlation was found in the *a priori* region of right superior frontal gyrus (BA 9; *x, y, z* = 21, 53, 51). At post-meal, *post hoc* regions showing significant inverse correlations were right superior frontal gyrus (BA 6) and right superior temporal gyrus.

Amplification follow-up ROI analysis. No *a priori* regions from either the primary (cross-sectional) or the exploratory (longitudinal) neuroimaging results were found to exhibit a significant correlation with percent weight change either from BL to 3M or from 3M to 9M.

Correlation with contrast values – Rumination. The clusters found to represent a

significant correlation between Rumination score and the contrast value from the interaction of timepoint (BL, 3M) and stimulus type (food, nonfood) are listed in Table 7.

At pre-meal, a positive correlation was found in the *a priori* regions of left middle frontal gyrus (BA 9; *x, y, z* = –48, 26, 34) and left anterior cingulate cortex (BA 24; *x, y, z* = –3, –10, 37). *Post hoc* regions showing positive correlations included an additional cluster in the left middle frontal gyrus, as well as left precentral gyrus, precuneus, and parietal cortex.

At post-meal, significant inverse correlations were found bilaterally in *a priori* regions of superior frontal gyrus (BA 10; *x, y, z* = –24, 50, 25; BA 9; *x, y, z* = 21, 50, 28), inferior frontal gyrus (BA 9; *x, y, z* = –51, 11, 28; BA 45; *x, y, z* = 54, 26, 4), and parahippocampal gyrus (BA 36; *x, y, z* = –24, –31, –17 and 24, –28, –17), as well as in right anterior cingulate cortex (BA 32; *x, y, z* = 12, 35, 10) and left amygdala (*x, y, z* = –21, –7, –8). *Post hoc* regions showing inverse correlations were bilateral medial frontal gyrus, left precentral gyrus, posterior cingulate gyrus, precuneus, superior parietal lobule, and middle temporal gyrus, and right postcentral gyrus, superior temporal gyrus, and lingual gyrus, as well as bilateral cerebellum.

Rumination follow-up ROI analysis – weight change BL to 3M. Percent weight change BL to 3M was found to be inversely correlated with activation in the left superior frontal gyrus (*x, y, z* = –27, 50, 22; *r* = –.301; *p* = .033) and the right caudate (*x, y, z* = 9, –1, 16; *r* = –.281; *p* = .048) at the BL post-meal session, such that greater activation (F>NF) in these regions was prospectively correlated with more weight loss (see Figure 2 and 3).

Rumination follow-up ROI analysis – weight change 3M to 9M. No *a priori* regions from either the pre-meal or the post-meal interaction correlation showed a significant correlation with weight change from 3M to 9M.

Discussion

This study investigated how the psychological variables emotion amplification and rumination are related to brain activation to food images in an obese diet-seeking sample, and how these factors are related to an individual's ability to lose weight and maintain weight loss. Contrary to hypotheses, neither Amplification nor Rumination score was significantly correlated with either short-term (BL to 3M) or long-term (3M to 9M) weight change, suggesting that neither variable itself is a significant determinant of weight management.

Neural Correlates of Emotion Amplification in the Context of Food Motivation and Weight Loss

Pre-diet (BL). It was anticipated that no clusters in *a priori* regions would survive the pre-meal BL analysis of correlations with TEARS Amplification after the cluster-size threshold was applied. This was supported in that, at pre-meal, only bilateral BA 39 (right angular gyrus and left middle temporal gyrus) showed significant, albeit negative, correlations with TEARS Amplification score. BA 39 encompasses the angular gyrus, which plays a role in action awareness (Farrer et al., 2008), as well as caudal aspects of the temporal cortex, which are involved in semantic memory, specifically for information about objects (Binder, Desai, Graves, & Conant, 2009). Additionally, Binder et al. (2009) suggested that the angular gyrus contributes to semantic processing by integrating complex information for the purposes of problem-solving and planning.

It was also anticipated that emotion amplification would be negatively correlated with activation in the DLPFC and dorsal striatum at the post-meal session, a hypothesis that was not supported. Because these hypotheses were driven by results from preliminary analyses with a larger sample (n = 75), the present null findings can likely be attributed to the significantly

smaller sample size. Among *post hoc* findings, inverse correlations were found in right superior frontal gyrus corresponding to the presupplementary motor area (pre-SMA), right middle frontal gyrus corresponding to the dorsal premotor cortex, and right inferior parietal lobule. The pre-SMA is implicated in attention to intended self-initiated movement (as opposed to stimulus-driven reactions) and in response selection, including both inhibition of inappropriate behavior as well as selection of appropriate responses (Hoffstaedter, Grefkes, Zilles, & Eickoff, 2013; Lau, Rogers, Haggard, & Passingham, 2004; Mostofsky & Simmonds, 2008). The dorsal premotor cortex appears to modulate execution of chosen movement (Hoffstaedter et al., 2013), which may explain why this region has shown increased activation in response to high food motivation conditions, such as external food sensitivity (Passamonti et al., 2009) and highly hedonic food images (Cornier, Von Kaenel, Bessesen, & Tregellas, 2007). The right inferior parietal lobule is part of a fronto-parietal network that modulates sustained attention and salience detection, thereby facilitating behavioral flexibility (Hopfinger, Buonocore, & Mangun, 2000; Singh-Curry & Husain, 2009).

Collectively, the present findings indicate that differences in emotion amplification ability are associated with differential brain response to food images in a sample of diet-seeking obese adults. Specifically, individuals who self-report as low compared with high amplifiers appear to respond to appetitive cues with greater activation in areas of the brain that have been implicated in self-awareness, intentional movement, and action preparation. These differences may influence how semantic or contextual information is processed in the service of behavioral self-regulation.

Post-diet (3M). At post-diet, TEARS Amplification score was found to be inversely correlated with pre-meal activation (F−NF) in left inferior occipital gyrus and with post-meal

activation (F−NF) in right inferior occipital gyrus. These results are consistent with a meta-analysis of studies of visual food cue processing, which revealed that the lateral occipital complex is commonly activated in comparisons of food versus nonfood images (van der Laan, de Ridder, Viergever, & Smeets, 2011). The authors suggested that, because food and nonfood images were generally matched within studies for valence and arousal levels, the likely explanation for greater visual cortex activation to food images is the emotional and salient nature of food pictures. Thus, differences in emotion amplification appear to be associated with differential responding in regions of the brain commonly recruited for visual processing. However, because Amplification score and weight change were not significantly correlated in the present analysis, it is unclear whether this differential responding negatively impacts efforts to manage eating behavior and weight.

Pre- to post-diet change (BL−3M). It was hypothesized that high emotion amplification would be associated with less post-diet increase in inhibitory regions (lateral PFC, dorsal striatum, dorsal ACC) and with less post-diet decrease in taste and reward processing regions (OFC, MPFC, insula). These hypotheses were partially supported. In the exploratory longitudinal analysis, Amplification score showed a positive correlation with the pre-meal contrast value in right superior frontal gyrus corresponding to the DLPFC, and with the post-meal contrast value in the right superior frontal gyrus corresponding to pre-SMA as well as right superior temporal gyrus. It is important to note that, for the pre-SMA, the maximally activated voxel for this contrast is very close to that for the cross-sectional baseline post-meal (F−NF) contrast. Therefore, according to the correlation, higher Amplification score was associated with a decrease in activation (food versus nonfood images) in the pre-SMA from BL to 3M. Follow-up inspection of the activation pattern of the DLPFC revealed that whereas the F−NF contrast

was not correlated with Amplification score at baseline (pre-diet), the same contrast was inversely correlated with Amplification score at 3M (post-diet), such that higher Amplification was associated with less activation to food versus nonfood images at 3M. In other words, high Amplification was associated with a greater decrease in food cue-elicited response in the DLPFC from BL to 3M.

The DLPFC plays an important role in self-regulation. For example, a recent neuroimaging study found the left DLPFC to be associated with resolution of conflict in the service of choosing the situationally appropriate course of action (Wittfoth, Schardt, Fahle, & Herrmann, 2009). Furthermore, the DLPFC shows activation during tasks of effortful emotion regulation (e.g., Goldin, McRae, Ramel, & Gross, 2008; McRae et al., 2010; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2004; Phan et al., 2005). With regard to food motivation and eating behavior, the DLPFC has been associated with heightened dietary restraint (Burger & Stice, 2011; Le et al., 2007), weight loss success (McCaffery et al., 2009), and cognitive control of food-related reward (Hollmann et al., 2011). Additionally, Kringelbach, de Araujo, and Rolls (2004) suggested that the left DLPFC might be involved in taste processing, particularly in relation to the generation of cognitive and behavioral processes related to the initiation and cessation of eating. Considered together, the extant literature and the present findings suggest that high ability and tendency to amplify emotions may be associated with hypo-activation in brain regions known to be involved in self-regulation.

In summary, individual differences in emotion amplification were associated with differential brain response to food images in areas of the brain typically involved with modulation of attention, self-awareness, and action preparation. In these regions, higher levels of amplification were correlated with relatively less activation to food than nonfood images post-

meal prior to starting the diet. Additionally, higher levels of amplification were associated with decreased pre-meal DLPFC activation from before to after the diet.

Neural Correlates of Rumination in the Context of Food Motivation and Weight Loss

It was hypothesized that at both pre-meal and post-meal, rumination would be positively correlated with activation in regions responsible for cognitive activity, self-related processing, and emotional processing (lateral PFC, ACC, MPFC, insula, and hippocampal formation including parahippocampal gyrus). These hypotheses were somewhat supported, in that significant albeit inverse correlations were found in lateral PFC at both pre-diet and post-diet timepoints. Consistent with hypotheses, activation in insula and hippocampal formation showed positive correlations with Rumination score at post-diet. Change in activation from pre-diet to post-diet was correlated with activation in lateral PFC, ACC, MPFC, and hippocampal formation. Unanticipated findings included correlations with caudate nucleus and amygdala. Findings will be discussed sequentially.

Pre-diet (BL). At pre-meal pre-diet, Rumination score was positively correlated with activation (F>NF) in left middle frontal gyrus corresponding to the DLPFC, as well as in the right superior frontal gyrus corresponding to the anterior orbitofrontal cortex (OFC). Similar regions were found to be activated more in depressed than non-depressed control participants during a rumination (compared with distraction) task (Cooney, Joormann, Eugène, Dennis, & Gotlib, 2010). Furthermore, Spielberg et al. (2012) found left DLPFC and right anterior OFC to be functionally connected as part of a network responsible for maintenance of goal pursuit. The anterior OFC has shown increased activation in response to both anticipation and receipt of taste reward (O'Doherty, Deichmann, Critchley, & Dolan, 2002). These findings suggest that, before eating, individuals who report higher levels of rumination display hyper-responsiveness in brain

regions that modulate behavioral and emotional self-regulation and contribute to evaluation of reward stimuli.

At post-meal pre-diet, Rumination score was inversely correlated with activation (F>NF) in left ventrolateral PFC (VLPFC) and DLPFC. VLPFC has shown increased activation during emotion regulation tasks, including emotion suppression and cognitive reappraisal (Goldin et al., 2008; Ochsner et al., 2004; Phan et al., 2005). As depicted in Figure 2, signal change in the DLPFC and BA 10 also showed an inverse correlation with percent weight change BL to 3M, such that greater post-meal activation to food versus nonfood at pre-diet (a) was associated with lower rumination score and (b) predicted more weight loss during the diet intervention. The more lateral portions of BA 10, such as those found in these analyses, have been associated with maintenance of internally generated thought (Burgess, Scott, & Frith, 2003), as well as with planning-related processes (Wagner, Koch, Reichenbach, Sauer, & Schlösser, 2006) and complex problem-solving (Strange, Henson, Friston, & Dolan, 2001). These findings suggest that higher levels of rumination are associated with less activation to food than nonfood images in cognitive control regions after eating, which is in turn prospectively associated with poorer weight loss outcome.

