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Caloric deprivation increases responsivity of attention and reward brain regions to intake, anticipated intake, and images of palatable foods

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ABSTRACT

Dietary restraint theoretically increases risk for binge eating, but prospective and experimental studies have produced contradictory findings, apparently because dietary restraint scales do not identify individuals who are reducing caloric intake. Yet, experimentally manipulated caloric deprivation increases responsivity of brain regions implicated in attention and reward to food images, which may contribute to binge eating. We tested whether self-imposed acute and longer-term caloric restriction increases responsivity of attention and reward regions to images, anticipated receipt, and receipt of palatable food using functional magnetic resonance imaging among female and male adolescents (Study 1 n = 34; Study 2 n = 51/81). Duration of acute caloric deprivation correlated positively with activation in regions implicated in attention, reward, and motivation in response to images, anticipated receipt, and receipt of palatable food (e.g., anterior cingulate cortex, orbitofrontal cortex, putamen, and precentral gyrus respectively). Youth in a longer-term negative energy balance likewise showed greater activation in attention (anterior cingulate cortex, ventral medial prefrontal cortex), visual processing (superior visual cortex), reward (caudate) and memory (hippocampus) regions in response to receipt and anticipated receipt of palatable food relative to those in neutral or positive energy balance. Results confirm that self-imposed caloric deprivation increases responsivity of attention, reward, and motivation regions to food, which may explain why caloric deprivation weight loss diets typically do not produce lasting weight loss.

Introduction

Theorists posit that dietary restraint increases risk for onset and maintenance of binge eating and bulimia nervosa (Fairburn, 1997; Polivy and Herman, 1985). Dieting refers to intentional and sustained restriction of caloric intake for the purposes of weight loss or maintenance. Dietary restriction must result in a negative energy balance for weight loss or a balance between intake and output for weight maintenance. Polivy and Herman (1985) argue that dieters' chronic hunger increases the risk for binge eating and that a reliance on cognitive control over eating leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. It is vital to investigate the potential adverse effects of dieting because approximately 50% of young adults report engaging in dieting behaviors (Field et al., 2009).

Consistent with dietary restraint theory, females with high versus low scores on dietary restraint scales are at greater risk for future onset of binge eating, bulimic symptoms, and bulimia nervosa (Killen et al., 1996; Neumark-Sztainer et al., 2006; Stice et al., 2008a) and increases in bulimic symptoms (Johnson and Wardle, 2005; Stice, 2001; Wertheim et al., 2001). However, randomized trials find that assignment to energy-deficit weight loss interventions versus waitlist control conditions results in significant decreases in binge eating for healthy young adults (Groesz and Stice, 2007; Presnell and Stice, 2003), overweight adults (Goodrick et al., 1998; Reeves et al., 2001), and young women with bulimia nervosa (Burton and Stice, 2006). It appears that these inconsistent findings emerged because the prospective studies used dietary restraint scales that do not identify individuals who are reducing caloric intake, whereas the experiments confirmed that participants entered an energy-deficit diet that produced weight loss. Individuals with high versus low scores on various dietary restraint scales do not consume fewer calories according to objective measures of intake during single eating episodes (Hetherington et al., 2000; Ouwens et al., 2003; Stice et al., 2004; Sysko et al., 2007), multiple eating episodes (Jansen et al., 2003; Martin et al., 2005; Rolls et al., 1997; Sysko et al., 2005), and over 2–12 week observation periods (Bathalon et al., 2000; Stice et al., 2007, 2010).

Although these findings raise questions about the veracity of the dietary restraint theory, some individuals may engage in true caloric restriction, which does increase risk for binge eating (Stice et al., 2010). Several findings appear consistent with this possibility. First, rats randomized to extreme caloric deprivation conditions (in which they lost 7%–20% of their body mass) eat significantly more calories

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during ad lib feeding and show a preference for high-fat foods immediately after the deprivation period relative to non-deprived rats (Hagan et al., 2003; Lucas and Sclafani, 1992; Ogawa et al., 2005). Second, humans randomized to periods of caloric deprivation work longer on operant tasks to earn high-calorie snack foods and eat more snack foods than non-deprived controls (Cameron et al., 2008; Epstein et al., 2003; Raynor and Epstein, 2003). Third, functional magnetic resonance imaging (fMRI) experiments indicate that activation in regions that have been implicated in attention (anterior cingulate cortex), reward valuation (amygdala), memory (hippocampus), and homeostatic feeding (hypothalamus) was significantly greater in response to pictures of palatable foods versus non-food control images after caloric deprivation versus a no deprivation condition (Fuhrer et al., 2008; LaBar et al., 2001; Leidy et al., 2011), though null effects also emerged (Siep et al., 2009; Uher et al., 2006), potentially because of the small samples used in these studies (ns = 10 to 20). One experiment found a greater response in attention (posterior cingulate cortex) and reward (amygdala, orbitofrontal cortex [OFC], insula, and striatum) regions to pictures of high-calorie versus low-calorie foods after a period of caloric deprivation versus a non-deprivation condition (Goldstone et al., 2009), suggesting that caloric deprivation increases the reward value of high-calorie foods more than low-calorie foods. Another experiment found that there was greater response in the primary gustatory cortex (anterior insula, frontal operculum) and regions associated with reward (dorsolateral prefrontal cortex, medial prefrontal cortex) in response to intake of food (chocolate milk, chicken soup) after caloric deprivation versus a non-deprivation condition (Uher et al., 2006).

