# Brain Activation in Restrained and Unrestrained Eaters: An fMRI Study

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Restraint theory has been used to model the process that produces binge eating. However, there is no satisfactory explanation for the tendency of restrained eaters (REs) to engage in counterregulatory eating, an ostensible analogue of binge eating. Using functional magnetic resonance imaging (fMRI), the authors investigated brain activation of normal weight REs ($N = 9$) and unrestrained eaters (UREs; $N = 10$) when fasted and fed and viewing pictures of highly and moderately palatable foods and neutral objects. When fasted and viewing highly palatable foods, UREs showed widespread bilateral activation in areas associated with hunger and motivation, whereas REs showed activation only in the cerebellum, an area previously implicated in low-level processing of appetitive stimuli. When fed and viewing high palatability foods, UREs showed activation in areas related to satiation and memory, whereas REs showed activation in areas implicated in desire, expectation of reward, and goal-defined behavior. These findings parallel those from behavioral research. The authors propose that the counterintuitive findings from preload studies and the present study are due to the fact that REs are less hungry than UREs when fasted and find palatable food more appealing than UREs when fed.

*Keywords:* restraint, fMRI, appetite, hunger level

*Restrained eating* refers to cognitively mediated efforts to restrict eating for the purpose of weight control. Restraint theory was developed by Herman and Polivy (1980), and the Restraint Scale (RS; Herman & Polivy, 1980) was designed to measure the restrained eating construct. Restraint has been used to model eating disregulation as well as the development and maintenance of bulimia nervosa (Polivy & Herman, 1985). The classic study illustrating the different eating patterns of restrained and unrestrained eaters (Herman & Mack, 1975) compared their intake of ice cream under two conditions. In a between-subjects analysis, ice cream consumption during an ostensible taste test was compared in the absence of a preload or following consumption of one or two milkshake preloads. The preloads produced opposite responses in the two groups; they decreased the intake of unrestrained eaters and increased the intake of restrained eaters. The counterintuitive increase in consumption of restrained eaters following the preload was called "counterregulatory eating" because restrained eaters did not just fail to reduce their intake after the preload; they actually increased it. The interaction between restraint and preload status has been replicated several times, though the significant interaction is due as much or more to the postpreload decrease in eating by unrestrained eaters than to an increase in post-preload eating in restrained eaters (Lowe, 1993; Ruderman, 1986).

Herman and Polivy's (1984) explanation of counterregulatory eating is based on two assumptions. First, it is assumed that restrained eaters' concerns about body weight leads them to impose a cognitively defined diet boundary on their food intake. Second, because of their weight loss aspirations, restrained eaters are assumed to often ignore signals of hunger, which gradually undermines their ability to generate or recognize normal hunger signals. At the same time, their "all or none" approach to dieting makes them vulnerable to overeating when their dietary standards are violated by disinhibiting influences such as consumption of diet-forbidden food or the experience of emotional distress (the so-called what the hell effect). Their history of repeated disinhibiton-induced overeating is assumed to shift their "satiety boundary" upward so that when they become disinhibited, food intake continues beyond the point at which further food intake would normally be inhibited. Restraint theory has also been extended to a *spiral model of dieting*, in which repeated dieting and overeating can gradually intensify and spiral into a clinical eating disorder (Heatherton & Polivy, 1992).

In recent years, four challenges to the validity of restraint theory have emerged. First, research that has decomposed the restrained eating construct has shown that no specific type of dieting can account for the counterregulatory eating pattern of restrained eaters (Lowe, 1993; Lowe & Kral, 2006; Lowe & Thomas, 2009). Second, restrained eaters assessed by various measures of restrained eating are not actually in negative energy balance—that is, they are not eating less than needed to maintain their weight (Stice,

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Cooper, Schoeller, Tappe, & Lowe, 2007; Stice, Fisher, & Lowe, 2004). Third, if measures of restrained eating reflect food restriction, then they should predict weight loss over time. However, studies have found that both the RS and other measures of restraint (Stice et al., 2004) and dieting (Lowe et al., 2006) prospectively predict weight gain. Fourth, if dieting is a major cause of binge eating and bulimia nervosa (Polivy & Herman, 1985), then individuals who are bulimic and who diet most frequently should binge the most, and normal weight individuals who lose weight on a diet should show increased bulimic symptoms. However, the opposite has been found in studies by Lowe, Gleaves, and Murphy-Eberenz (1998) and by Presnell and Stice (2003), respectively.

In addition to restrained eaters' vulnerability to counterregulatory eating, research by Herman, Polivy, and others over the past 35 years has produced evidence of a wide variety of cognitive, social, emotional, and behavioral anomalies in restrained eaters (Herman & Polivy, 1980, 2004, 2005). However, despite many hundreds of studies that have been published on restrained eating, no satisfactory explanation exists for either counterregulatory eating or the other anomalous behaviors exhibited by restrained eaters in past research (Lowe & Kral, 2006). The purpose of the present study was to explore the potential value of neuroimaging (in particular, functional magnetic resonance imaging [fMRI]) to elucidate the reasons that restrained eaters behave as they do. The potential value of applying neuroimaging to better understand the nature of anomalous behavioral responding by restrained eaters can be seen in research in which brain activation in normal weight and obese individuals have been compared.

In past behavioral research, researchers compared normal weight and obese people on a number of specific eating patterns and found differences between the two weight groups, whereas a similar number of studies showed no differences (Spitzer & Rodin, 1981). Neuroimaging researchers who have compared brain regions thought to be important in eating behavior in obese and normal weight individuals, by contrast, have shown much more consistent differences between these groups (Tataranni & Del Parigi, 2003). One explanation for why differences have been found more consistently in the brain than in behavior is that both the independent and dependent variables studied in neuroimaging research are typically simpler than those studied in behavioral research. For instance, behavioral studies examine the influence of variables such as consumption of forbidden foods or arousal of negative affect on actual food consumption. Neuroimaging studies typically involve the manipulation of simpler independent variables (e.g., hunger status and pictures of food) on a simpler dependent measure (activation of specific brain regions). Of importance, part of what makes behavioral output a more complex outcome than brain activation is that behavioral production involves the conscious and unconscious integration of multiple sources of information (cognitive and physiological reactions to a preload, experimenter instructions about tasting and rating different foods, interpretation of internal hunger and satiety signals, participant hypotheses about the study's purpose, self-consciousness about eating, etc.), whereas the experimental context in neuroimaging studies involves more passive and automatic brain activation to simpler stimuli, such as being food deprived or looking at pictures of food. Therefore, extending past behavioral research on restrained eating to the direct assessment of brain activation in response to relatively simple stimuli may help resolve issues left unresolved by past investigations that have involved only behavioral outcomes.

Although neuroimaging research on eating behavior is still in its early stages, there are enough studies to identify several brain regions implicated in the regulation of eating behaviors. Researchers have suggested the presence of an orexigenic network based on brain responses to the ingestion of a meal in hungry normal weight individuals (mainly in the limbic and paralimbic areas and hypothalamic region; Tataranni & Del Parigi, 2003). More specifically, the dorsolateral prefrontal cortex, orbitofrontal cortex, and insular cortex have been shown to be associated with ingestive behavior (Killgore et al., 2003). Together with the orbitofrontal cortex, the insular cortex appears to be critically involved in processing food stimuli and motivating appetitive behavior (Killgore & Yurgelun-Todd, 2006). The insular cortex is also important in monitoring internal bodily states and has been shown to have increased activation in a hungry state and decreased activation after participants are sated (Gautier et al., 2001). Other areas shown to be associated with hunger include the parahippocampal gyrus (Del Parigi et al., 2002), the superior temporal gyrus (Killgore et al., 2003; Wang et al., 2004), and the lentiform nucleus (Tataranni et al., 1999). The cerebellum has also been implicated in modulating lower level processing of food stimuli, in particular in sending satiety signals to higher level processing areas of the brain (Gautier et al., 2000; Wang et al., 2006).