Unexpectedly, the caudate nucleus showed an inverse correlation between Rumination score and activation to food versus nonfood images, as well as an inverse correlation with weight change during the diet (see Figure 3). Specifically, lower Rumination score was correlated with greater activation in the caudate; greater activation in the caudate (i.e., F>NF) was prospectively associated with more weight loss, whereas less activation in the caudate (i.e., F<NF) was prospectively associated with less weight loss. The caudate is part of the striatum, and the cluster of activation found in the present analysis falls in the dorsal extent, which has been

associated with various aspects of behavioral self-regulation (Featherstone & McDonald, 2004; Seger & Cincotta, 2006; Yin, Knowlton, & Balleine, 2004). Additionally, the dorsal striatum appears to play a role in decision-making by maintaining information about contingencies (O'Doherty et al., 2004). Studies of food motivation and obesity have implicated the caudate in evaluation and anticipation of potential reward, with findings generally showing that obese relative to healthy weight exhibited greater food picture-elicited activation in the caudate, particularly after eating and when the pictures are of high calorie foods (Dimitropoulos et al., 2012; Nummenmaa et al., 2012; Rothemund et al., 2007). Additionally, Pelchat et al. (2004) found greater caudate activity to be associated with greater food craving. Taken together, the caudate appears to play a role in the modulation of goal-directed behavior. Findings from the present study suggest that differences in the tendency to ruminate are also associated with differential brain function in regions that contribute to flexible behavioral self-regulation.

Post-diet (3M). At pre-meal post-diet, Rumination score was inversely correlated with activation (F>NF) in the left DLPFC. This contrasts with the finding that at pre-meal prior to beginning the diet, Rumination score showed a positive correlation with activation in this same region. Thus, whereas greater tendency to ruminate was associated with *hyper*-responsiveness in regions responsible for cognitive and behavioral self-regulation pre-diet, it was associated with *hypo*-responsiveness in the same regions post-diet.

At post-meal post-diet, several original hypotheses were supported. Rumination score was positively correlated with activation to food versus nonfood images in bilateral middle frontal gyrus corresponding to the DLPFC, right inferior frontal gyrus corresponding to lateral PFC, bilateral insula, right parahippocampal gyrus, and left hippocampus. Thus, in each of these regions, higher Rumination score was associated with more activation to food versus nonfood

images. These findings are consistent with the extant literature indicating that rumination is associated, in general, with differential brain activity during fMRI cognitive paradigms (Ray et al., 2005), and specifically with greater activation in regions involved in both cognitive and emotional/interoceptive processes (Cooney et al., 2010; Thomas et al., 2011). Additionally, Pelchat et al. (2004) found that the hippocampus, insula, and caudate were all associated with food craving, a process that might be cognitively similar to rumination (i.e., difficulty disengaging from thoughts of the craved food or a particular line of thinking).

The three insula clusters (two on the left and one on the right) showed a positive correlation between activation (F>NF) and Rumination score at post-meal post-diet. The insular cortex is a commonly activated region in fMRI studies due to its involvement in processes across a variety of domains, to include olfactory/gustatory, social/emotional, sensorimotor, and cognitive (Cauda et al., 2012; Kurth, Zilles, Fox, Laird, & Eickhoff, 2010; Simmons et al., 2012). Therefore, it is not surprising that the insula represented a significant correlation between a cognitive-emotional response style and food-cue elicited brain activity. Several studies have found that obese compared with healthy weight individuals exhibit greater food cue-elicited activation in the insula (e.g., Rothemund et al., 2007; Scharmüller, Übel, Ebner, & Schienle, 2012; Stoeckel et al., 2008), and activation in the insula has been correlated with appetite (Porubská, Veit, Preissl, Fritsche, & Birbaumer, 2006). Thus, individuals with a ruminative response style may experience difficulty shutting down the emotional and appetitive response to food images, even after eating. Although the insula may be structurally and functionally parceled into subregions "specialized" for particular domains, it is striking that the three clusters of activation in the present analysis represent multiple subregions.

At post-meal post-diet, an additional set of *a priori* regions, the hippocampus and

parahippocampal gyrus, showed a positive correlation between Rumination score and activation to food versus nonfood images. Both the hippocampus and the parahippocampal gyrus are part of a medial temporal lobe network that modulates various aspects of memory, including declarative memory (Eichenbaum, 2004), autobiographical memory (Fink et al., 1996) and the association of learned information (Henke, Weber, Kneifel, Wieser, & Buck, 1999). Additionally, these regions tend to show differential patterns of activation to changing conditions of food motivation. For example, Martin et al. (2009) found that obese versus healthy weight participants showed greater activation to food images in the parahippocampal gyrus at pre-meal and the hippocampus at post-meal. Gautier et al. (2001) found that satiety was associated with decreased activation in the parahippocampal gyrus. Additionally, Bohon, Stice, and Spoor (2009) found that female emotional eaters showed greater activation in the parahippocampal gyrus during anticipation of food in a negative mood. Taken together, studies indicate that the hippocampus and parahippocampal gyrus are activated under conditions of high food motivation, and that negative affect may exaggerate this effect. Thus, the positive correlation between activation in these regions and rumination score might reflect among high ruminators difficulty shutting down the desire to eat, even (a) after eating and (b) following a weight loss intervention.

Pre- to post-diet change (BL−3M). It was hypothesized that high rumination would be associated with less post-diet increase in cognitive control regions (lateral PFC, dorsal ACC) and with less post-diet decrease in self and emotion processing regions (OFC, MPFC, insula). These hypotheses were partially supported. In the exploratory longitudinal analyses, Rumination score was positively correlated with the pre-meal contrast value in left middle frontal gyrus corresponding to the DLPFC. In other words, from pre- to post-diet, high rumination was associated with decreased activation to food versus nonfood images in the pre-meal condition.

At post-meal, Rumination score was inversely correlated with the interaction contrast value in bilateral superior frontal gyrus corresponding to DLPFC, bilateral inferior frontal gyrus, corresponding to VLPFC, right anterior cingulate cortex (ACC), bilateral parahippocampal gyrus, and left amygdala. Thus, higher rumination score was correlated with increased activation (food versus nonfood) in these regions from pre to post-diet. One role for the ACC is to modulate decision-making by focusing attention on reward contingencies (Bush et al., 2002). The cluster of activation in the ACC has shown greater activation among obese relative to healthy weight participants viewing high calorie food images (Brooks, Cedernaes, & Schiöth, 2013; Stoeckel et al., 2008). The amygdala plays an integral role in both positive and negative emotion processing (Hamann, Ely, Hoffmann, & Kilts, 2002), as well as in encoding information into memory (Hamann, Ely, Grafton, & Kilts, 1999; McGaugh, 2004). Activation of the amygdala during memory encoding may result in otherwise neutral stimuli becoming valenced, particularly in the case of individuals who are vulnerable to depression (van Eijndhoven et al., 2011). Additionally, the amygdala is active during processing of food stimuli (Siep et al., 2009; van der Laan et al., 2011), particularly when food is felt to be highly rewarding (Beaver et al., 2006; Goldstone et al., 2009). It could be that among individuals who tend to ruminate, attempts to diet or to control eating behavior result in food becoming even more salient and more difficult to resist.

In summary, individual differences in rumination were associated primarily with differential brain response to food images in areas of the brain typically involved with cognitive control, interoception, self-related processing, and emotion processing. The present findings also suggest that individuals who tend to ruminate show greater food cue-elicited response in both the primary gustatory cortex as well as in regions of the brain implicated in emotional and cognitive

regulation. Additionally, at post-meal pre-diet, activation in brain regions involved with behavioral self-regulation was inversely correlated with Rumination score and prospectively associated with more weight loss during the initial three month weight loss period. Thus, tendency to ruminate may interfere weight management efforts if it is also associated with limited flexibility in self-regulation.

General Discussion

Eating is inherently an emotional experience, and it is difficult to disentangle homeostatic and hedonic aspects of food motivation. Although the current society could be considered obesogenic, not everyone meets criteria for obesity, and not everyone has difficulty managing eating behavior in the service of maintaining appropriate body weight. Additionally, many people engage in unhealthy eating behaviors, including using food to regulate emotions. Thus, individual variability must play a role: psychological or personality-related factors inevitably influence a person's relationship with food. Results from the present investigation suggest that emotion amplification and rumination are not in and of themselves a liability in weight loss efforts. However, these factors are associated with brain response patterns to food images that increase vulnerability to maladaptive eating patterns, which could result in difficulty managing weight. In particular, engagement of brain areas implicated in behavioral self-regulation (i.e., DLPFC and caudate) may be a determining factor for whether rumination is detrimental to weight management efforts.

One outstanding question is under what circumstances emotion amplification and/or rumination might contribute to overeating or unhealthy eating behavior. Because there is limited research addressing this association, it would be fruitful to examine and discuss potential aspects of each psychological characteristic that could influence how a person responds to food cues. In

the following sections, neuroimaging findings from the present study will be integrated with the extant behavioral literature on emotions and eating.

Emotion amplification. In the present study, TEARS Amplification score was associated with decreased activation from pre- to post-diet in the DLPFC, suggesting that some aspects of emotion amplification negatively impact brain networks supporting self-regulation. One possibility is that the end emotion state, either increased positive or negative affect, is itself associated with increased eating. There is considerable research evidence to support this hypothesis. For example, experimentally induced positive mood has been associated with increased consumption of high calorie foods among emotional eaters (Bongers, Jansen, Havermans, Roefs, & Nederkoorn, 2013; Bongers, Jansen, Houben, & Roefs, 2013), disinhibited eaters (Bongers, Jansen, Houben, & Roefs, 2013; Turner, Luszczynska, Warner, & Schwarzer, 2010), healthy weight individuals (Evers et al., 2013), and obese individuals (Udo et al., 2012). Likewise, negative affect was associated with increased food consumption following a negative mood induction and food exposure condition (Jansen et al., 2008; Loxton, Dawe, & Cahill, 2011). Thus, it is possible that among high amplifiers, emotional arousal itself, regardless of valence, may contribute to eating and potentially overeating (Evers et al., 2013). Although mood was not manipulated in the present study, it is possible that decreased activation of inhibitory brain regions upon exposure to food cues is a neural mechanism of the relationship between affective state and food consumption.

A second aspect of emotion amplification that may interfere with effective self-regulation is the process of amplification itself, which has been shown to be effortful and psychologically as well as physically taxing (McRae, Ciesielski, & Gross, 2012; Muraven, Tice, & Baumeister, 1998). There are many studies demonstrating that self-regulatory resource depletion is

associated with disinhibited eating (see Muraven & Baumeister, 2000, for a review). To the extent that the demands of undergoing a behavioral weight management intervention deplete self-regulatory resources, a similar process likely occurred among participants in the present study. Among high amplifiers, decreased DLPFC activation from pre- to post-diet might reflect this self-regulatory depletion and provide a neural mechanism for the disinhibitory effects of appetizing food images. At the present time, research studies investigating emotion amplification are relatively scarce, and no studies have addressed the relations among effortful emotion amplification (versus a more passive mood induction), brain function, and food consumption. Further research in this area could clarify the unique contributions of the amplification process and the end emotional state to (a) brain response to food cues and (b) eating behavior.

Emotion amplification might also negatively impact self-regulation via the rewarding properties inherent in and associated with food and eating. Elements of food such as the sensory attributes, experienced and anticipated consequences, personal and cultural meanings, and actions of associated agents collectively form the experience of eating (Desmet & Schifferstein, 2008). Food consumption is often associated with heightened affect, such as during celebrations or other social gatherings. This is reflected at a neural level: the visual presentation of food images elicits activity in neural networks that process both properties of the food stimuli themselves, as well as emotional and cognitive associations that have formed in relation to food (Simmons et al., 2005). The hedonic effects and associations of food cues might be especially influential for high relative to low amplifiers, which could lead to greater difficulty effectively regulating food intake both on a day-to-day basis and in situations where energy dense food is present. Thus, it may be critical for high amplifiers in particular to spend additional time

learning how to manage the rewarding aspects of food and food-related occasions.

Rumination. In the current study, greater tendency to ruminate was associated with greater pre-diet but lower post-diet activation in regions of the brain implicated in cognitive, emotional, and behavioral self-regulation. Additionally, individuals who endorsed higher levels of rumination showed greater post-diet activation in various limbic and paralimbic brain regions implicated in emotional processing, memory formation, and food motivation. Rumination also correlated inversely with pre-diet activation in a network that modulates flexible behavioral self-regulation, which in turn was associated prospectively with greater weight loss. Collectively, these results suggest that the tendency to ruminate might be associated with psychological and neural processes that interfere with attempts to manage eating behavior and weight.

