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Inducing Negative Affect Increases the Reward Value of Appetizing Foods in Dieters

Dylan D. Wagner*, **Rebecca G. Boswell**, **William M. Kelley**, and **Todd F. Heatherton**
Department of Psychological and Brain Sciences, Dartmouth College, Hanover, NH, USA

Abstract

Experiencing negative affect frequently precedes lapses in self-control for dieters, smokers, and drug addicts. Laboratory research has similarly shown that inducing negative emotional distress increases the consumption of food or drugs. One hypothesis for this finding is that emotional distress sensitizes the brain's reward system to appetitive stimuli. Using functional neuroimaging, we demonstrate that inducing negative affect in chronic dieters increases activity in brain regions representing the reward value of appetitive stimuli when viewing appetizing food cues. Thirty female chronic dieters were randomly assigned to receive either a negative ($n = 15$) or neutral mood induction ($n = 15$) immediately followed by exposure to images of appetizing foods and natural scenes during functional magnetic resonance imaging (fMRI). Compared to chronic dieters in a neutral mood, those receiving a negative mood induction showed increased activity in the orbitofrontal cortex to appetizing food images. In addition, activity to food images in the orbitofrontal cortex and ventral striatum was correlated with individual differences in the degree to which the negative mood induction decreased participants' self-esteem. These findings suggest that distress sensitizes the brain's reward system to appetitive cues thereby offering a mechanism for the oft-observed relationship between negative affect and disinhibited eating.

Keywords

Reward; Food; Obesity; Dieting; Mood; Emotion; Cue-Reactivity; Orbitofrontal Cortex

Obesity rates continue to rise in the United States despite the fact that many people undertake dieting to control their body weight. One of the primary reasons for this may be the simple fact that most diets fail, particularly over the long-term (Jeffery et al., 2000), leading many to become lifelong chronic dieters. Chronic dieters are constantly attempting to regulate their weight but are beset by lapses in self-control (Heatherton, Polivy, & Herman, 1991; Herman & Mack, 1975). Paradoxically, chronic dieters are more likely to engage in binge eating (Field et al., 2003), are at greater risk for obesity (Stice, Cameron, Killen, Hayward, & C. Barr Taylor, 1999), and are more susceptible to eating following exposure to appetizing food cues (Federoff, Polivy, & Herman, 1997; Harris, Bargh, & Brownell, 2009; Papies & Hamstra, 2010). Gaining control over the obesity epidemic requires a better understanding of the factors that sabotage dieters' efforts at weight control. The accumulated evidence indicates that negative affect is a primary determinant of self-regulatory failures across a range of addictive and maladaptive behaviors (e.g., Baumeister, 1997; Heatherton, 2011). With respect to dieting, extensive laboratory research has demonstrated that inducing negative mood leads dieters to eat greater quantities of food (Frost, Goolkasian, Ely, & Blanchard, 1982; Heatherton, Herman, & Polivy, 1991; 1992;

*Corresponding author: Dylan D. Wagner, Department of Psychological and Brain Sciences, 6207 Moore Hall, Dartmouth College, Hanover, NH, 03755, Telephone: (603) 646-9377, Fax: (603) 646-1419, dylan.d.wagner@dartmouth.edu.

Heatherton, Striye, & Wittenberg, 1998; Schotte, Cools, & McNally, 1990). What is it about emotional distress that sabotages self-control?

Theories based primarily on human research posit that the reason emotional distress causes self-regulatory failure is because it reduces self-awareness and constricts attention to the immediate environment (Heatherton & Baumeister, 1991). This escape from awareness may lead dieters to focus on immediate hedonic goals (e.g. eating appetizing foods) instead of long-term regulatory ones. Neuroscientific research conducted primarily in non-human animals, suggests that distress leads to a simultaneous dampening of prefrontal control over behavior which is accompanied by a concomitant sensitization of the threat (Arnsten, 2009, Whalen, 1998) and reward systems (Piazza & Le Moal, 1996) to biologically salient stimuli. Specifically, inducing emotional distress (e.g., social-defeat stress) in non-human animals leads to release of glucocorticoids which in turn sensitize the brain's reward system to food and drugs (Adam & Epel, 2007; Covington & Miczek, 2005; Deroche et al., 1995), as well as to their predictive cues (Peciña, Schulkin, & Berridge, 2006), thereby increasing reactivity to appetitive stimuli. Findings such as these suggest that effective self-regulation relies on the capacity of self-control to keep impulses in check. Distress, then, serves to disrupt this balance, leading to reduced top-down control from the prefrontal cortex in conjunction with amplified response to the hedonic properties of salient stimuli both of which conspire to induce self-regulatory collapse (for a review see Heatherton & Wagner, 2011).