Several of the brain areas noted above have also been implicated in hedonic eating behaviors. The dorsolateral prefrontal cortex has been shown to be associated with evaluation of the potential biological relevance of a stimulus and the current affective state and needs of the individual (Killgore et al., 2003). More specifically, the left dorsolateral prefrontal cortex has been shown to be activated when subjects are viewing high-calorie foods and is implicated in expectation of reward, salient decision making, and monitoring of behavioral consequences (Geliebter et al., 2006; Killgore et al., 2003; Watanabe, 1996; Watanabe, Hikosaka, Sakagami, & Shirakawa, 2002). The orbitofrontal cortex has been implicated in desire for food (Killgore et al., 2003; LaBar, Gitelman, Parrish, Nobre, & Mesulam, 2001; Wang et al., 2006) as well as motivation to eat (Killgore & Yurgelun-Todd, 2006) and expectation of a reward (O'Doherty, 2004; Rolls, 2004; Watanabe, 1996). Studies have also suggested that the saliency of food is represented in the orbitofrontal cortex (Gottfried, O'Doherty, & Dolan, 2003; Kringelbach, O'Doherty, Rolls, & Andrews, 2003). Consistent with the behavioral theory that cues can acquire motivating and hedonic properties, visual cues associated with appetizing food stimuli have been shown to activate the orbitofrontal cortex (Beaver et al., 2006). Activation of the insular cortex has also been associated with the craving and desire for food (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001; Wang et al., 2004). Lastly, the parahippocampal gyrus has been shown to play a role in the affective evaluation of food stimuli (Pelchat, Johnson, Chan, Valdez, & Ragland, 2004).

Most neuroimaging studies show activation of a much larger number of brain areas in response to hunger than in response to satiation (Tataranni & Del Parigi, 2003). This is consistent with the theory that control of energy homeostasis is inherently biased toward the defense of body weight, by way of more robust responses to energy restriction than to energy surpluses. Research

does suggest the presence of a satiation domain, represented almost exclusively by right prefrontal areas (Tataranni & Del Parigi, 2003). Additionally, the left cingulate gyrus has been shown to be activated at the termination of a meal. It is likely that there is a neural network that includes the prefrontal region, cingulate cortex, and hypothalamus that sends a signal to stop eating when satiety is reached, followed by a behavioral response.

The specific purpose of the present study was to identify the neural substrates activated in response to viewing images of highly palatable foods relative to moderately palatable foods and to neutral objects, both when participants were fasted and when they had been recently fed. Given that no functional neuroimaging study to date had examined the neural correlates of normal weight restrained and unrestrained eaters, we conducted an initial pilot study comparing the brain activation of restrained and unrestrained eaters under conditions analogous to those previously examined in eating regulation research (Herman & Polivy, 1984). On the basis of results from that pilot study (reviewed in the Method section), several predictions were made a priori. We hypothesized that normal weight-restrained eaters would be more responsive to palatable food stimuli following a meal (and normal weight-unrestrained eaters would be less responsive after the meal and more responsive prior to the meal). Specifically, it was expected that unrestrained eaters would show greater activation in response to palatable food stimuli in the fasted state, in areas associated with hunger (e.g., areas of the orbitofrontal cortex, limbic and paralimbic areas). When fed, this activation was expected to decrease, whereas activation in areas associated with satiety (e.g., cingulate gyrus) was expected to increase. Restrained eaters in the fed state, however, were expected to show more activation in areas associated with reward and hedonic experience even after being given a meal (e.g., areas of the orbitofrontal cortex). These hypotheses also follow directly from the contrasting responses of restrained and unrestrained eaters to preloads shown in past research.

## Method

The present study attempted to closely replicate the design and results of an unpublished pilot study, described here briefly. The pilot study compared the brain activation of restrained and unrestrained eaters under conditions analogous to those previously examined in eating regulation research (Herman & Polivy, 1984). The purpose of the study was to begin to identify the brain areas activated in response to viewing images of highly palatable foods relative to moderately palatable foods and to neutral objects, both when participants were fasted and recently fed. The fasted and fed states were used to approximate the no-preload and preload conditions of past eating regulation studies. The independent variables (restraint status, hunger state, type of stimuli) were tested for their relationship to activation in brain areas of interest. The dependent variable was the fMRI blood oxygen-level dependent (BOLD) signal response. Brain responses to pictures of highly palatable foods were of primary interest. Participant groups for this pilot study consisted of 3 normal weight-unrestrained women (mean age = 21 years; body mass index [BMI] = 21.03 kg/m$^2$) and 3 normal weight-restrained women (mean age = 23 years; BMI = 21.20 kg/m$^2$).

Unrestrained eaters showed widespread bilateral activation to high-palatability food stimuli in a fasted state, in areas associated with hunger and expectation of reward (e.g., medial frontal lobe, fusiform gyrus; Killgore et al., 2003; Rolls, 2004). In contrast, restrained eaters yielded no activation in the fasted state that survived statistical threshold. However, in a fed state, viewing high-palatability food stimuli, restrained eaters yielded activation in the left-hemisphere orbitofrontal cortex and temporal lobe. The areas activated in the orbitofrontal cortex have been associated with the expectation of reward and hedonic experience (Kringelbach, 2004; Rolls, 2004; Wang et al., 2004). Under the same conditions, activation for unrestrained eaters decreased and was found in areas shown to be activated at the termination of a meal (e.g., cingulate gyrus; Tataranni & Del Parigi, 2003). These results suggest that restrained eaters may eat more food when fed than fasted because they paradoxically find food more rewarding when their appetite has been "primed" by recent food intake. Although the reason for the brain activation patterns of restrained eaters is unclear, these results could be useful in suggesting alternative explanations for restrained eaters' susceptibility to overeating (Lowe & Kral, 2006). The present study was identical to the pilot study in the procedure and data analysis methods, with two exceptions. In the pilot study, participants were scanned in the morning, following an evening fast (as opposed to the afternoon following a daytime fast). Additionally, SPM2 was used to analyze the data in the pilot study (as opposed to SPM5 in the present study).

### Participants

Participants were recruited from large introductory level courses at a mid-Atlantic university. Only women were invited to participate. All of the prospective participants were given a packet consisting of (a) a consent form explaining the purposes of the study; (b) a basic demographic sheet; (c) the Herman and Polivy (1980) RS; (d) a questionnaire assessing dieting and weight history; and (e) a questionnaire assessing food preferences. All of the questionnaires were brief (1–2 pages), and the packet took approximately 15 min to complete. Packets were completed in the classroom, immediately following the completion of class. From the larger pool of women who completed the eligibility questionnaire packets, 19 healthy right-handed students who met all criteria signed up for and participated in the experimental session. To be included in the study, participants had to be female, right-handed, with a BMI no greater then 25 kg/m$^2$ and no lower then 20 kg/m$^2$ (determined by self-reported height and weight and confirmed on site), and at least 18 years old. To be included in the study, all participants had to rate the highly palatable food images as a 6 or 7 and the moderately palatable food images as a 3 or 4 (based on a 7-point Likert scale) on the food preference questionnaire.

Participants were divided into restrained ($n$ = 9) and unrestrained ($n$ = 10) eaters on the basis of their score on the RS (Herman & Polivy, 1980). From an initial sample size of 50 potential participants, RS scores were divided into quartiles. Restrained eaters were chosen from the highest quartile (a score of 16 or higher) and unrestrained eaters from the lowest quartile (a score of 10 or lower) of the distribution of RS scores. Early restraint research used a sample-specific median split to divide groups into restrained and unrestrained eaters; medians in these studies have ranged from 12 to 17. For the past decade or so, most studies have simply used a score of 15 to divide the sample (Jarry, Polivy,

Herman, Arrowood, & Pliner, 2006; Trottier, Polivy, & Herman, 2005).

Participants were also screened with a health history questionnaire and a weight and dieting history questionnaire. Women who reported that they were currently on a diet to lose weight were excluded from the study because evidence indicates that dieters differ from restrained eaters in a number of ways (Lowe & Kral, 2006). Additionally, women more than five pounds below their highest adult weight were excluded to help ensure that weight suppression, which is normally higher in restrained eaters (Lowe, 1984), was not confounded with restraint group. In both groups, participants were excluded from the study if they had a history of an eating disorder or were current binge eaters (assessed with the weight and dieting history questionnaire). Current smokers and women who recently began taking any medication known to affect weight and appetite (within the past 6 months) were also excluded. Table 1 shows the characteristics of the participants. The sample was overwhelmingly Caucasian, with only 1 African American in the unrestrained group.