Rumination is a core feature of depression, such that individuals with a ruminative response style experience longer depressive episodes (Aldao & Nolen-Hoeksema, 2010; Bagby, Rector, Bacchiochi, & McBride, 2004; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Individuals who experience depression may be more likely to overeat (Ouwens, van Strien, & van Leeuwe, 2009), a relationship that is exaggerated by the presence of obesity (Murphy et al., 2009), especially among women (Stunkard, Faith, & Allison, 2003). Although participants in the present study did not endorse clinically significant levels of depressive symptoms, there is some evidence that "everyday" negative affect arising from daily hassles and work-related rumination is associated with unhealthy food consumption (Cropley et al., 2011; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008). Because rumination is, by definition, distinct from active problem-solving, it tends to amplify and/or prolong negative affect by failing to resolve the distressing circumstances. In this case, individuals are likely to turn to food consumption as a form of distraction and avoidance, initiating and perpetuating a learning process that eating

improves affect (Byrne et al., 2003; Evers et al., 2010; Heatherton & Baumeister, 1991; Heatherton, Striepe, & Wittenberg, 1998; Spoor et al., 2007; Tice, Bratslavsky, & Baumeister, 2001).

Rumination might also negatively impact weight management efforts because it tends to co-occur with cognitive inflexibility and poor problem-solving ability (Davis & Nolen-Hoeksema, 2000; Lyubomirsky et al., 1999; Nolen-Hoeksema, 1991; Whitmer & Banich, 2007). Koster et al. (2011) hypothesized that rumination is characterized by "impaired disengagement" which is evident at a neural level such that heightened limbic and attenuated prefrontal cortex activation contribute to sustained emotion processing. Results from the present analysis are generally consistent with this model: rumination was associated with greater post-meal activation in insula, amygdala, and hippocampal formation. Therefore, this "impaired disengagement" might be especially apparent when an individual with a ruminative response style views food stimuli or consumes food. Greater tendency to engage in rumination is associated with insufficient down-regulation of these emotion processing and food motivation brain regions after eating. Additionally, hypo-activation of a behavioral control network further reflects a pattern of brain activation that interferes with the flexible and creative problem-solving necessary for diet success (Elfhag & Rössner, 2005; Reyes et al., 2012).

Treatment implications. There is some evidence to suggest that eating behavior is driven more by habit than by deliberate emotion regulation goals (Adriaanse, de Ridder, & Evers, 2011). In this particular investigation, emotion amplification and rumination might represent habitual ways of responding to affective stimuli, including food, in the environment. In this sense, eating and overeating might represent an emotion-driven behavior that is at times maladaptive or contrary to higher-level goals (Ellard et al., 2010). Thus, personality and

psychological factors might form the "lens" through which these food stimuli are viewed, augmenting certain properties and automatic associations about the food and potentially diminishing others. One treatment approach might be to remove the "lens" and encourage greater objectivity in the processing of food and food-related stimuli; in doing so, previous emotional, cognitive, and behavioral associations are weakened and new learning begins to take place.

A possible treatment modality is mindfulness and/or acceptance, which have a history of positive effects on psychological and physical health. Among fMRI studies, mindfulness and acceptance of negative affect is associated with less activation in emotion generating and elaborating regions such as amygdala, and ventral and medial PFC. Additionally, mindfulness and acceptance have been shown to mediate changes in weight, food-related cognition, and emotional distress (Dalen et al., 2010; Forman, Butryn, Hoffman, & Herbert, 2009; Kristeller & Hallett, 1999; Lillis, Hayes, Bunting, & Masuda, 2009; Niemeier, Leahey, Reed, Brown, & Wing, 2011). In particular, researchers who conducted a set of studies that tested the effect of mindful attention on appetitive response to food cues found that, after practicing mindful attention, participants displayed reduced approach bias toward appetizing food images, both trained and novel (Papies, Barsalou, & Custers, 2011). The authors suggested that engaging in mindful attention practice created a mindset that made it easier for participants to create separation between the stimulus and their reaction to it. Use of these or similar techniques might be especially beneficial for individuals endorsing high levels of emotion amplification and/or rumination.

Limitations

Several limitations of the present study should be acknowledged. The first is that the

participants of the study were all clinically obese, with no healthy weight comparison group in this analysis. Although data were collected from a sample of healthy weight individuals, comparison was beyond the scope of this paper. However, it will be important to investigate how emotion amplification and rumination are related to brain activation in a group of weight stable healthy weight participants. Weight status may be an important moderator of the relationship between emotion processing characteristics and brain response to food cues. Relatedly, the participants in the study were seeking a diet intervention, and results may not be generalizable to a non-diet seeking population.

A second limitation arises from the nature of TEARS as a measure. Because TEARS were designed to capture the *process* of emotion regulation, irrespective of emotion valence and strategy or strategies employed, assumptions about these factors should be considered with caution. Additionally, questionnaire items were worded in such a way as to be valence neutral; however, participants likely interpreted items to be inquiring about *either* positive *or* negative valence, and this may have differed among participants. Moreover, high amplification may be either a reactive or proactive process, and it may occur with varying amounts of effort on the part of the individual. A closer look at the psychological and behavioral correlates of TEARS would help to clarify additional processes contributing to individual differences in emotion regulation.

Similarly, a third limitation relates to the use of the RSQ as a measure of rumination. There are several versions of the RSQ, including the Ruminative Responses Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003), and factor analyses have parsed rumination into multiple constructs with differential associations with depressive symptomatology. On a related note, although the ruminative response style was originally studied with respect to depression, none of the participants in the present investigation met criteria for clinical depression; in fact,

this was an exclusionary criterion. Thus, the ruminative responses endorsed by these participants may differ qualitatively from those endorsed by an individual experiencing clinical depression. It is unclear the extent to which this "true" depressive rumination might be associated with different patterns of brain activation to food cues.

A fourth limitation relates broadly to data analysis methods and interpretation. It should be emphasized that the correlational analyses help to illustrate relationships among personality/psychological variables, brain response to food cues, and weight loss outcomes. However, it is not possible to disentangle to what extent one factor "causes" or is the "effect of" another. Additionally, while not a limitation, findings must be interpreted in the context of the food motivation paradigm and the circumstances surrounding each scanning timepoint. Likewise, amplification and rumination were investigated as correlates but participants were not instructed to amplify emotions or ruminate during scanning.

Conclusions and Future Directions

Despite the aforementioned limitations, this is the first study addressing the relations among brain function, rumination, emotion amplification, and weight-related outcomes using fMRI and a sample of obese individuals undergoing a behavioral weight loss intervention. Participant differences in self-reported emotion amplification and rumination were correlated with differential change in brain response to food images in limbic, paralimbic, and prefrontal cortex. Notably, greater self-reported tendency to ruminate was correlated with less activation in DLPFC and caudate, and activation in these regions was prospectively correlated with more weight loss. These findings suggest that personality and psychological characteristics influence how food stimuli are perceived and processed, which in turn determines behavioral response and ultimately may facilitate or interfere with goal achievement. To the extent that food cue-elicited

responses are automatic or habit-driven, the use of mindfulness and/or acceptance techniques might be beneficial in changing an individual's relationship with food. A natural direction for future research would be a longitudinal neuroimaging studies investigating both baseline predictors of mindfulness/acceptance treatment success as well as pre-treatment to post-treatment change.

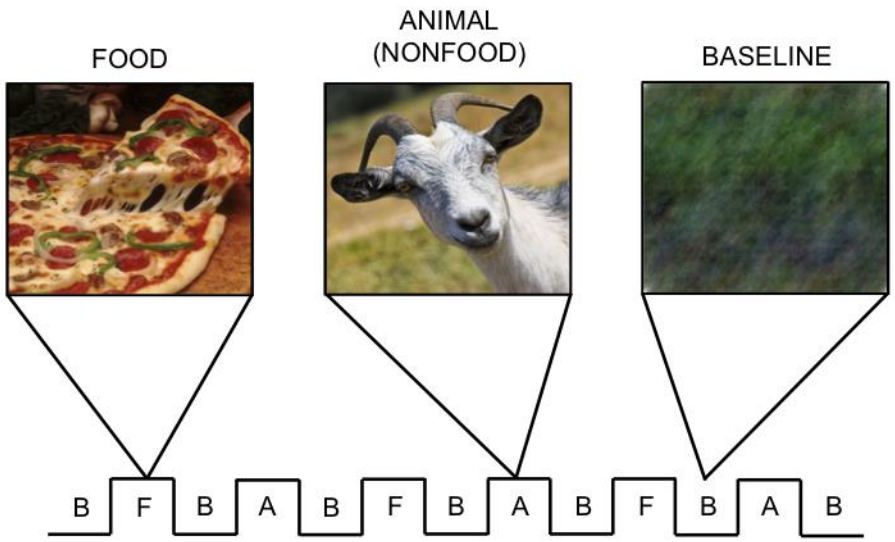

Figure 1. Food motivation paradigm. Participants viewed pictures of Food (F) and animals (A; Nonfood), as well as blurry images (B) created from the food and animal images. This was a block design, with 13 blocks containing 10 images each. Food and animal images alternated with blurry images. Order was 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.

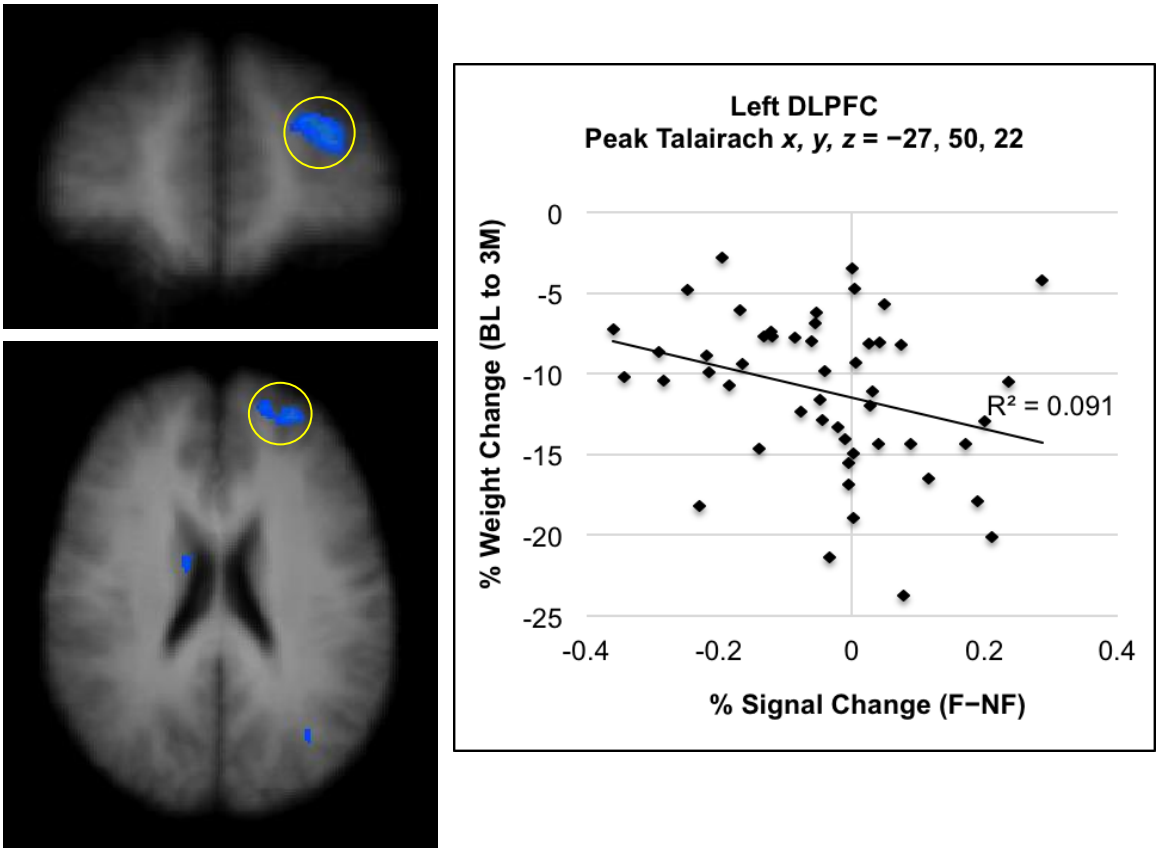

Figure 2. Correlation of brain activation and percent weight change in the left dorsolateral prefrontal cortex (DLPFC). Signal change (F–NF) in the left DLPFC (–27, 50, 22) was inversely correlated with percent weight change from BL to 3M, such that activation F>NF was correlated with more weight loss, whereas activation NF>F was correlated with less weight loss. Region of interest was defined from the baseline post-meal session correlation of beta value (F–NF) and Rumination score. Coronal (top) and axial (bottom) perspectives of the DLPFC cluster are depicted in radiological convention (left = right).