A large body of electrophysiological and neuroimaging research has shown that the reward value of food is represented in the orbitofrontal cortex and striatum. In animal studies, the orbitofrontal cortex increases its firing rate during the presentation of visual or olfactory food cues (for a review see Rolls, 2000), which in turn, is modulated by the animal's relative satiety (Critchley & Rolls, 1996). In humans, functional neuroimaging research has demonstrated that activity in the orbitofrontal cortex is increased when tasting, smelling or viewing food images (Beaver et al., 2006; Demos, Kelley, & Heatherton, 2011; Gottfried, O'Doherty, & Dolan, 2003; Killgore & Todd, 2006; Kringelbach, O'Doherty, Rolls, & Andrews, 2003; Morris & Dolan, 2001; O'Doherty, Rolls, Francis, Bowtell, & McGlone, 2001; O'Doherty et al., 2000; Porubská, Veit, Preissl, Fritsche, & Birbaumer, 2006; Rothenmund et al., 2007; Schienle, Schäfer, Hermann, & Vaitl, 2009; Simmons, Martin, & Barsalou, 2005; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001; Stoeckel et al., 2008; Wang et al., 2004, 2009; Yokum, Ng, & Stice, 2011). Moreover, when viewing food images, activity in the OFC is correlated with individual differences in hunger (Morris & Dolan, 2001) and reward sensitivity (Beaver et al., 2006) and also predicts future weight gain (Yokum et al., 2011).

If emotional distress leads to sensitization of the human brain's reward system to food and drugs, then it follows that brain regions involved in representing the reward value of appetitive stimuli, such as the orbitofrontal cortex and striatum (O'Doherty, 2004), would show an exaggerated response to food cues following emotional distress. To test this hypothesis, we used functional neuroimaging to measure food-cue related activity in thirty chronic dieters. We used a validated mood manipulation procedure based on interpersonal distress to induce negative mood in half the chronic dieting sample and neutral mood in the other half. Immediately after, participants in both conditions were exposed to appetizing food images. Prior to and following the experiment, participants were asked to complete a self-esteem measure to index the degree to which they were affected by the experimental manipulations. We predicted that, compared to chronic dieters in a neutral mood, those who experienced negative emotional distress would subsequently show an exaggerated neural response in brain regions involved in representing the reward value of appetizing food cues.

Materials and Methods

Participants

Thirty healthy right-handed female dieters (mean age = 19.7; range: 18–22 years old) with normal or corrected-to-normal visual acuity and no history of neurological problems participated in this study. Chronic dieters were recruited from a larger sample of college students who completed the Restraint Scale, a validated measure of chronic dieting tendencies (Heatherton, Herman, Polivy, King, & McGree, 1988; Herman & Mack, 1975) as part of a mass testing session. Participants with restraint scores greater than 16 were considered chronic dieters (see Heatherton et al., 1991) and invited to participate in the current study (mean restraint: 18.8; range: 16–26). All participants remained unaware of these selection criteria until debriefing. Participant selection was limited to young women as young women show a higher incidence of dieting (e.g. Herman & Mack, 1975), are at increased risk for weight gain (Holm-Denoma, Joiner, Vohs, & Heatherton, 2008) and because of known gender differences in reasons for dieting (e.g., Pingitore, Spring, & Garfield, 1997). Prior to scanning, participants were randomly assigned to receive either a negative mood induction (N = 15) or a neutral mood induction (N = 15). Negative and neutral mood induction groups did not differ in terms of weight, estimated body mass index (BMI), self-esteem or scores on the Restraint Scale (all $p > 0.34$; Table 1). All participants gave informed consent in accordance with the guidelines set by the Committee for the Protection of Human Subjects at Dartmouth College.