## Experimental Protocol

Participants meeting the appropriate inclusion/exclusion criteria were invited to participate in the experimental session, which involved the use of a 1.5-Tesla GE EchoSpeed Plus scanner with echoplanar imaging capability (33mT/m, rapid switching gradients) required for fMRI data collection. The protocol was approved by the Institutional Review Board of Drexel University. A written informed consent was obtained from all participants prior to participation. Participants were instructed to begin an 8-hr fast at 8:00 a.m. or 8:30 a.m. on the morning of their session, and scanning was conducted at 4:00 p.m. or 4:30 p.m. on the same day. Fasting was confirmed through self-report upon arrival. Participants gave subjective ratings of hunger and fullness, using Likert-type scales.

The food stimuli were chosen on the basis of images used in the pilot study. Images were obtained through Google Image. Stimuli were presented to participants using Presentation ® (Version 9.81) through magnetic resonance (MR)-compatible goggles, and responses were recorded using an MR-compatible response pad (Resonance Technologies, Inc.). Highly palatable images depicted French fries, pizza, a chocolate chip cookie, and an ice cream sundae. Moderately palatable images were an apple, a slice of bread, a bowl of carrots, and a plain baked potato. Neutral stimuli were pictures of objects (e.g., tree, car, rock, stapler) and were as closely matched as possible to the food stimuli, based on the size of the pictured objects.

Table 1
*Characteristics of the Participants*

| Variable | Restrained eaters (n = 9) | Unrestrained eaters (n = 10) |
|---|---|---|
| Age (years) | 19.7 years (± 1.09) | 20.6 years (± 2.71) |
| BMI | 22.1 kg/m² (± 0.59) | 21.5 kg/m² (± 1.86) |
| RS | 16.8 (± 3.15) | 7.3 (± 2.67) |
| Weight suppression | 2.9 lbs. (± 2.94) | 3.9 lbs. (± 2.68) |

*Note.* BMI = body mass index; RS = Restraint Scale.

Participants' heads were positioned inside an 8-channel head coil. They were placed in the magnetic resonance imaging (MRI) scanner and shown images of food and nonfood stimuli. The scanning procedure took approximately 30 min. Participants were then removed from the scanner and were given two cans of Vanilla Ensure (a total of 500 calories; 16 oz; 473 ml). Participants waited 45 min after consuming the liquid meal before they were placed back into the MRI scanner to allow the energy and nutrients from the meal to be absorbed and travel to the brain. During their 45-min wait, participants either read magazines or did homework that they had brought with them to the session. Ratings of hunger and fullness were given again just prior to returning to the scanner to confirm that participants felt less hungry and more full after the meal than before (see Table 2). The waiting period was set at 45 min because that period is long enough to ensure that sufficient time had passed for the preload's energy and nutrients to be absorbed and travel to the brain (a process that begins about 20 min after ingestion), but not so long (e.g., 90 min or longer) that some participants might begin to feel hungry again. The same initial scanning procedure (showing pictures of food and nonfood items) was repeated after the 45-min waiting period. Following the second fMRI scan, participants' heights and weights were measured. They were then paid $75 for their participation in the experimental session.

## Measures

Several measures were created to identify demographic information and general weight and dieting information. The demographics questionnaire asked about participants' age, ethnicity, height, weight, smoking status, current medications, and highest level of education completed. The dieting and weight history questionnaire asked participants to respond to questions relating to weight suppression: "What is the most you have ever weighed since reaching your current height" and "In your lifetime, how many times have you lost 10 lbs or more?" It also asked about current dieting status: "Are you currently dieting to lose weight," and asked whether the participant had ever been diagnosed with an eating disorder. Current or past binge eating was addressed with two questions: "During the past 3 months, did you often eat an unusually large amount of food within a two-hour period (an amount that most people would agree is unusually large)" and "During the times when you ate an unusually large amount of food, did you often feel you could not stop eating or control what or how much you were eating?"

The food preference questionnaire asked participants to rate on a 7-point Likert scale from 1 (*not at all*) to 7 (*very much*) how much they liked the foods that they would be viewing in the fMRI scanner. In order to be included in the study, participants had to rate the highly palatable food stimuli as a 6 or 7 and the moderately palatable food stimuli as a 3 or 4.

The RS (Herman & Polivy, 1980) is a 10-item self-report questionnaire designed to identify individuals who are chronically concerned about their weight and try to control it by limiting their food intake. The RS has good internal consistency in normal weight individuals (Cronbach's $\alpha > .75$; Allison, Kalinsky, & Gorman, 1992; Herman & Polivy, 1975; Klem, Klesges, Bene, & Mellon, 1990). Test–retest reliability coefficients range from .74 to .95 (Allison et al., 1992; Klesges,

Table 2
*Measures of Hunger Before and After Eating A Meal[a]*

| Question | Restrained | Unrestrained |
|---|---|---|
| Time 1 | | |
| How hungry do you feel right now? | 6.56 (± 1.33) | 7.30 (± 0.68) |
| How strong is your desire to eat right now? | 7.00 (± 1.73) | 7.50 (± 0.85) |
| How much food do you think you could eat right now? | 6.44 (± 1.33) | 7.10 (± 1.19) |
| How full does your stomach feel right now? | 2.56 (± 1.42) | 1.90 (± 0.99) |
| Time 2 | | |
| How hungry do you feel right now? | 3.44 (± 2.29) | 3.70 (± 1.49) |
| How strong is your desire to eat right now? | 3.89 (± 2.62) | 4.20 (± 1.62) |
| How much food do you think you could eat right now? | 4.33 (± 2.06) | 4.10 (± 1.97) |
| How full does your stomach feel right now? | 5.44 (± 1.88) | 5.40 (± 1.43) |

[a] Values are the mean (± *SD*) item response for four questions on the verbal hunger questionnaire.

Klem, & Bene, 1989). The total score for the measure predicts disinhibitory eating in laboratory settings (Polivy & Herman, 1999).

Lastly, the verbal hunger questionnaire consists of four questions frequently used in appetite research related to hunger and fullness (e.g., Lowe, Friedman, Mattes, Kopyt, & Gayda, 2000). The validity of items in the verbal hunger questionnaire is supported by the tendency for items to vary in predicted directions, such as with fasting, refeeding, and upon administration of anorectic drugs (Blundell & Rogers, 1980; Friedman, Ulrich, & Mattes, 1999; Hill, Magson, & Blundell, 1984; Silverstone, 1982). It consists of the following four questions, rated on 9-point Likert scales: "1) How hungry do you feel right now?" (ranging from "not at all" to "as hungry as I have ever felt"); "2) How strong is your desire to eat right now?" (ranging from "very weak" to "very strong"); "3) How much food do you think you could eat right now?" (ranging from "nothing" to "a large amount"); and "4) How full does your stomach feel right now?" (ranging from "not at all full" to "very full"; Lowe et al., 2000). This measure was used to confirm that, on average, participants reported higher levels of hunger and lower levels of fullness prior to the first scan than the second scan.

*fMRI parameters.* The scanning began with the collection of high-resolution T1-weighted imaging sequences acquired in the axial plane to locate the positions for in-plane structural images. Twenty-six (whole-brain) contiguous (no-gap) 5-mm axial high-resolution T1-weighted structural slices (matrix size = 256 × 256; return time [TR] = 600; echo time [TE] = 15 ms; field of view [FOV] = 21 cm; number of excitations [NEX] = 1; and slice thickness = 5 mm) were collected for overlay of functional data onto the anatomical data. Precise localization-based standard anatomic markers (AC–PC Line) were used for all participants (Talairach & Tournoux, 1988). Functional images were acquired with a gradient-echo echoplanar free-induction decay (EPI-FID) sequence (T2-weighted: 64 × 64 matrix; FOV = 21cm; slice thickness = 5 mm; TR = 4 s; and TE = 54 ms) in the same plane as the structural images. The size of the imaging voxel was 1.72 mm × 1.72 mm × 5 mm. A total of 128 volumes, each containing 26-slice whole-brain sections, were acquired during the fMRI scan.