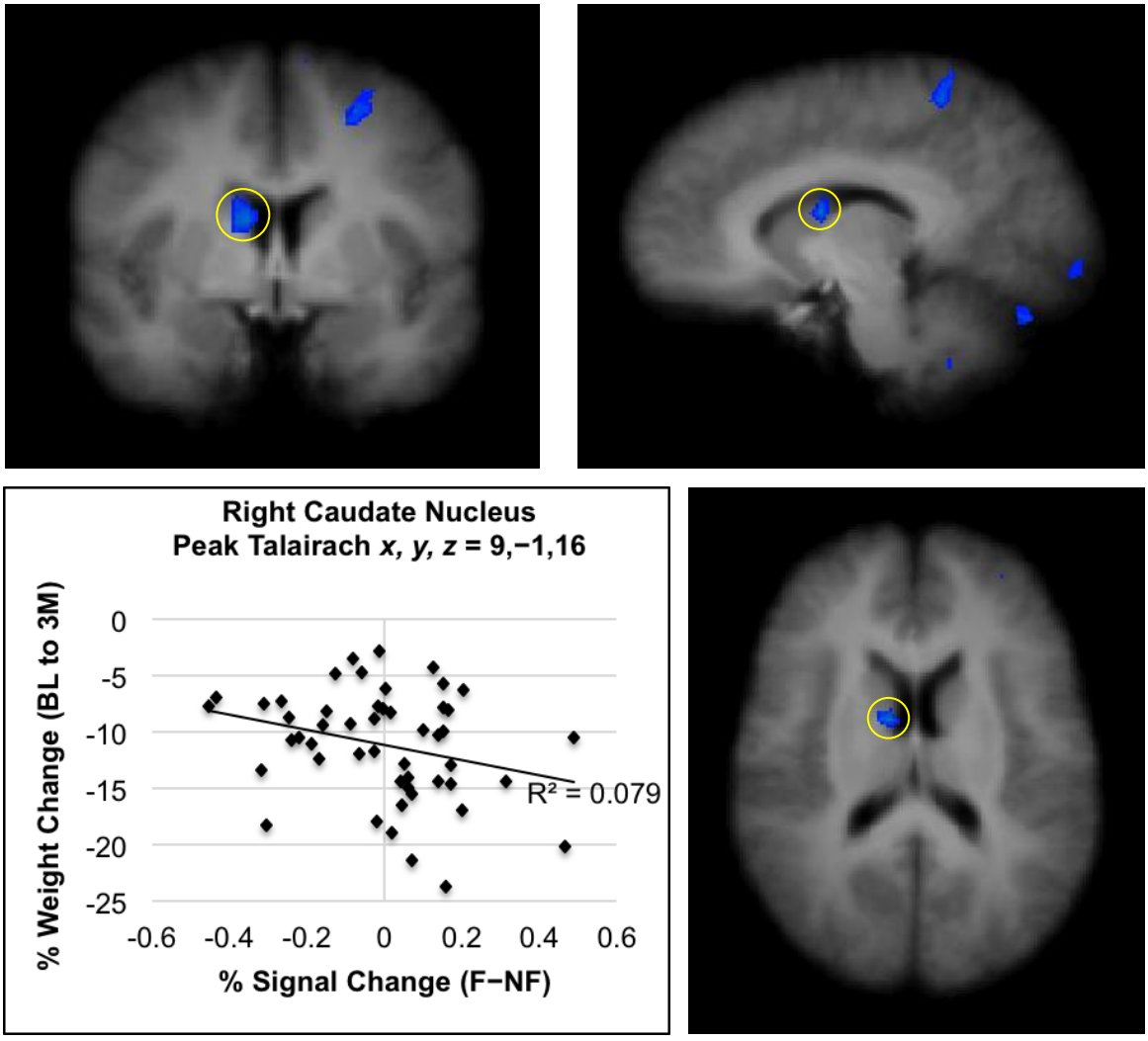

Figure 3. Correlation of brain activation and percent weight change in the right caudate nucleus. Signal change (F–NF) in the right caudate nucleus (9, –1, 16) was inversely correlated with percent weight change from BL to 3M, such that activation F>NF was correlated with more weight loss, whereas activation NF>F was correlated with less weight loss. Region of interest was defined from the baseline post-meal session correlation of beta value (F–NF) and Rumination score. Coronal (top left), sagittal (top right) and axial (bottom) perspectives of the caudate cluster are depicted in radiological convention (left = right).

Table 1
Participant Demographics and Characteristics

Characteristic	*n* = 50
Mean Age at Baseline (SD)	39.02 (7.99)
Mean BMI at BL (kg/m^2) (SD)	35.16 (3.37)
Percent weight change BL to 3M	–11.09 (4.83)
Percent weight change 3M to 9M	1.04 (5.22)
N (%) Female	34 (68)
N (%) Caucasian	38 (76)
N (%) ≥ Some College Education	47 (94)
TEARS Amplification	23.18 (5.11)
Rumination	2.88 (2.72)

Note. M = mean; SD = standard deviation; BMI = Body Mass Index; BL = Baseline; 3M = 3-Month Follow-up; 9M = 9-Month Follow-up; TEARS = The Emotion Amplification and Reduction Scales

Table 2
Correlation Matrix

	BL Age	BL BMI	%WtCh BL-3M	%WtCh 3M-9M	Amplification
BL BMI	.159				
%WtCh BL-3M	–.342*	–.038			
%WtCh 3M-9M	–.170	.079	.422**		
Amplification	–.143	.107	.260	.142	
Rumination	–.005	.029	.270	.208	.122

Note. BL = Baseline timepoint; BMI= Body Mass Index; 3M = 3-month follow-up timepoint; 9M = 9-month follow-up timepoint; %WtCh = percent weight change

*$p < .05$ **$p < .01$

Table 3
Differences in Questionnaire Scores and Weight Change, by Gender

	Mean (SD)			
Variable	Males (*n*=16)	Females (*n*=34)	*t*	*p*
Age	38.63 (8.81)	39.21 (7.71)	–.237	.813
IQ (WASI)	108.94 (8.37)	115.21 (9.93)	–2.184	.034*
BL BMI (kg/m^2)	35.16 (2.88)	35.17 (3.62)	–.008	.993
3M BMI (kg/m^2)	30.07 (2.68)	31.82 (3.60)	–1.728	.090
% Weight Change (BL to 3M)	–14.36 (5.73)	–9.56 (3.47)	–3.094	.006**
% Weight Change (3M to 9M)	.18 (6.57)	1.45 (4.51)	–.803	.426
TEARS Amplification	22.63 (5.12)	23.44 (5.17)	–.523	.604
Rumination	1.38 (1.71)	3.56 (2.83)	–2.882	.006**

Note. BL = Baseline timepoint; BMI= Body Mass Index; 3M = 3-month follow-up timepoint; 9M = 9-month follow-up timepoint;

*$p < .05$ **$p < .01$

Table 4
Regions Reaching Significance During GLM Contrasts of Food>Nonfood, Correlated with TEARS Amplification Score

Region and contrast	BA	Peak Talairach Coordinates			mm^3	*r*	*p*
		x	*y*	*z*			
Pre-Diet (BL)							
Pre-meal							
Angular gyrus	R39	39	–55	34	453	–.479	0.000435
Middle temporal gyrus	L39	–33	–55	28	630	–.542	0.000048
Post-meal							
Superior frontal gyrus	R6	12	14	52	372	–.539	0.000053
Middle frontal gyrus	R6	27	8	58	269	–.480	0.000413
Inferior parietal lobule	R40	54	–37	49	314	–.551	0.000034
Post-Diet (3M)							
Pre-meal							
Inferior occipital gyrus	L18	–27	–91	–38	405	–.484	0.000373
Post-meal							
Inferior occipital gyrus	R18	30	–85	–5	372	–.498	0.000234

Note. L = left; R = right; BA = Brodmann Area

Table 5
Regions Reaching Significance During GLM Contrasts of Food>Nonfood, Correlated with Rumination Score

Region and contrast	BA	Peak Talairach Coordinates			mm^3	*r*	*p*
		x	*y*	*z*			
Pre-Diet (BL)							
Pre-meal							
Middle frontal gyrus	L9	–48	26	31	6870	.567	0.000018
Superior frontal gyrus	R11	18	62	–15	236	.593	0.000006
	R6	21	–4	52	756	.521	0.000105
Precentral gyrus	L44	–51	11	10	333	.505	0.000185
Postcentral gyrus	L2	–55	–25	43	280	.465	0.000673
	R3	27	–37	43	1219	.535	0.000064
	L43	–57	–7	19	308	.472	0.000532
Superior parietal lobule	L7	–12	–61	58	208	.476	0.000476
Inferior parietal lobule	L40	–30	–34	34	193	.574	0.000003
	R40	48	–37	37	261	–.459	0.000798
Fusiform gyrus	R20	51	–22	–20	369	–.492	0.000288
Cuneus	R18	24	–70	13	325	.435	0.000059
Cerebellum	L	–24	–49	–14	281	.488	0.000222
Post-meal							
Inferior frontal gyrus	L47	–39	26	–8	245	–.511	0.000147
	L45	–48	17	4	312	–.491	0.000295
Superior frontal gyrus	L10	–27	50	22	533	–.491	0.000298
	L6	–3	8	61	358	–.478	0.000445

Middle frontal gyrus	L6	–24	–1	49	245	–.466	0.000646
Caudate	R	9	–1	16	239	–.536	0.000060
Precuneus	R7	9	–43	55	356	–.503	0.000200
	R19	24	–67	37	200	–.488	0.000324
Inferior parietal lobule	R40	48	–40	43	457	–.476	0.000472
Middle/Inferior temporal gyrus	R21/20	63	–22	–14	334	–.486	0.000349
Occipital gyrus	L19	–33	–70	28	1783	–.653	0.000000
Cerebellum	R	33	–43	–23	344	–.529	0.000078
		3	–70	–17	207	–.480	0.000414
Pons	R	3	–43	–35	260	–.488	0.000321
Post-Diet (3M)							
Pre-meal							
Middle frontal gyrus	L9	–27	38	28	243	–.474	0.000515
Precuneus	L7	–21	–55	37	252	.448	0.001102
Superior temporal gyrus	L22	–45	–37	–2	356	–.537	0.000058
Middle temporal gyrus	R19	39	–76	22	387	.495	0.000259
Cerebellum	R	36	–37	–26	361	–.516	0.000123
Brainstem	L	–12	–22	–8	554	–.557	0.000027
Post-meal							
Middle frontal gyrus	R10	33	47	19	1997	.568	0.000017
	L10	–36	44	10	281	.500	0.000218
	L9	–33	20	28	199	.578	0.000011
Inferior frontal gyrus	R45	51	23	10	261	.433	0.001678
	R44	42	8	22	382	.535	0.000062
Insula	R	33	14	–5	213	.480	0.000417

	L	–39	17	4	1042	.549	0.000037
	L	–39	–1	10	266	.518	0.000117
Parahippocampal gyrus	R35	24	–25	–17	1348	.538	0.000057
Hippocampus	L	–33	–19	–11	197	.537	0.000057
Superior frontal gyrus	R6	6	14	55	664	.477	0.000457
Precuneus	R7	21	–43	52	339	.528	0.000082
		15	–46	31	299	.449	0.001080
		9	–52	58	202	.477	0.000364
Superior temporal gyrus	L42	–54	–28	16	215	.507	0.000173
	R22	42	–16	–5	571	.519	0.000112
Middle temporal gyrus	R37/39	42	–58	10	673	.480	0.000415
Cerebellum	L	–21	–49	–11	617	.480	0.000422