Mood Induction Procedures

The negative and neutral mood induction procedures used in the present study were adapted from the widely used Velten (1968) mood induction procedure (VMIP) in which participants read a series of sixty increasingly negative self-referential statements designed to induce feelings of inadequacy and dysphoria (e.g. *“I’ve doubted that I’m a worthwhile person”*). Female dieters frequently report greater body dissatisfaction, lower self-esteem, and greater social anxiety than their non-dieting peers (Heatherton, 1993; Johnson & Wardle, 2005). Accordingly, for the negative mood induction we implemented a social distress version of the original VMIP using statements designed to induce increasing feelings of social rejection and isolation (e.g. *“I’m convinced that no one likes me”*).

A pilot study was conducted to ensure that the social distress VMIP had measurable effects on mood and self-esteem. Twenty-nine participants performed the neutral VMIP (Velten, 1968), followed by the State Self-Esteem Scale (Heatherton & Polivy, 1991) and a 24-item mood scale (Heatherton & Vohs, 2000). Participants then completed the social distress VMIP and once again state self-esteem and mood were assessed. Results of this pilot indicated that the social distress VMIP led to reduced positive mood, $t(28) = 3.27$, $p = 0.003$, increased negative mood, $t(28) = 2.24$, $p = 0.03$, and a reduction in social state self-esteem, $t(28) = 2.46$, $p = 0.02$.

Food Cue-Reactivity Task

The food cue-reactivity task was adapted from prior research on neural cue-reactivity to appetizing foods (e.g. Demos, Kelley, & Heatherton, 2011; Demos, Heatherton & Kelley, in press) and consisted of one hundred and fifty images from one of three categories: 50 images of appetizing, high-calorie foods (e.g. desserts, fast food, snacks), 50 images involving people and 50 natural scenes, such as landscapes. Participants were asked to indicate via a button-press whether an image took place indoors or outdoors. This *incidental* task was chosen so as to disguise the purpose of the study and also to ensure that participants were attending to the images. All images conformed to an image dimension of 480×360 pixels at 72dpi.

Task and experimental design

Prior to scanning, participants were randomly assigned to receive either the negative ($N = 15$) or neutral ($N = 15$) mood induction procedures. Participants were instructed that they would be completing two tasks: a reading task (i.e. the mood induction task) and a simple categorization task (i.e. the cue-reactivity task). At no time was it mentioned that the tasks were designed to manipulate mood or measure responses to appetizing foods. For the mood induction procedure, participants were instructed to read each statement and to recall moments in their lives where they've experienced the feelings described. For the image categorization task, participants were instructed to categorize, via button press, each image according to whether it took place indoors or outdoors. Following scanning, participants completed a self-esteem assessment (Fleming & Courtney, 1984) and were debriefed regarding the purposes of the experiment and the nature of the mood induction. If they had received the negative mood induction, they were offered a list of statements to read from the positive version of the VMIP in order to return their mood to baseline.

The cue-reactivity task used a rapid event-related design with trials consisting of a single image (food, people or nature images) displayed for 2000ms. The order of trial types and the duration of the interstimulus interval (between 500ms and 8000ms, mean duration 3200ms) were pseudorandomized. During the interstimulus interval, null event trials consisting of a white fixation cross against a black background were shown for 2500ms and were used to introduce jitter into the fMRI time series in order to increase the estimation efficiency of task effects.

Image Acquisition

Magnetic resonance imaging was conducted with a Philips Achieva 3.0 Tesla scanner using an eight-channel phased array coil. Structural images were acquired using a T1-weighted MP-RAGE protocol (160 sagittal slices; TR: 9.9ms; TE: 4.6 ms; flip angle: 8°; $1 \times 1 \times 1$ mm voxels). Functional images were acquired using a T2*-weighted echo-planar sequence (TR: 2500ms; TE: 35ms; flip angle: 90°; field of view: 24cm). For each participant, a single functional run of 520 whole-brain volumes (36 axial slices per whole-brain volume, 3.5mm thickness, 0.5mm gap; 3×3 mm in-plane resolution) was collected.