## Design/Paradigm

The fasted and fed states were used to approximate the no-preload and preload conditions of past eating regulation studies. Brain responses to pictures of highly palatable foods were of primary interest (as an analogue to the ice cream consumed in past preload studies) and the pictures of nonfood objects, and moderately palatable foods were included to control for the impact of visual perception and perception of food stimuli, respectively. The study was designed to measure BOLD signal responses to neutral and food-related stimuli in fasted and fed states. An event-related design was used (interstimulus interval = 4–24 ms). The design was modeled on the presently well accepted methods for fMRI (Brett, Penny, & Kiebel, 2003; Buckner & Logan, 2001). Each stimulus was shown for 2 s, and participants were asked to respond to the question "Is this a food?" using a response pad designed for use in the MRI environment (Resonance Technologies, Inc.). There were three stimulus conditions: highly palatable food, moderately palatable food, and a neutral condition that consisted of nonfood objects. The neutral stimuli were shown 78 times. The other two categories of experimental (food) stimuli were shown 30 times each.

*Image processing.* The postacquisition preprocessing and statistical analysis was performed using SPM5 (statistical parametric mapping; Wellcome Department of Cognitive Neurology, 2005). The standard preprocessing procedures outlined in the SPM5 manual were used (FIL Methods Group, 2007). First, slice timing correction was performed. A 3D-automated image-registration routine (six-parameter rigid body, sinc interpolation; second-order adjustment for movement) was then applied to the volumes to realign them with the first volume of the first series used as a spatial reference. All functional and anatomical volumes were then transformed into the standard anatomical space using the participant's coregistered structural image and the SPM normalization procedure (Ashburner & Friston, 1999). This procedure uses a sinc interpolation algorithm to account for brain size and position with a 12-parameter affine transformation, followed by a series of nonlinear basic function transformations using seven, eight, and seven nonlinear basis functions for the x, y, and z directions, respectively, with 12 nonlinear iterations to correct for morphological differences between the template and a given brain volume.

All volumes underwent spatial smoothing by convolution with a Gaussian kernel of 8 cubic mm full width at half maximum (FWHM).

Subject-level statistical analyses (t-maps) were created using the general linear model in SPM5. The four condition events (baseline MR signal or nontask condition, neutral objects [N], highly palatable food items [H], and moderately palatable food items [M]) were modeled using a canonical hemodynamic response function (hrf). Contrast maps were obtained through the following linear contrasts of event types for each hunger state: N + M + H versus baseline (overall effect), N − M and N − H separately, M − N and M − H, and H − N and H − M. Group-level random effects analyses were completed (Brett et al., 2003; Friston, 2003). Hunger state effects were accomplished by entering whole-brain contrasts into paired-sample t tests (as implemented in SPM5). All contrasts were set to an a priori significance threshold of $p < .001$, uncorrected, with a minimum cluster-size threshold set at eight contiguous voxels. The most liberal cluster detection correction was used ($p < .001$, cluster size of eight) based on common alpha corrections in previous fMRI pilot studies (Friston, Worsley, Frackowiak, Mazziotta, & Evans, 1994) and based on alpha corrections used in the pilot study. MNI coordinates were transformed using Brett's equation,[1] and the Talaraich Daemon was used to gather the anatomical names.[2]

## Results

Descriptive data for restrained and unrestrained eaters are reported in Table 1. There were no significant differences between restrained and unrestrained eaters on age, $t(17) = 0.847$, $p = .41$; BMI, $t(17) = 1.69$, $p = .15$; or weight suppression, $t(17) = 0.771$, $p = .45$. As intended, the mean RS score was significantly lower in unrestrained than in restrained eaters (7.3 ± 2.66 vs. 16.8 ± 3.15), $t(17) = −7.096$, $p < .001$.

The measure of hunger confirmed that, on average, participants reported high levels of hunger prior to the first scan. Hunger ratings significantly decreased and fullness ratings significantly increased following the preload (see Table 2). Of note, the average rating on the fullness item was a 5.4 (based on a 9-point Likert scale), suggesting that participants perceived themselves to be moderately full 45 min after consuming the Ensure. There were no significant differences between levels of hunger reported by restrained and unrestrained eaters, either before or after the consumption of the Ensure.

### fMRI Data

Prior to testing for the specific hypotheses of interest, we first determined that the two manipulated variables (picture type and fasting state) had a clear-cut differential impact on brain activation by identifying cerebral regions activated in response to presentations of the combined (high and moderate) food stimuli relative to nonfood stimuli collapsing across group and hunger state (one-sample t tests). Additionally, prior to random effects analysis (also referred to as RFX) of participant groups, we analyzed contrast images in paired-sample t tests to test for effects of hunger state (fasted vs. fed) across both participant groups. These analyses are not reported in full, because they were merely used as a way to check that the manipulations at a basic level were powerful enough

to affect brain activation. As expected, these analyses indicated that in a fasted state, highly palatable stimuli yielded differential activation in a number of brain areas compared with moderately palatable stimuli. Differences in brain activation were also evident in the fasted relative to the fed state, averaged across highly and moderately palatable foods. Comparing the hunger and sated conditions produced clear-cut differences in levels of brain activation to pictures of both highly and moderately palatable food.[3]

At the paired-sample t test level, *RFX* refers to an analysis of residual activation in one group after overlapping activation of another group has been removed. To describe this, the terminology "restrained versus unrestrained" (RvU, and vice versa) is used. Through this kind of analysis, activation can be analyzed above and beyond that which might be shared in common with another relevant condition. As noted in the introduction, the primary hypothesis of the study was that normal weight-restrained eaters would be more responsive to food stimuli following a meal (and normal weight-unrestrained eaters would be less responsive after the meal and more responsive prior to the meal).

### Fasted State

First, the analysis was done on the fasted-state data, with highly versus moderately palatable stimuli (HvM) (see Figure 1). Under the same conditions (fasted, HvM), unrestrained relative to restrained (UvR) yielded significantly significant residual activation in the right superior temporal gyrus and the left parahippocampal gyrus. Unrestrained eaters also had significant activation in the left lentiform nucleus (putamen) and the left middle frontal gyrus (in the dorsolateral prefrontal cortex) (see Table 3). Under the same conditions (fasted, HvM), restrained relative to unrestrained (RvU) yielded significant activation in the cerebellum (specifically, the cerebellar lingual; see Table 4).

### Fed State

In a fed state, when examining HvM palatability stimuli, RvU showed significant activation again in the cerebellum, this time in the pyramis. We also found activation in two areas of the left middle frontal gyrus (part of the orbitofrontal cortex). Other areas activated include the left superior frontal gyrus and the left precentral gyrus, both in the dorsolateral prefrontal cortex (Brodmann area 9). Additionally, restrained eaters showed activation in the left insular cortex (see Table 4, Figure 1). When fed and viewing HvM palatability food stimuli, UvR yielded statistically significant activation in the left cingulate gyrus, the right inferior frontal gyrus, right precuneus, and left parahippocampal gyrus (see Table 3, Figure 1).

The above analyses specifically examined the activation in response to highly palatable food stimuli in restrained and unrestrained eaters. Results from the pilot study suggested that the left prefrontal activation in restrained eaters when fed was specific to

**Fasted State**                              **Fed State**
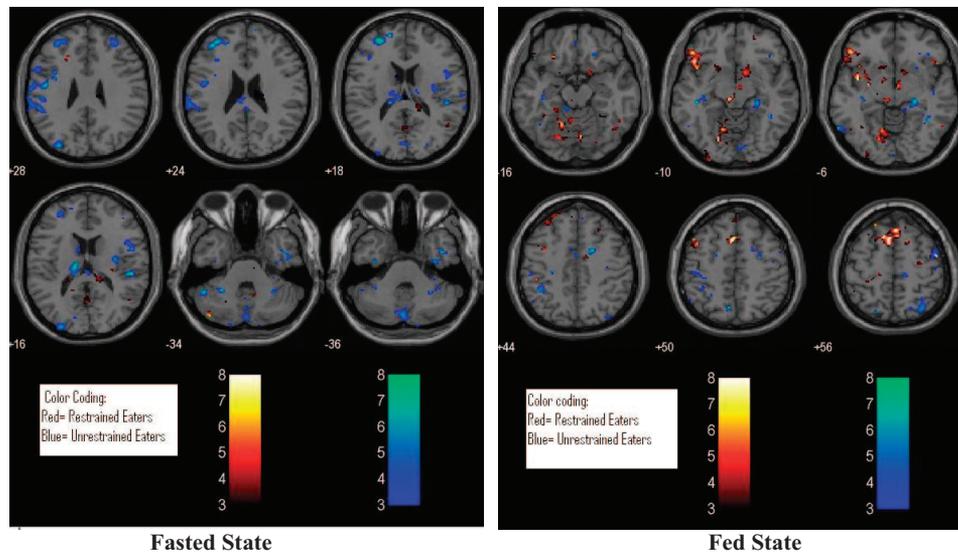
*Figure 1.* Brain activation in a fasted and fed state when viewing highly palatable food stimuli. Color-coded areas represent activation in restrained eaters (red) and unrestrained eaters (blue). Upper limit *z* score of 8 (represented by color-coded bars) was used to portray activated areas.

highly palatable foods. In an attempt to further examine that finding, we included analyses of activation in the present study in response to moderate stimuli in a fed state. We chose the analysis of moderate minus neutral to replicate the analysis in the pilot study and to control for nonfood objects. When fed and viewing moderately palatable food stimuli, unrestrained eaters had activation in the left uncus and left cingulate gyrus. We found activation for restrained eaters in the left and right anterior cingulate gyrus.