Note. L = left; R = right; BA = Brodmann Area

Table 6
Regions Reaching Significance During GLM Contrasts of Timepoint (BL, 3M) × Stimulus Type (Food, Nonfood), Correlated with TEARS Amplification Score

Region and contrast	BA	Peak Talairach Coordinates			mm^3	*r*	*p*
		x	*y*	*z*			
Pre-meal							
Superior frontal gyrus	R9	21	53	51	274	.505	0.000186
Post-meal							
Superior frontal gyrus	R6	9	14	52	474	–.493	0.000278
Superior temporal gyrus	R22	42	–37	22	554	–.565	0.000020

Note. L = left; R = right; BA = Brodmann Area

Table 7
Regions Reaching Significance During GLM Contrasts of Timepoint (BL, 3M) × Stimulus Type (Food, Nonfood), Correlated with Rumination Score

Region and contrast	BA	Peak Talairach Coordinates					
		x	*y*	*z*	mm^3	*r*	*p*
Pre-meal							
Middle frontal gyrus	L9	–48	26	34	11293	.670	0.000000
	L6	–30	8	58	641	.557	0.000026
Cingulate gyrus	L24	–3	–10	37	189	.500	0.000219
Precentral gyrus	L6	–51	11	10	396	.507	0.000171
Precuneus	L7	–6	–46	46	251	.532	0.000069
Parietal cortex	L40	–27	–37	49	553	.539	0.000054
Post-meal							
Superior frontal gyrus	L10	–24	50	25	1065	–.521	0.000104
	R9	21	50	28	342	–.493	0.000272
Inferior frontal gyrus	L9	–51	11	28	309	–.481	0.000401
	R45	54	26	4	2896	–.558	0.000025
Anterior cingulate cortex	R32	12	35	10	3960	–.567	0.000018
Parahippocampal gyrus	L36	–24	–31	–17	953	–.567	0.000018
	R36	24	–28	–17	265	–.499	0.000224
Amygdala	L	–21	–7	–8	398	–.489	0.000317
Medial frontal gyrus	L/R6	0	29	34	3520	–.551	0.000034
	L6	–12	8	55	323	–.517	0.000123
Precentral gyrus	L44	–51	17	7	1871	–.544	0.000044
	L6	–42	–1	46	463	–.569	0.000016

Postcentral gyrus	R7	12	–49	67	658	–.518	0.000116
		21	–49	64	290	–.462	0.000734
Posterior cingulate gyrus	L30	–9	–64	10	329	–.429	0.001862
Precuneus	L7	–9	–52	61	380	–.425	0.002077
Superior parietal lobule	L7	–24	–44	55	563	–.511	0.000148
Superior temporal gyrus	R22	51	–10	4	589	–.530	0.000076
	R13	54	–43	22	2896	–.558	0.000025
Middle temporal gyrus	L39	–45	–58	13	401	–.507	0.000173
	L21	–66	–34	–14	232	–.486	0.000342
Middle/Inferior temporal gyrus	L20	–60	–25	–14	481	–.492	0.000288
Lingual gyrus	R19	18	–49	–5	234	–.514	0.000136
Cerebellum	R/L	0	–43	–35	262	–.443	0.001268

Note. L = left; R = right; BA = Brodmann Area

References

Adolfsson, B., Carlson, A., Undén, A.-L., & Rössner, S. (2002). Treating obesity: A qualitative evaluation of a lifestyle intervention for weight reduction. *Health Education Journal, 61*(3), 244-258. doi: 10.1177/001789690206100306

Adriaanse, M. A., de Ridder, D. T. D., & Evers, C. (2011). Emotional eating: Eating when emotional or emotional about eating? *Psychology and Health, 26*, 23-39. doi: 10.1080/08870440903207627

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy, 48*(10), 974-983. doi: 10.1016/j.brat.2010.06.002

Antin, T. M. J., & Hunt, G. (2012). Food choice as a multidimensional experience. A qualitative study with young African American women. *Appetite, 58*(3), 856-863. doi: 10.1016/j.appet.2012.01.021

Bachman, J. L., Phelan, S., Wing, R. R., & Raynor, H. A. (2011). Eating frequency is higher in weight loss maintainers and normal-weight individuals than in overweight individuals. *Journal of the American Dietetic Association, 111*(11), 1730-1734. doi: 10.1016/j.jada.2011.08.006

Bagby, R. M., Rector, N. A., Bacchiochi, J. R., & McBride, C. (2004). The stability of the Response Styles Questionnaire rumination scale in a sample of patients with major depression. *Cognitive Therapy and Research, 28*, 527-538. doi: 10.1023/B:COTR.0000045562.17228.29

Baumeister, R. F., & Heatherton, T. F. (1996). Self-regulation failure: An overview. *Psychological Inquiry, 7*(1), 1-15. doi: 10.1207/s15327965pli0701_1

Beaver, J. D., Lawrence, A. D., van Ditzhuijzen, J., Davis, M. H., Woods, A., & Calder, A. J. (2006). Individual differences in reward drive predict neural responses to images of food. *The Journal of Neuroscience, 26(19),* 5160-5166. doi: 10.1523/JNEUROSCI.0350-06.2006

Befort, C. A., Stewart, E. E., Smith, B. K., Gibson, C. A., Sullivan, D. K., & Donnelly, J. E. (2007). Weight maintenance, behaviors and barriers among previous participants of a university-based weight control program. *International Journal of Obesity, 32*(3), 519-526. doi: 10.1038/sj.ijo.0803769

Binder, J. R., Desai, R. H., Graves, W. W., & Conant, L. L. (2009). Where is the semantic system? A critical review and meta-analysis of 120 functional neuroimaging studies. *Cerebral Cortex, 19,* 2767-2796. doi: 10.1093/cercor/bhp055

Bongers, P., Jansen, A., Havermans, R., Roefs, A., & Nederkoorn, C. (2013). Happy eating: The underestimated role of overeating in a positive mood. *Appetite, 67,* 74–80. doi: 10.1016/j.appet.2013.03.017

Bongers, P., Jansen, A., Houben, K., & Roefs, A. (2013). Happy eating: The Single Target Implicit Association Test predicts overeating after positive emotions. *Eating Behaviors, 14,* 348–355. doi: 10.1016/j.eatbeh.2013.06.007

Brownell, K. D. & Wadden, T. A. (1992). Etiology and treatment of obesity: Understanding a serious, prevalent, and refractory disorder. *Journal of Consulting and Clinical Psychology, 60(4),* 505-517. doi: 10.1037/0022-006X.60.4.505

Bohon, C., Stice, E., & Spoor, S. (2009). Female emotional eaters show abnormalities in consummatory and anticipatory food reward: A functional magnetic resonance imaging study. *International Journal of Eating Disorders, 42(3),* 210–221. doi: 10.1002/eat.20615

Brooks, S. J., Cedernaes, J., & Schiöth, H. B. (2013). Increased prefrontal and parahippocampal activation with reduced dorsolateral prefrontal and insular cortex activation to food images in obesity: A meta-analysis of fMRI studies. *PLoS ONE, 8(4),* e60393. doi: 10.1371/journal.pone.0060393

Bruce, A. S., Holsen, L. M., Chambers, R. J., Martin, L. E., Brooks, W. M., Zarcone, J. R., . . . Savage, C. R. (2010). Obese children show hyperactivation to food pictures in brain networks linked to motivation, reward and cognitive control. *International Journal of Obesity, 34*(10), 1494-1500. doi: 10.1038/ijo.2010.84

Bruce, J. M., Hancock, L., Bruce, A., Lepping, R. J., Martin, L., Lundgren, J. D., . . . Savage, C. R. (2011). Changes in brain activation to food pictures after adjustable gastric banding. *Surgery for Obesity and Related Diseases*. doi: 10.1016/j.soard.2011.07.006

Burger, K. S. & Stice, E. (2011). Relation of dietary restraint scores to activation of reward-related brain regions in response to food intake, anticipated intake, and food pictures. *NeuroImage, 55,* 233–239. doi: 10.1016/j.neuroimage.2010.12.009

Burgess, P. W., Scott, S. K., & Frith, C. D. (2003). The role of the rostral frontal cortex (area 10) in prospective memory: a lateral versus medial dissociation. *Neuropsychologia, 41(8),* 906-918. doi: 10.1016/S0028-3932(02)00327-5

Burke, L. E., Swigart, V., Turk, M. W., Derro, N., & Ewing, L. J. (2009). Experiences of self-monitoring: Successes and struggles during treatment for weight loss. *Qualitative Health Research, 19*(6), 815-828. doi: 10.1177/1049732309335395

Bush, G. Vogt, B. A., Holmes, J., Dale, A. M., Greve, D., Jenike, M. A., & Rosen, B. R. (2002). Dorsal anterior cingulate cortex: A role in reward-based decision making. *Proceedings of the National Academy of Sciences, 99,* 523–528. doi: 10.1073/pnas.012470999

Butryn, M. L., Phelan, S., Hill, J. O., & Wing, R. R. (2007). Consistent self-monitoring of weight: A key component of successful weight loss maintenance. *Obesity, 15,* 3091–3096. doi: 10.1038/oby.2007.368

Byrne, S., Cooper, Z., & Fairburn, C. (2003). Weight maintenance and relapse in obesity: A qualitative study. *International Journal of Obesity, 27*, 955–962. doi: 10.1038/sj.ijo.802305

Carnell, S., Gibson, C., Benson, L., Ochner, C. N., & Geliebter, A. (2012). Neuroimaging and obesity: current knowledge and future directions. *Obesity Reviews, 13*(1), 43-56. doi: 10.1111/j.1467-789X.2011.00927.x

Cauda, F., Costa, T., Torta, D. M., Sacco, K., D'Agata, F., Duca, S., ... & Vercelli, A. (2012). Meta-analytic clustering of the insular cortex Characterizing the meta-analytic connectivity of the insula when involved in active tasks. *NeuroImage, 61(1)*, 343-355. doi: 10.1016/j.neuroimage.2012.04.012

Cawley, J. & Meyerhoefer, C. (2011). The medical care costs of obesity: An instrumental variables approach. *Journal of Health Economics, 31*, 219–230. doi: 10.1016/j.jhealeco.2011.10.003

Champagne, C. M., Broyles, S. T., Moran, L. D., Cash, K. C., Levy, E. J., Lin, P.-H., . . . Myers, V. H. (2011). Dietary intakes associated with successful weight loss and maintenance during the weight loss maintenance trial. *Journal of the American Dietetic Association, 111*(12), 1826-1835. doi: 10.1016/j.jada.2011.09.014

Cooney, R. E., Joormann, J., Eugène, F., Dennis, E. L., & Gotlib, I. H. (2010). Neural correlates of rumination in depression. *Cognitive, Affective, & Behavioral Neuroscience, 10(4),* 470-478. doi: 10.3758/CABN.10.4.470

Cornier, M. A., Von Kaenel, S. S., Bessesen, D. H., & Tregellas, J. R. (2007). Effects of overfeeding on the neuronal response to visual food cues. *American Journal of Clinical Nutrition, 86(4),* 965-971.