Image Preprocessing and Analysis

fMRI data were analyzed using the general linear model for event-related designs in SPM8 (Wellcome Department of Cognitive Neurology, London, UK). For each functional run, data were preprocessed to remove sources of noise and artifact. Images were corrected for differences in acquisition time between slices and realigned within and across runs via a rigid body transformation in order to correct for head movement. Images were then unwarped to reduce residual movement-related image distortions not corrected by realignment. Functional data were normalized into a standard stereotaxic space (3mm isotropic voxels) based on the SPM8 EPI template that conforms to the ICBM 152 brain template space (Montreal Neurological Institute) and approximates the Talairach and Tournoux atlas space. Finally, normalized images were spatially smoothed (6mm full-width-at-half-maximum) using a Gaussian kernel.

For each participant, a general linear model (GLM) was constructed to investigate food-cue specific brain activity. This GLM, incorporating task effects and covariates of no interest (a box-car regressor modeling the period of VMIP, a linear trend to account for low-frequency drift and 6 movement parameters derived from realignment corrections) was convolved with a canonical hemodynamic response function and used to compute parameter estimates (β) and contrast images (containing weighted parameter estimates) for each visual scene category at each voxel. Contrast images for each subject, comparing food vs. natural scenes

were entered into a second-level, random-effects analysis with participant treated as the random effect. Monte Carlo simulations using AFNI's AlphaSim were used to calculate the minimum cluster size at an uncorrected threshold of $p < 0.001$ required for a whole brain correction of $p < 0.05$. Simulations (10,000 iterations) were performed on the volume of our study-wide whole-brain mask using smoothness estimated from the residuals obtained from the GLM and resulting in a minimum cluster size of 56 contiguous voxels. Between-group comparisons were conducted in *a priori* regions of interest (i.e. the OFC, defined functionally and the nucleus accumbens, defined anatomically). The OFC region was defined from the statistical comparisons of food vs. scenes across both groups using a spherical ROI (6mm) centered on the peak voxel of clusters demonstrating an effect of food vs. natural scenes. As both groups contributed equally to the ROI-defining statistical map, this ROI is statistically unbiased with regards to between group effects. Finally, *a priori* anatomical ROIs of the left and right nucleus accumbens were defined using labels provided with the Harvard-Oxford probabilistic atlas of cortical and subcortical structures (Desikan et al., 2006). Additional exploratory between-group comparisons were conducted in regions derived from the main effect for food vs. natural scenes and were corrected for multiple comparisons across all 10 ROIs.

Results

Behavioral Results

Following the mood induction procedure, participants in the negative group showed a reduction in self-esteem compared to their previously assessed baseline, $t(14) = 2.73$, $p = 0.016$ (Mean Pre: 117; Mean Post: 102.3), whereas participants in the neutral group did not $t(14) = 0.13$, $p = 0.9$ (Mean Pre: 119.4; Mean Post: 120.3). There was also a trend towards reduced self-esteem in the negative compared to the neutral mood induction groups, $t(28) = 1.98$, $p = 0.058$.

fMRI Results

When contrasting the BOLD response to appetizing food images to neutral natural scenes across both groups of dieters, a number of brain regions were found to exhibit food-cue related activity (Table 2; Figure 2a). These regions were subsequently interrogated for an effect of mood manipulation condition. Regions of interest analysis showed that participants who underwent the negative mood induction exhibited greater food-cue related activity in the left orbitofrontal cortex (MNI coordinates: $-27,36, -12$) than did participants in the neutral mood induction procedure, $t(28) = 2.20$, $p = 0.036$ (Figure 2b). No other region differentiated between negative and neutral mood induction groups, both at a corrected threshold for multiple comparisons across all ROIs, but also uncorrected (all $p > 0.17$). Analysis of the nucleus accumbens, using *a priori* anatomical ROIs, revealed no between group differences in food-cue related activity (left NAc. $t(28) = 1.17$, $p = 0.25$; right NAc. $t(28) = 0.92$, $p = 0.37$).