To help clarify the relative activation findings, we examined conjunctive activation using MRIcroN (Rorden, Bonilha, & Nichols, 2007). Z-score maps for restrained and unrestrained eaters when fasted and fed and viewing highly palatable food items were overlaid on a single-subject template provided by MRIcroN software. The statistical map was set to show values from 3 to 8: values less than 3 were not shown, based on the minimum z scores

produced in the RFX analyses. The upper threshold of eight was also imposed on the basis of the statistical maps produced by SPM5. We identified anatomical localizations of the yellow-activated brain regions using the Talairach daemon. When fasted, restrained and unrestrained eaters had overlapping activation in the left superior parietal lobule as well as in the left fusiform gyrus (in the occipital lobe). When fed, we found overlapping activation in the right brainstem (pons) and the parahippocampal gyrus.

## Discussion

It was hypothesized that normal weight-restrained (relative to unrestrained) eaters would be more responsive to food following a meal than when hungry and that unrestrained (relative to restrained) eaters would be less responsive after the meal and more responsive when hungry. Results were expected to parallel, at a

Table 3

*Local Maxima of BOLD fMRI Signal Change for the Fasted State and the Fed State, Unrestrained Compared With Restrained Eaters (High-Moderate) P − uncorrected < .001; Minimum Cluster Size = 8*

| Region | Hemisphere | X | Y | Z | Z score |
|---|---|---|---|---|---|
| Fasted | | | | | |
| Superior temporal gyrus | R | 48 | −3 | −4 | 4.28 |
| Parahippocampal gyrus | L | −15 | −19 | −27 | 4.12 |
| Putamen | L | −21 | −2 | −2 | 4.10 |
| Middle frontal gyrus | L | −42 | 16 | 29 | 3.71 |
| Fed | | | | | |
| Cingulategyrus | L | −9 | −37 | 31 | 4.21 |
| Inferior frontal gyrus | R | 39 | 7 | 31 | 3.96 |
| Precuneus | R | 12 | −45 | 31 | 3.70 |
| Parahippocampal gyrus | L | −18 | −51 | −3 | 3.53 |

*Note.* BOLD = blood oxygen-level dependent; fMRI = functional magnetic resonance imaging; R = right; L = left.

Table 4

*Local Maxima of BOLD fMRI Signal Change for the Fasted State and the Fed State, Restrained Compared With Unrestrained Eaters (High-Moderate) P − uncorrected < .001; Minimum Cluster Size = 8*

| Region | Hemisphere | X | Y | Z | Z score |
|---|---|---|---|---|---|
| Fasted | | | | | |
| Cerebellar lingual | L | −3 | −41 | −13 | 4.13 |
| Fed | | | | | |
| Cerebellum (pyramis) | R | 18 | −80 | −29 | 4.59 |
| Middle frontal gyrus | L | −48 | 59 | 17 | 4.52 |
| Superior frontal gyrus | L | −33 | 51 | 30 | 4.32 |
| Insula | L | −30 | 22 | 18 | 4.28 |
| Middle frontal gyrus | L | −36 | 25 | −26 | 4.19 |
| Precentral gyrus | L | −42 | 4 | 33 | 4.07 |

*Note.* BOLD = blood oxygen-level dependent; fMRI = functional magnetic resonance imaging; R = right; L = left.

neurophysiological level, the eating patterns shown by unrestrained and restrained eaters in no-preload and preload conditions in past research (Herman & Polivy, 1984). Overall, the results we found in the present study supported and extended those found in the pilot study. Brain activation in restrained eaters differed from that of unrestrained eaters matched on BMI, and, in line with predictions, these differences emerged when comparing participants across states of hunger and fullness. In the discussion below of differences in brain activation, the literature supporting the potential role of each brain area was reviewed in the introduction and is not repeated here.

When fasted, unrestrained eaters showed significant activation to highly palatable food stimuli in a wide variety of brain areas shown to be associated with hunger (superior temporal gyrus, parahippocampal gyrus, dorsolateral prefrontal cortex, lentiform nucleus), expectation of reward (left dorsolateral prefrontal cortex), and reinforcement (lentiform nucleus). In contrast, restrained eaters yielded significant activation only in the cerebellar lingual, an area implicated in modulating lower level processing of food stimuli. However, when fed and viewing highly palatable foods, restrained eaters showed activation in the orbitofrontal cortex (associated with hunger, desire for food, and expectation of a reward), the left dorsolateral prefrontal cortex (implicated in reward, decision making, and monitoring of behavioral consequences), and the left insular cortex (associated with desire for food). Lastly, we also found activation for restrained eaters in the cerebellum, an area also activated when restrained eaters were fasted and viewed highly palatable food items. However, when fed, the specific area of the cerebellum activated was the pyramis, which is specifically linked to motor processing and planning (Crossman & Neary, 2000). In contrast, fed unrestrained eaters yielded activation in the right prefrontal cortex (associated with inhibition of further eating) and the left cingulate gyrus (shown to be activated at the termination of a meal). Fed unrestrained eaters also showed greater activation in areas associated with memory (precuneus, parahippocampal gyrus).

The contrast between the level of activation in restrained and unrestrained eaters in a fasted state is striking. The absence of activation in brain areas associated with hunger in this study (even in the MRIcroN analyses, in which restrained eaters showed no detectable activation in areas associated with hunger) along with activation in a brain area associated with satiety (the cerebellum) converge in implying that when food deprived, restrained eaters do not experience typical hunger or at least experience it differently than do unrestrained eaters. Additionally, the hypothesis that fed-restrained eaters would show greater activation in brain areas associated with appetitive motivation in response to palatable food stimuli was supported. We then analyzed moderately palatable stimuli to determine whether, as in the pilot study, the effect was specific to highly palatable foods. This result was confirmed as well. When fed and viewing moderately palatable food stimuli, both groups had activation in areas typically associated with satiation rather than reward.

The RFX analyses produced areas of significant residual activation for restrained and unrestrained eaters in particular conditions; however, this does not necessarily mean that the group whose activation was subtracted out displayed no activation at all in these conditions. For example, the fact that unrestrained eaters showed widespread residual activation when fasted and viewing

highly palatable foods does not necessarily mean that restrained eaters showed no activation in these same areas. It simply means that unrestrained eaters' BOLD signal activation was sufficiently strong that it survived the subtraction analyses despite any overlapping activation by restrained eaters. We therefore used MRIcroN (Rorden et al., 2007) to determine the extent to which participants in one group evidenced activation that may not have emerged from our RFX analyses because of the subtraction procedure. When fasted and viewing highly palatable foods, the areas of overlapping brain activation revealed by MRIcroN were in visual and visuospatial processing areas. The fact that restrained eaters showed no significant activation in areas associated with hunger or motivation to eat indicates that unrestrained eaters' residual activation in this condition was not simply stronger than activation levels shown by restrained eaters but that the restrained eaters themselves did not show significant activation. If the unrestrained eaters are considered to have a normal appetite system that appropriately regulates food intake (by consistently creating hunger signals that motivate eating under conditions of energy depletion and consistently generating satiety signals that terminate eating under conditions of energy repletion; see Herman & Polivy, 1984), then this evidence is consistent with the hypothesis that restrained eaters do not generate normal hunger signals when energy deprived. This finding suggests that restrained eaters' reduced consumption in the no-preload condition of preload studies occurs not because their "diet boundary" is intact (Herman and Polivy, 1984; Lowe, 1993) but because they are not as highly motivated to eat in this condition relative to unrestrained eaters.