Cropley, M., Michalianou, G., Pravettoni, G., & Millward, L. J. (2012). The relation of post-work ruminative thinking with eating behaviour. *Stress and Health*, *28*(1), 23-30. doi: 10.1002/smi.1397

Dalen, J., Smith, B. W., Shelley, B. M., Sloan, A. L., Leahigh, L., & Begay, D. (2010). Pilot study: Mindful Eating and Living (MEAL): Weight, eating behavior, and psychological outcomes associated with a mindfulness-based intervention for people with obesity. *Complementary Therapies in Medicine, 18(6),* 260–264. doi:10.1016/j.ctim.2010.09.008

Davis, R. N. & Nolen-Hoeksema, S. (2000). Cognitive inflexibility among ruminators and nonruminators. *Cognitive Therapy and Research, 24*(6), 699-711. doi: 10.1023/a:1005591412406

de Araujo, I. E. & Rolls, E. T. (2004). Representation in the human brain of food texture and oral fat. *The Journal of Neuroscience, 24*(12), 3086-3093. doi: 10.1523/jneurosci.0130-04.2004

DelParigi, A., Chen, K., Salbe, A. D., Hill, J. O., Wing, R. R., Reiman, E. M., & Tataranni, P. A. (2006). Successful dieters have increased neural activity in cortical areas involved in the control of behavior. *International Journal of Obesity*, *31*(3), 440-448. doi: 10.1038/sj.ijo.0803431

Desmet, P. M. A. & Schifferstein, H. N. J. (2008). Sources of positive and negative emotions in food experience. *Appetite, 50,* 290–301. doi:10.1016/j.appet.2007.08.003

Dimitropoulos, A., Tkach, J., Ho, A., & Kennedy, J. (2012). Greater corticolimbic activation to

high-calorie food cues after eating in obese vs. normal-weight adults. *Appetite, 58*(1), 303-312. doi: 10.1016/j.appet.2011.10.014

Drapkin, R. G., Wing, R. R., & Shiffman, S. (1995). Responses to hypothetical high risk situations: Do they predict weight loss in a behavioral treatment program or the context of dietary lapses? *Health Psychology, 14*(5), 427-434. doi: 10.1037/0278-6133.14.5.427

Eichenbaum, H. (2004). Hippocampus: cognitive processes and neural representations that underlie declarative memory. *Neuron, 44(1),* 109-120. doi: 10.1016/j.neuron.2004.08.028

Elfhag, K. & Rössner, S. (2005). Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obesity Reviews, 6,* 67–85. doi: 10.1111/j.1467-789X.2005.00170.x

Ellard, K. K., Fairholme, C. P., Boisseau, C. L., Farchione, T. J., & Barlow, D. H. (2010). Unified protocol for the transdiagnostic treatment of emotional disorders: Protocol development and initial outcome data. *Cognitive and Behavioral Practice, 17(1),* 88-101. doi: 10.1016/j.cbpra.2009.06.002

Evers, C., Adriaanse, M., de Ridder, D. T. D., de Witt Huberts, J. C. (2013). Good mood food: Positive emotion as a neglected trigger for food intake. *Appetite, 68,* 1–7. 10.1016/j.appet.2013.04.007

Evers, C., Marijn Stok, F., & de Ridder, D. T. D. (2010). Feeding your feelings: Emotion regulation strategies and emotional eating. *Personality and Social Psychology Bulletin, 36*(6), 792-804. doi: 10.1177/0146167210371383

Farrer, C., Frey, S. H., Van Horn, J. D., Tunik, E., Turk, D., Inati, S., & Grafton, S. T. (2008). The angular gyrus computes action awareness representations. *Cerebral Cortex, 18,* 254-261. doi: 10.1093/cercor/bhm050

Featherstone, R. E. & McDonald, R. J. (2004). Dorsal striatum and stimulus-response learning: lesions of the dorsolateral, but not dorsomedial, striatum impair acquisition of a stimulus-response-based instrumental discrimination task, while sparing conditioned place preference learning. *Neuroscience, 124*(1), 23-31. doi: 10.1016/j.neuroscience.2003.10.038

Fink, G. R., Markowitsch, H. J., Reinkemeier, M., Bruckbauer, T., Kessler, J., & Heiss, W. D. (1996). Cerebral representation of one's own past: Neural networks involved in autobiographical memory. *The Journal of Neuroscience, 16(13)*, 4275-4282.

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

Forman, E. M., Butryn, M. L., Hoffman, K. L., & Herbert, J. D. (2009). An open trial of an acceptance-based behavioral intervention for weight loss. *Cognitive and Behavioral Practice, 16*, 223–235. doi: 10.1016/j.cbpra.2008.09.005

Forman, S. D., Cohen, J. D., Fitzgerald, M., Eddy, W. F., Mintun, M. A., & Noll, D. C. (1995). Improved assessment of significant activation in functional magnetic resonalnce imaging (fMRI): Use of a cluster-size threshold. *Magnetic Resonance in Medicine, 33*(5), 636-647. doi: 10.1002/mrm.1910330508

Friston, K. J., Holmes, A. P., Poline, J. B., Grasby, P. J., Williams, S. C. R., Frackowiak, R. S. J., & Turner, R. (1995). Analysis of fMRI time-series revisited. *NeuroImage, 2*(1), 45-53. doi: 10.1006/nimg.1995.1007

Gautier, J.-F., Del Parigi, A., Chen, K., Salbe, A. D., Bandy, D., Pratley, R. E., . . . Tataranni, P. A. (2001). Effect of satiation on brain activity in obese and lean women. *Obesity, 9*(11),

676-684. doi: 10.1038/oby.2001.92

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*, 577–586. doi: 10.1016/j.biopsych.2007.05.031

Goldstone, A. P., Prechtl de Hernandez, C. G., Beaver, J. D., Muhammed, K., Croese, C., Bell, G., ... & Bell, J. D. (2009). Fasting biases brain reward systems towards high- calorie foods. *European Journal of Neuroscience, 30(8),* 1625-1635. doi: 10.1111/j.1460-9568.2009.06949.x

Grilo, C. M., Shiffman, S., & Wing, R. R. (1989). Relapse crises and coping among dieters. *Journal of Consulting and Clinical Psychology, 57*(4), 488-495. doi: 10.1037/0022-006x.57.4.488

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237. doi: 10.1037/0022-3514.74.1.224

Hamann, S. B., Ely, T. D., Grafton, S. T., & Kilts, C. D. (1999). Amygdala activity related to enhanced memory for pleasant and aversive stimuli. *Nature Neuroscience, 2(3),* 289-293. doi: 10.1038/6404

Hamann, S. B., Ely, T. D., Hoffman, J. M., & Kilts, C. D. (2002). Ecstasy and agony: activation of the human amygdala in positive and negative emotion. *Psychological Science, 13(2),* 135-141. doi: 10.1111/1467-9280.00425

Hamilton, N. A., Karoly, P., Gallagher, M., McCurdy, D., & Karlson, C. (2009). The assessment of emotion regulation in cognitive context: The emotion amplification and reduction scales. *Cognitive Therapy and Research, 33*, 255–263. doi: 10.1007/s10608-007-9163-9

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*(1), 86-108. doi: 10.1037/0033-2909.110.1.86

Heatherton, T. F., Striepe, M., & Wittenberg, L. (1998). Emotional distress and disinhibited eating: The role of self. *Personality and Social Psychology Bulletin, 24*(3), 301-313. doi: 10.1177/0146167298243007

Henke, K., Weber, B., Kneifel, S., Wieser, H. G., & Buck, A. (1999). Human hippocampus associates information in memory. *Proceedings of the National Academy of Sciences, 96(10)*, 5884-5889. doi: 10.1073/pnas.96.10.5884

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43*(4), 647-660. doi: 10.1111/j.1467-6494.1975.tb00727.x

Hoffstaedter, F., Grefkes, C., Zilles, K., & Eickoff, S. B. (2013). The "what" and "when" of self-initiated movements. *Cerebral Cortex, 23*, 520-530. doi: 10.1093/cercor/bhr391

Hollmann, M., Hellrung, L., Pleger, B., Schlogl, H., Kabisch, S., Stumvoll, M., . . . Horstmann, A. (2011). Neural correlates of the volitional regulation of the desire for food. *International Journal of Obesity, 36*(5), 648-655. doi: 10.1038/ijo.2011.125

Holsen, L. M., Zarcone, J. R., Thompson, T. I., Brooks, W. M., Anderson, M. F., Ahluwalia, J. S., . . . Savage, C. R. (2005). Neural mechanisms underlying food motivation in children and adolescents. *NeuroImage, 27*(3), 669-676. doi: 10.1016/j.neuroimage.2005.04.043

Hopfinger, J. B., Buonocore, M. H., & Mangun, G. R. (2000). The neural mechanisms of top-down attentional control. *Nature Neuroscience, 3(3)*, 284-291. doi: 10.1038/72999

Jansen, A., Vanreyton, A., van Balveren, T., Roefs, A., Nederkoorn, C., & Havermans, R. (2008). Negative affect and cue-induced overeating in non-eating disordered obesity. *Appetite, 51*, 556–562. doi: 10.1016/j.appet.2008.04.009

Karoly, P. (1993). Mechanisms of self-regulation: A systems view. *Annual Review of Psychology, 44*, 23–52. doi: 10.1146/annurev.ps.44.020193.000323

Karoly, P. (1999). A goal-systems-self-regulatory perspective on personality, psychopathology, and change. *Review of General Psychology, 3*, 264-291.

Kasch, K. L., Klein, D. N., & Lara, M. E. (2001). A construct validation study of the Response Styles Questionnaire Rumination scale in participants with a recent-onset major depressive episode. *Psychological Assessment, 13*, 375-383. doi: 10.1037/1040-3590.13.3.375

Kayman, S., Bruvold, W., & Stern, J. S. (1990). Maintenance and relapse after weight loss in women: Behavioral aspects. *The American Journal of Clinical Nutrition, 52*(5), 800-807.

Kemp, E., Bui, M., & Grier, S. (2011). Eating their feelings: Examining emotional eating in at-risk groups in the United States. *Journal of Consumer Policy, 34*(2), 211-229. doi: 10.1007/s10603-010-9149-y

Klem, M. L., Wing, R. R., McGuire, M. T., Seagle, H. M., & Hill, J. O. (1997). A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *The American Journal of Clinical Nutrition, 66*(2), 239-246.

Kopelman, P. (2007). Health risks associated with overweight and obesity. *Obesity Reviews, 8*, 13-17. doi: 10.1111/j.1467-789X.2007.00311.x

Kopelman, P. G. (2000). Obesity as a medical problem. *Nature, 404*(6778), 635-643. doi: 10.1038/35007508

Koster, E. H. W., De Lissnyder, E., Derakshan, N., & De Raedt, R. (2011). Understanding depressive rumination from a cognitive science perspective: The impaired disengagement hypothesis. *Clinical Psychology Review, 31*, 138–145. doi: 10.1016/j.cpr.2010.08.005

Kringelbach, M. L., de Araujo, I. E. T., & Rolls, E. T. (2004). Taste-related activity in the human dorsolateral prefrontal cortex. *NeuroImage, 21,* 781–788. doi: 10.1016/j.neuroimage.2003.09.063

Kristeller, J. L. & Hallett, C. B. (1999). An exploratory study of a meditation-based intervention for binge eating disorder. *Journal of Health Psychology, 4*, 357–363. doi: 10.1177/135910539900400305

Kruger, J., Blanck, H., & Gillespie, C. (2006). Dietary and physical activity behaviors among adults successful at weight loss maintenance. *International Journal of Behavioral Nutrition and Physical Activity, 3*(1), 1-10. doi: 10.1186/1479-5868-3-17

Kubiak, T., Vögele, C., Siering, M., Schiel, R., & Weber, H. (2008). Daily hassles and emotional eating in obese adolescents under restricted dietary conditions—The role of ruminative thinking. *Appetite, 51*(1), 206-209. doi: 10.1016/j.appet.2008.01.008

Kurth, F., Zilles, K., Fox, P. T., Laird, A. R., & Eickhoff, S. B. (2010). A link between the systems: functional differentiation and integration within the human insula revealed by meta-analysis. *Brain Structure and Function, 214(5-6),* 519-534. doi: 10.1007/s00429-010-0255-z

LaBar, K. S., Gitelman, D. R., Parrish, T. B., Kim, Y.-H., Nobre, A. C., & Mesulam, M. M. (2001). Hunger selectively modulates corticolimbic activation to food stimuli in humans. *Behavioral Neuroscience, 115*(2), 493-500. doi: 10.1037/0735-7044.115.2.493

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). *International Affective Picture System (IAPS): Technical Manual and Affective Ratings*. Gainesville, FL: National Institute of Mental Health Center for the Study of Emotion and Attention.

Lau, H. C., Rogers, R. D., Haggard, P., & Passingham, R. E. (2004). Attention to intention.