Correlation with post-scan change in self-esteem

Next, we examined the relationship between food-cue related activity and individual differences in the degree to which participants were affected by the negative mood induction (using changes in self-esteem scores as a proxy) and correlated these with the BOLD response from the orbitofrontal cortex and anatomically derived ROIs in the nucleus accumbens. This analysis revealed a significant relationship between reductions in self-esteem and food-cue related activity in the orbitofrontal cortex ($r = 0.52$, $p = 0.047$) and bilaterally in the nucleus accumbens (left: $r = 0.54$, $p = 0.040$; right: $r = 0.53$, $p = 0.042$), indicating that participants who exhibited a greater decrease in self-esteem following the

negative mood induction subsequently demonstrated a greater increase in food-cue related activity in the OFC and nucleus accumbens (Figure 3).

Discussion

Negative affect has long been associated with lapses in self-control (Heatherton, 2011; Heatherton & Wagner, 2011; Schachter, Goldman, & Gordon, 1968). The present study investigated the hypothesis, derived from animal models of drug addiction, that emotional distress sensitizes the brain's reward system to appetitive stimuli (e.g. Piazza & Le Moal, 1998, 1996; Sinha, 2001) and their cues (e.g. Peciña et al., 2006). Compared to non-distressed dieters, those who underwent a negative mood induction showed greater food cue-related activity in the orbitofrontal cortex. Moreover, additional analyses showed that food-cue related activity in the OFC and ventral striatum was correlated with individual differences in the amount of distress produced by the negative mood induction.

In both animals and humans, the orbitofrontal cortex and ventral striatum are involved in representing the reward value and motivational salience of appetitive stimuli such as food (Demos et al., 2011; Gottfried, O'Doherty, & Dolan, 2003; Killgore & Yurgelun-Todd, 2006; Morris & Dolan, 2001; O'Doherty, Rolls, Francis, Bowtell, & McGlone, 2001; Rolls, 2000; Simmons, Martin, & Barsalou, 2005; Wang et al., 2004), but also drugs of abuse (Brody et al., 2002; David et al., 2005; Wagner, Dal Cin, Sargent, Kelley, & Heatherton, 2011; Wilson, Sayette, & Fiez, 2004). Moreover, previous research has shown that activity in the OFC indexes satiety and the subjective pleasantness of food during consumption (Kringelbach, O'Doherty, Rolls, & Andrews, 2003; O'Doherty et al., 2000; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). For example, when consuming appetizing foods, activity in the OFC decreases as satiety increases, suggesting that the OFC represents moment-by-moment changes in the subjective reward value of foods (O'Doherty et al., 2000). Finally, the results reported here are consistent with a recent meta-analysis of food cue exposure research (van der Laan, de Ridder, Viergever, & Smeets, 2011) showing that the left OFC is the most commonly recruited region when in comparisons of food and nonfood items (e.g. tools, scenery).

Numerous laboratory studies have shown that when dieters experience negative emotional distress they subsequently eat more than non-dieters or dieters in a neutral mood (e.g. Frost et al., 1982; Heatherton et al., 1991; 1992; Heatherton, Polivy, Herman, & Baumeister, 1993; Heatherton et al., 1998). Particularly relevant is a study by Frost and colleagues (1982) in which dieters who underwent the negative VMIP subsequently ate more food than both depressed nondieters and a control group of dieters in a neutral mood (Frost et al., 1982). Using the same type of mood induction procedure, we found that when dieters experience negative affect, neural activity in regions associated with the hedonic value of appetizing food show increased activity relative to dieters in a neutral mood. Additional research shows that negative mood increases women's attentional bias towards appetizing food cues as well as their subjective hunger (Hepworth, Mogg, Brignell & Bradley, 2010). These behavioral results, in conjunction with our neuroimaging findings, suggest that negative affect sensitizes the reward system to the hedonic value of appetizing foods thereby precipitating self-regulatory failure and, in turn, overeating. One limitation of the present study is that we recruited only dieters, however, given the behavioral research outlined above it is likely that non-dieters are unlikely to show an effect of mood on neural responses to food cues. Future research should seek to confirm this conjecture.