The MRIcroN comparisons in the fed condition indicated that restrained and unrestrained eaters showed overlapping activation in an area associated with memory (parahippocampal gyrus), which may be due to the fact that identical images were shown during the two scanning procedures. There was also overlapping activation in the pons, an area of the brain thought to relay sensory information and regulate respiration. Of note, when examined on their own (i.e., without subtraction analyses), fed unrestrained eaters viewing palatable foods did not exhibit significant activation in brain areas associated with reward or hedonic experience. It therefore appears that restrained eaters' activation in these areas was not simply stronger than that shown by unrestrained eaters but that the unrestrained eaters did not themselves exhibit any activation that survived statistical threshold. Taken together, these MRIcroN exploratory analyses suggest that the restrained and unrestrained eaters studied here differed qualitatively, not just quantitatively, in their brain responses to highly palatable foods in fasted and fed states.

Overall, the results parallel findings in the behavioral literature on eating regulation in restrained and unrestrained eaters. In preload studies using the RS to classify participants, a significant crossover interaction between restraint group and preload status on food consumption is typically found. This interaction is due to restrained eaters consuming less than unrestrained eaters without a preload and more than unrestrained eaters following a preload (Herman & Polivy, 1984; Ruderman, 1986). The difference in intake between restrained and unrestrained eaters is typically larger in the preload than in the no-preload condition (with restrained eaters eating more than unrestrained eaters in the former and less in the latter). The much more widespread brain activation shown in this study by unrestrained than restrained eaters when

fasted and viewing pictures of palatable food is consistent with the greater ice cream intake of nonpreloaded unrestrained than restrained eaters in past eating regulation studies (Herman & Polivy, 1984). Similarly, the activation of left-sided, reward-related brain areas in the orbitofrontal cortex, dorsolateral prefrontal cortex, and insular cortex in fed restrained eaters is consistent with their tendency to increase, rather than decrease, their ice cream consumption following preloads in past studies.

These results suggest that restrained eaters may have actually experienced stronger appetitive motivation to eat palatable food when recently fed than when hungry or, alternatively, that the consumption of a preload enhances appetitive drive for palatable food, whereas fasting reduces it. This characterization could also be applied to the apparent state of appetitive motivation of nonpreloaded and preloaded restrained eaters in past studies. The counterintuitive nature of the behavioral findings has traditionally been explained by reference to the diet-breaking effects of a preload in combination with impairments in restrained eaters' hunger and satiety responses (stemming from their history of dieting and overeating; Herman & Polivy, 1984; Lowe, 1993). However, the obtained pattern of brain activation suggests that, relative to unrestrained eaters, restrained eaters' eating behavior in the preload paradigm reflects differences in the effects of fasting and feeding on the hedonic appeal of palatable food rather than the effects of the preload on the integrity of their dietary restraint. That is, rather than restrained eaters' diets remaining intact in a no-preload condition and being breached in a preload condition (Herman & Polivy, 1984), restrained eaters may eat more food when fed than fasted because they paradoxically find food more rewarding when their appetite has been "primed" by recent food intake. This interpretation is consistent with a great deal of evidence that dieting behavior does not account for the counterregulatory eating of restrained eaters (e.g., Lowe, 1993; Lowe & Kral, 2006; Stice et al., 2007).

An alternative interpretation might suggest that the prefrontal activation represents a modulation of limbic system-generated emotional responses to personally desired stimuli (the high-palatability foods).[4] Recent evidence suggests that functional connectivity within frontal limbic circuits (Banks, Eddy, Angstadt, Nathan, & Phan, 2007) is involved in the regulation of salient personal decision making (Barbas, 1995; Cardinal, Parkinson, Hall, & Everitt, 2002). The amygdala appears to play a role in directing attention to emotionally salient stimuli, particularly stressful or disturbing stimuli (Davidson, 2003). Under this model, response of the orbitofrontal cortex in fed restrained eaters may represent an effort to modulate fear or threat mediated by limbic activity. This would make sense psychologically because the sight of highly palatable food could represent a threat to fed restrained eaters, who tend to have elevated body dissatisfaction and fear of weight gain. The orbitofrontal cortex, perhaps particularly the left ventral medial prefrontal cortex, provides a biasing signal to avoid immediate reward, and thus maintain pursuit of one's longer term goals (Cardinal et al., 2002). If this is the case here, then one might presume that prefrontal regions are activated not as a positive reward response, but as part of a top-down modulation of lower limbic circuitry meant to cope with the appeal (and therefore the threat) of "superfluous" high-calorie foods. Further neuroimaging research will be needed to differentiate between this and the reward-based interpretation described above.

Although the reason for the paradoxical brain activation patterns of restrained eaters is unclear, these results are consistent with the hypothesis that normal weight individuals who often diet (as most restrained eaters do) are prone to weight gain and frequently diet not to become thin but to avoid becoming fat (Lowe & Butryn, 2007; Lowe & Levine, 2005; Stice et al., 2007). Restraint theory suggests that when restrained eaters are exercising restraint, their "diet boundary" is intact (Herman & Polivy, 1984), and they are consciously trying to inhibit the desire to eat. Thus, a possible alternative explanation for the differential activation of restrained eaters when fed and viewing highly palatable foods is that it is a reflection of activation of brain areas normally involved in the exercise of self-control. In other words, rather than restrained eaters responding to the palatable nature of the food stimuli, their response may be more a reflection of the self-control they are accustomed to exercising (or at least trying to exercise) when confronted with highly palatable foods. However, this interpretation is not supported by existing research on the neural correlates of self-control and behavioral inhibition. Studies suggest that the right prefrontal cortex is critical in inhibiting behavioral responses (Alonso-Alonso & Pascual-Leone, 2007; Aron, Behrens, Smith, Frank, & Poldrak, 2007), in resisting temptation (Knoch & Fehr, 2007), and in monitoring states of self (Platek et al., 2006). Other areas that have been suggested as playing a role in self-control are the subthalamic nucleus (Aron et al., 2007) and caudate tail (Li, Huang, Constable, & Sinha, 2006). Because none of these areas were activated when restrained eaters were fed and viewing highly palatable foods in the present study, it appears that the activation that we found is unlikely to reflect a manifestation of anticipatory restraint or self-control.

The majority of results found in the present study mirrored those found in the small pilot study. Nonetheless, there were differences in findings between the two studies. The pilot study revealed some areas of activation that were not identified in the present study. For example, in the pilot study, when fasted, unrestrained eaters had additional activation in the lingual gyrus, the fusiform gyrus, and superior and inferior parietal lobule (associated with attention). When fed, unrestrained eaters had additional activation in the lateral geniculum body (associated with vision). Lastly, restrained eaters had activation in the middle temporal gyrus when fed.

Additionally, the present study revealed some areas of activation that were not found in the pilot study. Restrained eaters, when fasted, had activation in the cerebellum, whereas the pilot study yielded no such activation. In the fasted state, unrestrained eaters yielded activation in two areas not shown to be activated in the pilot study: superior temporal gyrus and parahippocampal gyrus. When fed, the present study yielded activation for unrestrained eaters in the right prefrontal cortex, precuneus, and parahippocampal gyrus. These areas were not identified in the pilot study. Lastly, restrained eaters when fed yielded several areas not activated in the pilot study, including the cerebellum, the dorsolateral prefrontal cortex, and the insular cortex. A possible explanation for the differences in activation between the present study and pilot study is that the former had a greater sample size and therefore greater statistical power. Greater statistical power would produce more

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[4] We thank an anonymous reviewer for suggesting this alternative explanation.

*real* differences (i.e., differences that would be found if an even larger sample size were used). The pilot study may have produced more *false positives* (i.e., significant differences that were due to chance). An additional explanation for discrepancies in activated areas is the difference in the fasting and scanning times in the present and pilot study. In the present study, participants' fast began after breakfast, whereas in the preliminary study, their fast began after dinner. One could argue that basic physiological hunger is not as intense in the morning before the first meal (e.g., many people eat little or no food for breakfast) as it is later in the afternoon, following an early morning breakfast. The present paradigm more closely reflected the time during the day when preload studies have typically been conducted (mostly in the afternoon) and so may have greater external validity in that sense.