Science, 303, 1208-1210. doi: 10.1126/science.1090973

Le, D. S. N. T., Pannacciulli, N., Chen, K., Del Parigi, A., Salbe, A. D., Reiman, E. M., & Krakoff, J. (2006). Less activation of the left dorsolateral prefrontal cortex in response to a meal: a feature of obesity. *The American Journal of Clinical Nutrition, 84*(4), 725-731.

Le, D. S. N. T., Pannacciulli, N., Chen, K., Salbe, A. D., Hill, J. O., Wing, R. R., . . . Krakoff, J. (2007). Less activation in the left dorsolateral prefrontal cortex in the reanalysis of the response to a meal in obese than in lean women and its association with successful weight loss. *The American Journal of Clinical Nutrition, 86*(3), 573-579.

Lillis, J., Hayes, S. C., Bunting, K., & Masuda, A. (2009). Teaching acceptance and mindfulness to improve the lives of the obese: A preliminary test of a theoretical model. *Annals of Behavioral Medicine, 37*, 58–69. doi:10.1007/s12160-009-9083-x

Lowe, M. R., & Fisher, E. B. (1983). Emotional reactivity, emotional eating, and obesity: A naturalistic study. *Journal of Behavioral Medicine, 6*, 135–149. doi: 10.1007/BF00845377

Lowe, M. R., & Levine, A. S. (2005). Eating motives and the controversy over dieting: Eating less than needed versus less than wanted. *Obesity, 13*(5), 797-806. doi: 10.1038/oby.2005.90

Loxton, N. J., Dawe, S., & Cahill, A. (2011). Does negative mood drive the urge to eat? The contribution of negative mood, exposure to food cues and eating style. *Appetite, 56*, 368–374. doi: 10.1016/j.appet.2011.01.011

Lyubomirsky, S., Tucker, K. L., Caldwell, N. D., & Berg, K. (1999). Why ruminators are poor problem solvers: Clues from the phenomenology of dysphoric rumination. *Journal of Personality and Social Psychology, 77*(5), 1041-1060. doi: 10.1037/0022-3514.77.5.1041

Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite, 50*, 1–11.

Macht, M., Haupt, C., & Ellgring, H. (2005). The perceived function of eating is changed during examination stress: a field study. *Eating Behaviors, 6*(2), 109-112. doi: 10.1016/j.eatbeh.2004.09.001

Macht, M., Roth, S., & Ellgring, H. (2002). Chocolate eating in healthy men during experimentally induced sadness and joy. *Appetite, 39*(2), 147-158. doi: 10.1006/appe.2002.0499

Macht, M., & Simons, G. (2011). Emotional eating. In I. Nyklíček, A. Vingerhoets & M. Zeelenberg (Eds.), *Emotion Regulation and Well-Being* (pp. 281–295). NY: Springer. doi: 10.1007/978-1-4419-6953-8_17

Mai, J. K., Paxinos, G., & Voss, T. (2008). *Atlas of the Human Brain* (3rd edition). San Diego: Academic Press.

Marsh, R., Steinglass, J. E., Gerber, A. J., O'Leary, K. G., Wang, Z., Murphy, D., . . . Peterson, B. S. (2009). Deficient activity in the neural systems that mediate self-regulatory control in bulimia nervosa. *Archives of General Psychiatry, 66*(1), 51-63. doi: 10.1001/archgenpsychiatry.2008.504

Martin, L. E., Holsen, L. M., Chambers, R. J., Bruce, A. S., Brooks, W. M., Zarcone, J. R., . . . Savage, C. R. (2009). Neural mechanisms associated with food motivation in obese and healthy weight adults. *Obesity, 18*(2), 254-260. doi: 10.1038/oby.2009.220

Martin, L. L., Shrira, I., & Startup, H. M. (2004). Rumination as a function of goal progress, stop rules, and cerebral lateralization. In C. Papageorgiou, & A. Wells (Eds.), *Depressive Rumination: Nature, Theory, and Treatment* (pp. 153-176). Chichester: Wiley. doi: 10.1002/9780470713853.fmatter

McCaffery, J. M., Haley, A. P., Sweet, L. H., Phelan, S., Raynor, H. A., Del Parigi, A., . . . Wing, R. R. (2009). Differential functional magnetic resonance imaging response to food pictures in successful weight-loss maintainers relative to normal-weight and obese controls. *The American Journal of Clinical Nutrition, 90*(4), 928-934. doi: 10.3945/ajcn.2009.27924

McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience, 27*, 1-28. doi: 10.1146/annurev.neuro.27.070203.144157

McGuire, M. T., Wing, R. R., Klem, M. L., Lang, W., & Hill, J. O. (1999). What predicts weight regain in a group of successful weight losers? *Journal of Consulting and Clinical Psychology, 67*(2), 177-185. doi: 10.1037/0022-006x.67.2.177

McRae, K., Ciesielski, B., & Gross, J. J. (2012). Unpacking cognitive reappraisal: Goals, tactics, and outcomes. *Emotion, 12*(2), 250-255. doi: 10.1037/a0026351

McRae, K., Hughes, B., Chopra, S., Gabrieli, J. D. E., Gross, J. J., & Ochsner, K. N. (2010). The neural bases of distraction and reappraisal. *Journal of Cognitive Neuroscience, 22*, 248–262. doi:10.1162/jocn.2009.21243

Mostofsky, S. H. & Simmonds, D. J. (2008). Response inhibition and response selection: Two sides of the same coin. *Journal of Cognitive Neuroscience, 20(5),* 751-761. doi: 10.1162/jocn.2008.20500

Muraven, M. & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*, 247-259. doi: 10.1037/0033-2909.126.2.247

Muraven, M., Tice, D. M., & Baumeister, R. F. (1998). Self-control as a limited resource:

Regulatory depletion patterns. *Journal of Personality and Social Psychology, 74*(3), 774-789. doi: 10.1037/0022-3514.74.3.774

Murdaugh, D. L., Cox, J. E., Cook III, E. W., & Weller, R. E. (2012). fMRI reactivity to high-calorie food pictures predicts short- and long-term outcome in a weight-loss program. *NeuroImage, 59*(3), 2709-2721. doi: 10.1016/j.neuroimage.2011.10.071

Murphy, J. M., Horton, N. J., Burke, J. D., Monson, R. R., Laird, N. M., Lesage, A., & Sobol, A. M. (2009). Obesity and weight gain in relation to depression: Findings from the Stirling County Study. *International Journal of Obesity, 33(3),* 335-341. doi: 10.1038/ijo.2008.273

Niemeier, H. M., Leahey, T., Reed, K. P., Brown, R. A., & Wing, R. R. (2011). An acceptance-based behavioral intervention for weight loss: A pilot study. *Behavior Therapy.* doi: 10.1016/j.beth.2011.10.005

Niemeier, H. M., Phelan, S., Fava, J. L., & Wing, R. R. (2007). Internal disinhibition predicts weight regain following weight loss and weight loss maintenance. *Obesity, 15*(10), 2485-2494. doi: 10.1038/oby.2007.295

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *The Journal of Abnormal Psychology, 100*(4), 569-582. doi: 10.1037/0021-843X.100.4.569

Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology, 102*(1), 20-28. doi: 10.1037/0021-843x.102.1.20

Nolen-Hoeksema, S., Stice, E., Wade, E., & Bohon, C. (2007). Reciprocal relations between rumination and bulimic, substance abuse, and depressive symptoms in female

adolescents. *Journal of Abnormal Psychology, 116*(1), 198-207. doi: 10.1037/0021-843x.116.1.198

Nummenmaa, L., Hirvonen, J., Hannukainen, J. C., Immonen, H., Lindroos, M. M., Salminen, P., & Nuutila, P. (2012). Dorsal striatum and its limbic connectivity mediate abnormal anticipatory reward processing in obesity. *PLoS ONE, 7(2)*, e31089. doi: 10.1371/journal.pone.0031089

O'Connor, D. B., Jones, F., Conner, M., McMillan, B., & Ferguson, E. (2008). Effects of daily hassles and eating style on eating behavior. *Health Psychology, 27(1, Suppl)*, S20-S31. doi: 10.1037/0278-6133.27.1.S20

O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science, 304(5669)*, 452–454. doi: 10.1126/science.1094285

O'Doherty, J. P., Deichmann, R., Critchley, H. D., & Dolan, R. J. (2002). Neural responses during anticipation of a primary taste reward. *Neuron, 33(5)*, 815-826. doi: 10.1016/S0896-6273(02)00603-7

Ochner, C. N., Kwok, Y., Conceição, E., Pantazatos, S. P., Puma, L. M., Carnell, S., . . . Geliebter, A. (2011). Selective reduction in neural responses to high calorie foods following gastric bypass surgery. *Annals of Surgery, 253*(3), 502-507. doi: 10.1097/SLA.0b013e318203a289

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*, 1215–1229. doi: 10.1162/089892902760807212

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D. E., &

Gross, J. J. (2004). For better or worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23,* 483–499. doi: 10.1016/j.neuroimage.2004.06.030

Ouwens, M. A., van Strien, T., van Leeuwe, J. F. J. (2009). Possible pathways between depression, emotional and external eating. A structural equation model. *Appetite, 53,* 245–248. doi: 10.1016/j.appet.2009.06.001

Ozier, A. D., Kendrick, O. W., Leeper, J. D., Knol, L. L., Perko, M., & Burnham, J. (2008). Overweight and obesity are associated with emotion- and stress-related eating as measured by the Eating and Appraisal Due to Emotions and Stress Questionnaire. *Journal of the American Dietetic Association, 108,* 49–56. doi: 10.1016/j.jada.2007.10.011

Pannacciulli, N., Del Parigi, A., Chen, K., Le, D. S. N. T., Reiman, E. M., & Tataranni, P. A. (2006). Brain abnormalities in human obesity: A voxel-based morphometric study. *NeuroImage, 31*(4), 1419-1425. doi: 10.1016/j.neuroimage.2006.01.047

Papies, E. K., Barsalou, L. W., & Custers, R. (2011). Mindful attention prevents mindless impulses. *Social Psychology and Personality Science*. doi: 10.1177/1948550611419031

Passamonti, L., Rowe, J. B., Schwarzbauer, C., Ewbank, M. P., von dem Hagen, E., & Calder, A. J. (2009). Personality predicts the brain's response to viewing appetizing foods: The neural basis of a risk factor for overeating. *The Journal of Neuroscience, 29(1),* 43-51. doi: 10.1523/JNEUROSCI.4966-08.2009

Pelchat, M. L., Johnson, A., Chan, R., Valdez, J., & Ragland, J. D. (2004). Images of desire: food-craving activation during fMRI. *NeuroImage, 23*(4), 1486-1493. doi: 10.1016/j.neuroimage.2004.08.023

Phan, K. L., Fitzgerald, D. A., Nathan, P. J., Moore, G. J., Uhde, T. W. & Tancer, M. E. (2005). Neural substrates for voluntary suppression of negative affect: A functional magnetic resonance imaging study. *Biological Psychiatry, 57*, 210–219. doi: 10.1016/j.biopsych.2004.10.030

Phelan, S., Hill, J. O., Lang, W., Dibello, J. R., & Wing, R. R. (2003). Recovery from relapse among successful weight maintainers. *The American Journal of Clinical Nutrition, 78*(6), 1079-1084.

Phelan, S., Roberts, M., Lang, W., & Wing, R. R. (2007). Empirical evaluation of physical activity recommendations for weight control in women. *Medicine and Science in Sports and Exercise, 39*(10), 1832–1836. doi: 10.1249/mss.ob013e31812383c3.