The propensity for negative moods to cause a breakdown in self-control is true not only of dieting but also of many other regulated behaviors such as drug addiction (e.g. Heatherton, 2011; Marlatt & Gordon, 1985; Sinha, 2009), alcoholism (Witkiewitz & Villarroel, 2009)

and smoking (Kassel, Stroud, & Paronis, 2003; Magid, Colder, Stroud, Mimi Nichter, & Nichter, 2009). For example, inducing negative mood in smokers increases both cigarette craving (Perkins et al., 2008; Tiffany & Drobes, 1990; Willner & Jones, 1996) and smoking intensity (McKee et al., 2011). Similar effects of negative mood induction on craving have been found in alcoholics (Cooney, Litt, Morse, Bauer, & Gaupp, 1997; Fox, Bergquist, Hong, & Sinha, 2007; Sinha et al., 2009) and drug addicts (Childress et al., 1994). More broadly, research has shown that experiencing emotionally negative life events is a potent trigger for drug relapse (Sinha, 2007; Witkiewitz & Villarroel, 2009). Even outside of the domain of drug addiction, experiencing negative mood is associated with failures of self-control such as excessive television viewing (Dittmar, 1994) or internet usage (LaRose, Lin, & Eastin, 2003). Hence, negative affect may act, in a general way, to enhance reward signals associated with desired behaviors, thereby making it harder to inhibit engaging in those behaviors (Heatherton & Wagner, 2011).

Limitations

A limitation of the present study is the lack of subjective ratings on participants' desire for the appetizing food cues. Although the use of an incidental task allowed us to mitigate experimenter demand we cannot, however, confirm that the negative mood induction increased participants' food cravings or likeability ratings for the food items. However, as reviewed above, a large body of behavioral research strongly suggests that experimental inductions of negative affect increase food cravings and consumption in dieters (e.g. Frost, Goolkasian, Ely, & Blanchard, 1982; Heatherton et al., 1991; 1992; Heatherton, Striipe, & Wittenberg, 1998; Hepworth et al. 2010). A related issue is whether the between group difference in food cue-related activity found in left OFC, and the correlation with decrements in self-esteem found in left OFC and the nucleus accumbens necessarily indicate increased reward-related processing. A large number of prior imaging studies have related both of these regions to subjective reports of food craving (Morris & Dolan, 2001), desirability (Goldstone et al., 2009), pleasantness (Kringelbach, O'Doherty, Rolls, & Andrews, 2003; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001), reward sensitivity (Beaver et al. 2006; Schienle et al. 2009), and satiety (O'Doherty et al., 2000). The findings of these studies along with the previously reviewed behavioral evidence in which negative affect increases craving and overeating, argues that differences we see in dieters who experienced negative affect reflects changes in the reward value of appetizing foods. Furthermore, although our task defined food-cue related activity as the difference between highly appetizing foods and non-food scenes, other research shows that in cases where high vs. low-calorie food cues are compared, activity in both the OFC and nucleus accumbens is largest for high calorie foods (i.e., Goldstone et al. 2009). Moreover, recent research has shown that food-cue related activity in the nucleus accumbens, assessed using the same task and stimuli as used in the present study, is predictive of future weight gain assessed six months later (e.g., Demos, Heatherton, & Kelley, 2012), strongly suggesting that activity in this region when viewing appetizing food cues reflects differential reward sensitivity.

Conclusions

Although the causes of self-regulatory failure are many (see Heatherton & Wagner, 2011), a frequent catalyst is negative affect (Heatherton, 2011). In humans, inducing negative emotional distress has been shown to increase eating in dieters (Heatherton et al., 1998), smoking in smokers (Tiffany & Drobes, 1990) and drug use in drug addicts (Childress et al., 1994). Research in non-human animals suggests a possible mechanism whereby distress impairs the self-regulatory functions of the prefrontal cortex (Ansten, 2009) while simultaneously sensitizing the reward system to food and drugs (Adam & Epel, 2007; Piazza & Le Moal, 1996). The current findings are consistent with this research, demonstrating that, in humans, inducing negative mood increases the neural response to appetizing food cues in

the orbitofrontal cortex and ventral striatum - both key regions involved in representing the reward value of food (O'Doherty, 2004). This interpretation is supported both by research in animals (e.g. Miczek & Mutschler, 1996; Peciña et al., 2006; Ramsey & Van Ree, 1993), but also by human research showing that stress induced increases in salivary cortisol in dieters is directly related to subsequent overeating (Epel, Lapidus, McEwen, & Brownell, 2001). Moreover, the artificial administration of glucocorticoids in humans increases eating without any concomitant increase in physical exertion (Tataranni et al., 1996).