This study had several limitations. Although the sample size was much larger than the pilot study and was similar to that used in most fMRI studies, it was still relatively small. The average recommended sample size in neuroimaging studies is 12–15 participants per group. However, given that the observed between-groups differences replicated the pilot study, greater confidence can be placed in the reliability of the results. All sessions were conducted in a single day, introducing the possibility that order effects could have influenced the results. Participants comprised normal weight female college students, thus limiting generalizability to the general population. However, most behavioral studies done on counterregulatory eating have been done with this population. In particular, the present results cannot be generalized to men. In previous research, men have shown differential brain activation in response to satiation compared with women (Gautier et al., 2001). Men have been vastly understudied in the preload paradigm, and it is unclear whether restrained men and women would show the same behavioral presentation. Another limitation is that we did not control for phase of the participants' menstrual cycle. Circulating hormones may influence appetite and eating behavior (Davidsen, Vistisen, & Astrup, 2007). Future studies need to examine gender differences in brain responses to food stimuli and whether these responses vary over the menstrual cycle in women. Additionally, because we used a quartile split on the RS to divide the population into unrestrained and restrained eaters, we cannot assume that the results will generalize to more moderate scores on the RS. In the same vein, it cannot be assumed that the activation patterns shown by the unrestrained eaters reflect appropriate or normal responding to the experimental stimuli because our unrestrained eaters were even less restrained than unrestrained eaters included in past studies.

An additional limitation is that we used Ensure as a preload, whereas laboratory studies on counterregulatory eating typically used an ice cream milkshake. Although participants were compared in states of hunger and fullness, drinking Ensure may have been psychologically different from drinking a milkshake, therefore making the results less comparable to the laboratory preload studies. Participants were not asked to rate how much they liked the Ensure; however, all participants consumed the full two cans. Another deviation from procedures typically used in preload studies was the 45-min wait between consumption of Ensure and the second scanning. As a result of this, participants may not have been as full as they have been in classic preload studies; however, research has shown that even small preloads of highly palatable foods have disinhibiting effects on restrained eaters (Knight &

Boland, 1989). A further limitation is that we used pictures of food rather than actual food intake following the preload. It is not known whether similar results would have been found if real foods had been used instead of pictures. Lastly, we did not examine whether brain areas that were activated were excitatory or inhibitory (i.e., activated vs. deactivated). Many of the areas in the prefrontal, orbitofrontal, and limbic/paralimbic areas are implicated in both hunger and in response to satiation. Therefore, it would be important to identify whether these areas were activated versus deactivated in response to the manipulations examined here. It is possible to examine activation versus deactivation using fMRI; however, we did not use this technique in the present study because the statistical basis for the comparison is controversial.

In conclusion, we used the relatively new technology of fMRI to address the nature of restrained eaters' vulnerability to counterregulatory eating, an eating pattern that has not been satisfactorily explained after more than three decades and hundreds of studies on dietary restraint (Lowe & Kral, 2006; Lowe & Thomas, 2009). The functions mediated by brain areas showing distinctive activation in restrained and unrestrained eaters in pertinent conditions corresponded well with the eating patterns of these groups in eating regulation studies. Furthermore, the tendency of fed restrained eaters to become activated in areas associated with reward when viewing highly palatable food is consistent with the hypothesis that restrained eaters are restricting their intake not to lose weight (Stice et al., 2007) but to avoid eating more than they need and thereby gaining weight (Lowe & Butryn, 2007). However, because this is the first fMRI study comparing normal weight restrained and unrestrained eaters, and because there are alternative interpretations of the present findings, more neuroimaging research is needed to better understand the intriguing but potentially problematic eating behavior of restrained eaters.

## References

Allison, D. B., Kalinsky, L. B., & Gorman, B. S. (1992). A comparison of the psychometric properties of three measures of dietary restraint. *Psychological Assessment, 4,* 391–398.

Alonso-Alonson, M., & Pascual-Leone, A. (2007). The right brain hypothesis for obesity. *Journal of American Medical Association, 297,* 1819–1822.

Aron, A. R., Behrens, T. E., Smith, S., Frank, M. J., & Poldrak, R. A. (2007). Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *Journal of Neuroscience, 27,* 3743–3752.

Ashburner, J., & Friston, K. J. (1999). Nonlinear spatial normalization using basis functions. *Human Brain Mapping, 7,* 254–266.

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala-frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience, 2,* 303–312.

Barbas, H. (1995). Anatomic basis of cognitive-emotional interactions in the primate prefrontal cortex. *Neuroscience and Biobehavioral Reviews, 19,* 499–510.

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,* 5160–5166.

Blundell, J. E., & Rogers, P. J. (1980). Effects of anorexic drugs on food intake, food selection and preferences and hunger motivation and subjective experiences. *Appetite, 1,* 151–165.

Brett, M., Penny, W. D., & Kiebel, S. J. (2003). Introduction to random

field theory. In R. S. J. Frackowiak, K. J. Friston, C. Frith, R. Dolan, K. J. Friston, C. J. Price, et al. (Eds.), *Human brain function* (2nd ed., pp. 867–879) London: Academic Press.

Buckner, R. L., & Logan, J. M. (2001). Functional neuroimaging methods: PET and fMRI. In R. Cabeza & A. Kingstone (Eds.), *Handbook for functional neuroimaging of cognition* (pp. 28–43). Cambridge, MA: MIT Press.

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience and Biobehavioral Reviews, 26,* 321–352.

Crossman, A. R., & Neary, D. (2000). *Neuroanatomy: An illustrated colour text* (2nd ed.). New York: Churchill Livingstone.

Davidsen, L., Vistisen, B., & Astrup, A. (2007). Impact of the menstrual cycle on determinants of energy balance: A putative role in weight loss attempts. *International Journal of Obesity, 31,* 1777–1785.

Davidson, R. J. (2003). Darwin and the neural bases of emotion and affective style. *Annals of the New York Academy of Sciences, 1000,* 316–336.

Del Parigi, A., Gautier, J. F., Chen, K., Salbe, A. D., Ravussin, E., Reiman, E., et al. (2002). Mapping the brain responses to hunger and satiation in humans using positron emission tomography. *Annals of the New York Academy of Sciences, 967,* 389–397.

FIL Methods Group. (2007). *SPM5 manual*. Retrieved March 21, 2007, from http://www.fil.ion.ucl.ac.uk/spm/doc/manual.pdf

Friedman, M. I., Ulrich, P., & Mattes, R. D. (1999). A figurative measure of subjective hunger sensations. *Appetite, 32,* 395–404.

Friston, K. (2003). Experimental design and statistical parametric mapping. In R. S. J. Frackowiak, K. J. Friston, C. Frith, R. Dolan, K. J. Friston, C. J. Price, S. Zeki, J. Ashburner, & W. D. Penny (Eds.), *Human brain function* (2nd ed., pp. 599–632). London: Academic Press.

Friston, K. J., Worsley, K. J., Frackowiak, R. S. J., Mazziotta, J. C., & Evans, A. C. (1994). Assessing the significance of focal activations using their spatial extent. *Human Brain Mapping, 1,* 210–220.

Gauiter, J. F., Chen, K., Salbe, A. D., Bandy, D., Prately, R. E., Heiman, M., et al. (2000). Differential brain responses to satiation in obese and lean men. *Diabetes, 49,* 838–846.

Gauiter, J. F., Chen, K., Salbe, A. D., Bandy, D., Prately, R. E., Heiman, M., et al. (2001). Effect of satiation on brain activity in obese and lean women. *Obesity Research, 9,* 676–684.

Geliebter, A., Ladell, T., Logan, M., Schneider, T., Sharafi, M., & Hirsch, J. (2006). Responsivity to food stimuli in obese and lean binge eaters using functional MRI. *Appetite, 46,* 31–35.

Gottfried, J. A., O'Doherty, J., & Dolan, R. J. (2003, August 22). Encoding predictive reward and value in human amygdala and orbitofrontal cortex. *Science, 301,* 1104–1107.

Heatherton, T. F., & Polivy, J. (1992). Chronic dieting and eating disorders: A spiral model. In J. H. Crowther, D. L. Tennenbaum, S. E. Hobfold, & M. A. Parris (Eds.), *The etiology of bulimia nervosa: The individual and familial context* (pp. 133–155). Washington, DC: Hemisphere Publication Services.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43,* 647–660.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint and eating behavior. *Journal of Abnormal Psychology, 84,* 666–672.