Polivy, J., Heatherton, T. F., & Herman, C. P. (1988). Self-esteem, restraint, and eating behavior. *Journal of Abnormal Psychology*, *97*, 354–356. doi: 10.1037/0021-843X.97.3.354

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40*(2), 193-201. doi: 10.1037/0003-066x.40.2.193

Polivy, J., & Herman, C. P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders, 26*, 153–164. doi: 10.1002/(SICI)1098-108X(199909)26:2<153::AID-EAT4>3.0.CO;2-R

Porubská, K., Veit, R., Preissl, H., Fritsche, A., & Birbaumer, N. (2006). Subjective feeling of appetite modulates brain activity: An fMRI study. *NeuroImage, 32*, 1273–1280. doi: 10.1016/j.neuroimage.2006.04.216

Radloff, L. S. (1977). The CES-D Scale. *Applied Psychological Measurement, 1*(3), 385-401. doi: 10.1177/014662167700100306

Ramnani, N. & Owen, A. M. (2004). Anterior prefrontal cortex: Insights into function from

anatomy and neuroimaging. *Nature Reviews Neuroscience, 5*, 184-194. doi: 10.1038/nrn1343

Ray, R., Ochsner, K., Cooper, J., Robertson, E., Gabrieli, J., & Gross, J. (2005). Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cognitive, Affective, & Behavioral Neuroscience, 5*(2), 156-168. doi: 10.3758/cabn.5.2.156

Reyes, N. R., Oliver, T. L., Klotz, A. A., LaGrotte, C. A., Vander Veur, S. S., Virus, A., . . . Foster, G. D. (2012). Similarities and differences between weight loss maintainers and regainers: A qualitative analysis. *Journal of the Academy of Nutrition and Dietetics, 112*(4), 499-505. doi: 10.1016/j.jand.2011.11.014

Roelefs, J., Muris, P., Huibers, M., Peeters, F., & Arntz, A. (2006). On the measurement of rumination: A psychometric evaluation of the ruminative response scale and the rumination on sadness scale in undergraduates. *Journal of Behavior Therapy and Experimental Psychiatry, 37,* 299–313. doi: 10.1016/j.jbtep.2006.03.002

Rothemund, Y., Preuschhof, C., Bohner, G., Bauknecht, H.-C., Klingebiel, R., Flor, H., & Klapp, B. F. (2007). Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *NeuroImage, 37*(2), 410-421. doi: 10.1016/j.neuroimage.2007.05.008

Scharmüller, W., Übel, S., Ebner, F., & Schienle, A. (2012). Appetite regulation during food cue exposure: A comparison of normal-weight and obese women. *Neuroscience Letters, 518,* 106–110. doi: 10.1016/j.neulet.2012.04.063

Seger, C. A., & Cincotta, C. M. (2006). Dynamics of frontal, striatal, and hippocampal systems during rule learning. *Cerebral Cortex, 16*(11), 1546-1555. doi: 10.1093/cercor/bhj092

Shick, S. M., Wing, R. R., Klem, M. L., McGuire, M. T., Hill, J. O., & Seagle, H. (1998). Persons successful at long-term weight loss and maintenance continue to consume a low-energy, low-fat diet. *Journal of the American Dietetic Association, 98*(4), 408-413. doi: 10.1016/s0002-8223(98)00093-5

Siep, N., Roefs, A., Roebroeck, A., Havermans, R., Bonte, M. L., & Jansen, A. (2009). Hunger is the best spice: An fMRI study of the effects of attention, hunger and calorie content on food reward processing in the amygdala and orbitofrontal cortex. *Behavioural Brain Research, 198(1),* 149-158. doi: 10.1016/j.bbr.2008.10.035

Simmons, W. K., Avery, J. A., Barcalow, J. C., Bodurka, J., Drevets, W. C., & Bellgowan, P. (2012). Keeping the body in mind: Insula functional organization and functional connectivity integrate interoceptive, exteroceptive, and emotional awareness. *Human Brain Mapping*. doi: 10.1002/hbm.22113

Simmons, W. K., Martin, A., & Barsalou, L. W. (2005). Pictures of appetizing foods activate gustatory cortices for taste and reward. *Cerebral Cortex, 15*, 1602–1608. doi: 10.1093/cercor/bhi038

Singh-Curry, V., & Husain, M. (2009). The functional role of the inferior parietal lobe in the dorsal and ventral stream dichotomy. *Neuropsychologia, 47(6),* 1434-1448. doi: 10.1016/j.neuropsychologia.2008.11.033

Small, D. M., Zald, D. H., Jones-Gotman, M., Zatorre, R. J., Pardo, J. V., Frey, S., & Petrides, M. (1999). Human cortical gustatory areas: A review of functional neuroimaging data. *NeuroReport, 10*(1), 7-13. doi: 10.1097/00001756-199901180-00002

Small, D. M., Zatorre, R. J., Dagher, A., Evans, A. C., & Jones-Gotman, M. (2001). Changes in brain activity related to eating chocolate. *Brain, 124*(9), 1720-1733. doi:

10.1093/brain/124.9.1720

Spielberg, J. M., Miller, G. A., Warren, S. L., Engels, A. S., Crocker, L. D., Banich, M. T., ... & Heller, W. (2012). A brain network instantiating approach and avoidance motivation. *Psychophysiology, 49(9),* 1200-1214. doi: 10.1111/j.1469-8986.2012.01443.x

Stern, E. R., Welsh, R. C., Gonzalez, R., Fitzgerald, K. D., Abelson, J. L., & Taylor, S. F. (2012). Subjective uncertainty and limbic hyperactivation in obsessive- compulsive disorder. *Human Brain Mapping*. doi: 10.1002/hbm.22038

Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. G., & Small, D. M. (2008). Relation of reward from food intake and anticipated food intake to obesity: A functional magnetic resonance imaging study. *Journal of Abnormal Psychology, 117*(4), 924-935. doi: 10.1037/a0013600

Stice, E., Spoor, S., Ng, J., & Zald, D. H. (2009). Relation of obesity to consummatory and anticipatory food reward. *Physiology & Behavior, 97*(5), 551-560. doi: 10.1016/j.physbeh.2009.03.020

Stoeckel, L. E., Weller, R. E., Cook, E. W., Twieg, D. B., Knowlton, R. C., & Cox, J. E. (2008). Widespread reward-system activation in obese women in response to pictures of high-calorie foods. *NeuroImage, 41*(2), 636-647. doi: 10.1016/j.neuroimage.2008.02.031

Strange, B. A., Henson, R. N. A., Friston, K. J., & Dolan, R. J. (2001). Anterior prefrontal cortex mediates rule learning in humans. *Cerebral Cortex, 11(11),* 1040-1046. doi: 10.1093/cercor/11.11.1040

Stroebe, W., Mensink, W., Aarts, H., Schut, H., & Kruglanski, A. W. (2008). Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology, 44*(1), 26-36. doi: 10.1016/j.jesp.2007.01.005

Stroebe, W., Papies, E. K., & Aarts, H. (2008). From homeostatic to hedonic theories of eating: Self-regulatory failure in food-rich environments. *Applied Psychology, 57,* 172-193. doi: 10.1111/j.1464-0597.2008.00360.x

Stunkard, A. J., Faith, M. S., & Allison, K. C. (2003). Depression and obesity. *Biological Psychiatry, 54*, 330–337. doi: 10.1016/S0006-3223(03)00608-5

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29*(1), 71-83. doi: 10.1016/0022-3999(85)90010-8

Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain. 3-Dimensional proportional system: an approach to cerebral imaging*. New York: Thieme Medical Publishers.

Tataranni, P. A., Gautier, J.-F., Chen, K., Uecker, A., Bandy, D., Salbe, A. D., . . . Ravussin, E. (1999). Neuroanatomical correlates of hunger and satiation in humans using positron emission tomography. *Proceedings of the National Academy of Sciences, 96*(8), 4569-4574. doi: 10.1073/pnas.96.8.4569

Thayer, R. E., Newman, J. R., & McClain, T. M. (1994). Self-regulation of mood: Strategies for changing a bad mood, raising energy, and reducing tension. *Journal of Personality and Social Psychology, 67*, 910–925. doi: 10.1037/0022-3514.67.5.910

Thomas, E. J., Elliott, R., McKie, S., Arnone, D., Downey, D., Juhasz, G., ... & Anderson, I. M. (2011). Interaction between a history of depression and rumination on neural response to emotional faces. Psychological Medicine, 41(9), 1845-1855. doi: 10.1017/S0033291711000043

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes

precedence over impulse control: If you feel bad, do it! *Journal of Personality and Social Psychology, 80,* 53–67. doi: 10.1037/0022-3514.80.1.53

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27(3),* 247-259. doi: 10.1023/A:1023910315561

Tugade, M., & Fredrickson, B. (2007). Regulation of positive emotions: Emotion regulation strategies that promote resilience. *Journal of Happiness Studies, 8*(3), 311-333. doi: 10.1007/s10902-006-9015-4

Turner, S. A., Luszczynska, A., Warner, L., & Schwarzer, R. (2010). Emotional and uncontrolled eating styles and chocolate chip cookie consumption: A controlled trial of the effects of positive mood enhancement. *Appetite, 54,* 143–149. doi: 10.1016/j.appet.2009.09.020

Udo, T., Grilo, C. M., Brownell, K. D., Weinberger, A. H., DiLeone, R. J., & McKee, S. A. (2013). Modeling the effects of positive and negative mood on the ability to resist eating in obese and non-obese individuals. *Eating Behaviors, 14,* 40–46. doi: 10.1016/j.eatbeh.2012.10.010

Van der Laan, L. N., De Ridder, D. T. D., Viergever, M. A., & Smeets, P. A. M. (2011). The first taste is always with the eyes: a meta-analysis on the neural correlates of processing visual food cues. *Neuroimage, 55(1),* 296-303. doi: 10.1016/j.neuroimage.2010.11.055

van Eijndhoven, P., van Wingen, G., Fernández, G., Rijpkema, M., Verkes, R. J., Buitelaar, J., & Tendolkar, I. (2011). Amygdala responsivity related to memory of emotionally neutral stimuli constitutes a trait factor for depression. *NeuroImage, 54(2),* 1677-1684. doi: 10.1016/j.neuroimage.2010.08.040

VanWormer, J. J., Martinez, A. M., Martinson, B. C., Crain, A. L., Benson, G. A., Cosentino, D.

L., & Pronk, N. P. (2009). Self-weighing promotes weight loss for obese adults. *American Journal of Preventive Medicine, 36*(1), 70-73. doi: 10.1016/j.amepre.2008.09.022

Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science, 11*(3), 249-254. doi: 10.1111/1467-9280.00250

Wagner, G., Koch, K., Reichenbach, J. R., Sauer, H., & Schlösser, R. G. (2006). The special involvement of the rostrolateral prefrontal cortex in planning abilities: an event-related fMRI study with the Tower of London paradigm. *Neuropsychologia, 44(12),* 2337-2347. doi: 0.1016/j.neuropsychologia.2006.05.014

Weiss, E. C., Galuska, D. A., Khan, L. K., Gillespie, C., & Serdula, M. K. (2007). Weight regain in U.S. adults who experienced substantial weight loss, 1999–2002. *American Journal of Preventive Medicine, 33*, 34–40. doi: 10.1016/j.amepre.2007.02.040

Whitmer, A. J. & Banich, M. T. (2007). Inhibition versus switching deficits in different forms of rumination. *Psychological Science, 18*, 546-553. doi: 10.1111/j.1467-9280.2007.01936.x

Wing, R. R., Papandonatos, G., Fava, J. L., Gorin, A. A., Phelan, S., McCaffery, J., & Tate, D. F. (2008). Maintaining large weight losses: The role of behavioral and psychological factors. *Journal of Consulting and Clinical Psychology, 76*(6), 1015-1021. doi: 10.1037/a0014159

Wittfoth, M., Schardt, D. M., Fahle, M., & Herrmann, M. (2009). How the brain resolves high conflict situations: double conflict involvement of dorsolateral prefrontal cortex. *NeuroImage, 44(3),* 1201-1209. doi: 10.1016/j.neuroimage.2008.09.026

Wyatt, H. R., Grunwald, G. K., Mosca, C. L., Klem, M. L., Wing, R. R., & Hill, J. O. (2002). Long-term weight loss and breakfast in subjects in the National Weight Control Registry.

Obesity, 10(2), 78-82. doi: 10.1038/oby.2002.13

Yeomans, M. R. & Coughlan, E. (2009). Mood-induced eating: Interactive effects of restraint and tendency to overeat. *Appetite, 52*(2), 290–298. doi: 10.1016/j.appet.2008.10.006

Yin, H. H., Knowlton, B. J., & Balleine, B. W. (2004). Lesions of dorsolateral striatum preserve outcome expectancy but disrupt habit formation in instrumental learning. *European Journal of Neuroscience, 19*(1), 181-189. doi: 10.1111/j.1460-9568.2004.03095.x