As mentioned above, prior work shows that the reward system's response to appetizing food cues predicts weight gain six months later (Demos, Heatherton & Kelley, in press) which suggests that the response profile of these regions to appetitive stimuli can be used to index real-world sensitivity to food. Thus, we propose that the oft observed relationship between negative affect and dieters' self-regulatory failures may be due to difficulty in inhibiting overwhelming food cravings driven by a reward system rendered hyper-responsive to food cues and that this in turn puts dieters at risk of breaking their diets and binge eating.

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Figure 1.

Schematic of the study design. Restrained eaters were randomly assigned to receive either a negative or neutral mood induction (see methods). Following this, participants performed a food-cue reactivity task while undergoing functional magnetic resonance imaging. The task involved making indoor or outdoor judgments on images containing high caloric appetizing foods or natural scenes.

Figure 2.

(A) The left orbitofrontal cortex (MNI coordinates: $-27, 36, -12$) demonstrated greater activity to appetizing food images compared to neutral scenes across both groups ($p < 0.05$, corrected). (B) Within this region, dieters in the negative mood condition exhibited a larger response to appetizing food cues. Error bars indicate SEM. Coordinates are in Montreal Neurological Institute (MNI) stereotaxic space.

Figure 3.

ROI analysis of the left orbitofrontal cortex (A) and the left and right nucleus accumbens (B & C) demonstrated that individual differences in the degree to which the negative mood induction affected dieters self-esteem was correlated with food-cue related activity. Dashed lines indicate 95% confidence intervals. The orbitofrontal cortex was defined functionally in an unbiased manner from the contrast of food vs. other scenes across both groups. The left and right nucleus accumbens were defined anatomically based on the Harvard-Oxford probabilistic cortical and subcortical atlas (Desikan et al. 2006).

Table 1

Characteristics of dieters in the negative and neutral mood induction groups

	Negative Mood Induction (<i>n</i> = 15)	Neutral Mood Induction (<i>n</i> = 15)
Age (years)	19.1	19.3
Weight (<i>lbs.</i>)	127.3	131.3
BMI (<i>kg/m²</i>)	20.9	21.9
Self-esteem	117	119.4
Restraint Score	19	18.7

Note: There were no between group differences in age, BMI, Self-Esteem or Restraint scores (all $p > 0.34$).

Table 2

Brain regions demonstrating greater food cue-related activity (food vs. nature scenes) across both negative and neutral mood induction groups

Brain Region	Side	BA	t-value	Coordinates of peak activation		
				x	y	z
Orbitofrontal cortex	L	11	5.50	-27	36	-12
Dorsolateral Prefrontal Cortex	L	46	5.94	-54	36	18
Dorsolateral Prefrontal Cortex	R	46	5.57	48	39	12
Fusiform Gyrus	L	18	5.78	-54	-45	-21
Fusiform Gyrus	R	37	4.80	51	-57	-15
Superior Parietal Lobule	R	7	4.05	30	-72	33
Inferior Occipital Gyrus	L	19	4.34	-39	-72	-9
Middle Occipital Gyrus	R	18	5.43	42	-90	-3
Inferior Occipital Gyrus	L	19	4.34	-39	-72	-9
Calcarine Sulcus	L	18	6.47	-12	-99	-12

Note: Regions showing greater activity to food vs. nature scenes across both negative and neutral mood groups ($p < 0.05$, corrected) are listed along with the best estimate of their location. Coordinates are in Montreal Neurological Institute (MNI) stereotaxic space. BA = approximate Brodmann's area.