Herman, C. P., & Polivy, J. (1980). Restrained eating. In A. J. Stunkard (Ed.), *Obesity* (pp. 208–255). Philadelphia: Saunders.

Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. In A. J. Stunkard & E. Stellar (Eds.), *Eating and its disorders* (pp. 141–156). New York: Raven Press.

Herman, C. P., & Polivy, J. (2004). The self-regulation of eating: Theoretical and practical problems. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 492–508). New York: Guilford Press.

Herman, C. P., & Polivy, J. (2005). Normative influences on food intake. *Physiology & Behavior, 86,* 762–772.

Hill, A. J., Magson, L. D., & Blundell, J. E. (1984). Hunger and palatability: Tracking ratings of subjective experience before, during and after consumption of preferred and less preferred food. *Appetite, 5,* 361–371.

Jarry, J. L., Polivy, J., Herman, C. P., Arrowood, A. J., & Pliner, P. (2006). Restrained and unrestrained eaters' attributions of success and failure to body weight and perception of social consensus: The special case of romantic success. *Journal of Social and Clinical Psychology, 25,* 885–905.

Killgore, W., Young, A. D., Femia, L. A., Bogorodzki, P., Rogowska, J., & Yurgelun-Todd, D. A. (2003). Cortical and limbic activation during viewing of high- versus low-calorie foods. *NeuroImage, 19,* 1381–1394.

Killgore, W., & Yurgelun-Todd, D. A. (2006). Affect modulates appetite-related brain activity to images of food. *International Journal of Eating Disorders, 39,* 357–363.

Klem, M. L., Klesges, R. C., Bene, C. R., & Mellon, M. W. (1990). A psychometric study of restraint: The impact of race, gender, weight, and marital status. *Addictive Behavior, 15,* 147–152.

Klesges, R. C., Klem, M. C., & Bene, C. R. (1989). Effects of dietary restraint, obesity, and gender on holiday eating behavior and weight gain. *Journal of Abnormal Psychology, 98,* 499–503.

Knight, L. J., & Boland, F. J. (1989). Restrained eating: An experimental disentanglement of the disinhibiting variables of perceived calories and food type. *Journal of Abnormal Psychology, 98,* 412–420.

Knoch, D., & Fehr, E. (2007). Resisting the power of temptations: The right prefrontal cortex and self-control. *Annals of the New York Academy of Sciences, 1104,* 123–134.

Kringelbach, M. L. (2004). Food for thought: Hedonic experience beyond homeostasis in the human brain. *Neuroscience, 126,* 807–819.

Kringelbach, M. L., O'Doherty, J., Rolls, E. T., & Andrews, C. (2003). Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cerebral Cortex, 13,* 1064–1071.

LaBar, K. S., Gitelman, D. R., Parrish, T. B., Nobre, A. C., & Mesulam, M. M. (2001). Hunger selectively modulates corticolimbic activation to food stimuli in humans. *Behavioral Neuroscience, 115,* 493–500.

Li, C. S., Huang, R. T., Constable, R. T., & Sinha, R. (2006). Gender differences in the neural correlates of response inhibition during a stop signal task, *NeuroImage, 32,* 1918–1929.

Lowe, M. R. (1984). Dietary concern, weight fluctuation and weight status: Further explorations of the Restraint Scale. *Behaviour Research and Therapy, 22,* 243–248.

Lowe, M. R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin, 114,* 100–121.

Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., et al. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite, 47,* 83–90.

Lowe, M. R., & Butryn, M. L. (2007). Hedonic hunger: A new dimension of appetite? *Physiology & Behavior, 91,* 432–439.

Lowe, M. R., Friedman, M. I., Mattes, R., Kopyt, D., & Gayda, C. (2000). Comparison of verbal and pictorial measures of hunger during fasting in normal weight and obese humans. *Obesity Research, 8,* 566–574.

Lowe, M. R., Gleaves, D. H., & Murphy-Eberenz, K. P. (1998). On the relation of dieting and bingeing in bulimia nervosa. *Journal of Abnormal Psychology, 107,* 263–271.

Lowe, M. R., & Kral, T. V. E. (2006). Stress-induced eating in restrained eaters may not be caused by stress or restraint. *Appetite, 46,* 16–21.

Lowe, M. R., & Levine, A. S. (2005). Eating motives and the controversy over dieting: Eating less than needed versus less than wanted. *Obesity Research, 13,* 797–806.

Lowe, M. R., & Thomas, J. G. (2009). Measures of restrained eating: Conceptual evolution and psychometric update. In D. Allison & M. L.

Baskin (Eds.), *Handbook of assessment methods for obesity and eating behaviors* (pp. 137–185). New York: Sage.

Neurobehavioral Systems, Inc. (2003–2009). Presentation (Version 9.81) [Computer Software and Manual]. Retrieved April 2007, from http://www.neurobs.com

O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: Insights from neuroimaging. *Current Opinion in Neurobiology, 14,* 769–776.

Pelchat, M. L., Johnson, A., Chan, R., Valdez, J., & Ragland, D. J. (2004). Images of desire: Food-craving activation during fMRI. *NeuroImage, 23,* 1486–1493.

Platek, S. M., Loughead, J. W., Gur, R. C., Busch, S., Ruparel, K., Phend, N., et al. (2006). Neural substrates for functionally discriminating self-face from personally familiar faces. *Human Brain Mapping, 27,* 91–98.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40,* 193–201.

Polivy, J., & Herman, C. P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders, 26,* 153–164.

Presnell, K., & Stice, E. (2003). An experimental test of the effect of weight-loss dieting on bulimic pathology: Tipping the scales in a different direction. *Journal of Abnormal Psychology, 112,* 166–170.

Rolls, E. T. (2004). The functions of the orbitofrontal cortex. *Brain & Cognition, 55,* 11–29.

Rorden, C., Bonilha, L., & Nichols, T. (2007). Rank-order versus mean based statistics for neuroimaging. *NeuroImage, 35,* 1531–10537.

Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin, 99,* 247–262.

Silverstone, T. (1982). Measurement of hunger and food intake in man. In T. Silverstone (Ed.), *Drugs and appetite* (pp. 81–92). London: Academic Press.

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

Spitzer, L., & Rodin, J. (1981). Human eating behavior: A critical review of studies in normal weight and overweight individuals. *Appetite, 2,* 293–329.

Stice, E., Cooper, J. A., Schoeller, D. A., Tappe, K., & Lowe, M. R. (2007). Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioral data suggest not. *Psychological Assessment, 14,* 449–458.

Stice, E., Fisher, M., & Lowe, M. R. (2004). Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. *Psychological Assessment, 16,* 51–59.

Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain.* Stuttgart, NY: Thieme.

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

Tataranni, P. A., Gautier, J. F., Chen, K., Uecker, A., Bandy, D., Salbe, A. D., et al. (1999). Neuroanatomical correlates of hunger and satiation in humans using positron emission tomography. *Proceedings of the National Academy of Sciences, USA, 96,* 4569–4574.

Trottier, K., Polivy, J., & Herman, C. P. (2005). Effects of exposure to unrealistic promises about dieting: Are unrealistic expectations about dieting inspirational? *International Journal of Eating Disorders, 37,* 142–149.

Wang, G. J., Volkow, N. D., Telang, F., Jayne, M., Ma, J., Rao, M., et al. (2004). Exposure to appetitive food stimuli markedly activates the human brain. *NeuroImage, 21,* 1790–1797.

Wang, G. J., Yang, J., Volkow, N. D., Telang, F., Ma, Y., Zhu, W., et al. (2006). Gastric stimulation in obese subjects activates the hippocampus and other regions involved in brain reward circuitry. *Proceedings of the National Academy of Sciences, USA, 103,* 15641–15645.

Watanabe, M. (1996, August 15). Reward expectancy in primate prefrontal neurons. *Nature, 382,* 629–632.

Watanabe, M., Hikosaka, K., Sakagami, M., & Shirakawa, S. (2002). Coding and monitoring of motivational context in the primate prefrontal cortex. *Journal of Neuroscience, 22,* 2391–2400.

Wellcome Department of Cognitive Neurology. (2005). Statistical parametric mapping [Computer software]. Retrieved May 30, 2003, from http://www.fil.ion.ucl.ac.uk/spm/