Collectively, data suggest that true caloric restriction may increase the reward value of food, such that the longer the caloric deprivation, the greater the activation of attention and reward regions in response to food, which putatively increases the likelihood of food intake. The fMRI experiments in which participants were randomly assigned to a period of caloric deprivation or a non-deprivation condition have strong internal validity, but limited external validity because participants did not voluntarily elect to engage in caloric restriction, as occurs in real-world dieters. Further, the duration of caloric deprivation ranged up to 24 h, which is presumably much longer than most dieters go without eating. Research in which participants decide how long they would calorically restrict themselves before the fMRI scans would have more external validity and would serve as a useful complement to the experimental caloric deprivation studies. Thus, in Study 1 we investigated neural activation in response to pictures of palatable foods, unpalatable foods, and glasses of water among adolescents who varied in degree of caloric restriction before the scan (which ranged from 1 to 16 h of caloric deprivation). By including both pictures of palatable and unpalatable foods, we were expressly able to determine whether degree of "self-inflicted" caloric deprivation correlates with hyper-responsivity of attention and reward regions for palatable versus unpalatable foods. In Study 2 we tested whether the number of hours since last caloric intake (which varied from 3 to 22 h) correlated with neural activation in response to receipt and anticipated receipt of a palatable food because only one previous study evaluated response to receipt of food (chocolate milk and chicken soup; Uher et al., 2006). Investigating neural responsivity to images of palatable food, anticipated receipt of palatable food, and actual intake of palatable food should provide a more comprehensive description of the impact of caloric restriction. We hypothesized that individuals who had deprived themselves of caloric intake for a longer period of time would show greater activation in regions implicated in attention and reward in response to images, anticipated receipt, and receipt of palatable food.

Another gap in the literature is that few studies have investigated the effects of elective longer-term caloric deprivation; the fMRI experiments described above only investigated the effects of acute caloric deprivation. It is important to investigate responsivity of attention and reward regions to food among individuals who are in a documented negative energy balance state for a prolonged period versus a balanced energy state or a positive energy balance state because dieting efforts are typically several weeks in duration (Williamson et al., 1992). In response to pictures of palatable foods, individuals who had successfully recovered from obesity showed greater activation in a region associated with inhibitory control (the superior frontal region) than obese and normal weight individuals, greater activation in a region involved in visual attention (middle temporal region) than normal weight individuals, and less activation in a motor readiness region (precentral gyrus) than obese individuals (McCaffery et al., 2009). Thus, in Study 2 we tested the hypothesis that participants in a negative energy balanced state over a 2-week period would show greater activation of attention and reward regions to receipt and anticipated receipt of a palatable food relative to participants who were in an energy balanced state or a positive energy balance state over this period. Participants in Study 2 determined whether they would be in a negative, neutral, or positive energy balance over the 2-week period during which the fMRI scans took place, thereby capturing elective dietary restriction. Another novel feature of these studies is that because we used larger samples (n = 34, 51, and 81) than employed in past studies (n = 10 to 20), we were able to conduct whole-brain analyses, rather than region of interest analyses, which should provide a more comprehensive and reliable index of the effect of elective dietary restriction in neural response to food.

Methods

Participants

In Study 1, participants were 34 healthy female adolescents (*M* age = 15.5 ± 0.9, BMI = 24.6 ± 5.6); 2% Asian/Pacific Islanders, 2% African Americans, 86% European Americans, 5% Native Americans, and 5% mixed racial heritage. In Study 2, participants were 162 adolescent males and females (*M* age = 15.3 ± 1.07, BMI = 20.8 ± 1.90); 4.1% Hispanic, 0.6% Native American, 0.6% Asian/Pacific Islanders, 76.5 European Americans, and 17.9% mixed racial heritage, however, for the present investigation analyses were performed on the 51 participants for whom we collected data on the degree of caloric deprivation before the scan and the 81 participants that met the weight change criteria described below. Those who reported binge eating or compensatory behaviors in the past 3 months, any use of psychotropic medications or illicit drugs, head injury with a loss of consciousness, or current Axis I psychiatric disorder per *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition criteria (American Psychiatric Association, 1994) were excluded. Informed consent was obtained from parents and assent from adolescents. The Oregon Research Institute Institutional Review Board approved these studies.

Anthropometrics and behavioral measures

Height was measured to the nearest millimeter using a direct reading stadiometer with the body positioned such that the heels and buttocks are against the vertical support of the stadiometer. Weight was assessed to the nearest 0.1 kg using digital scales with participants wearing light clothing without shoes or coats. At each anthropomorphic assessment, two measures of height and weight were obtained and averaged. BMI was then calculated as kg/m^2. For Study 2, weight was collected twice, 14 days apart. For each anthropometric assessment, participants fasted for 5–15 h and abstained from exercising for 24 h prior to weighing. To assess the impact of longer-term energy balance we used weight change over the 2-week period during which the scan occurred to group participants into three a priori groups; those that lost ≥1 kg (negative energy balance), those that remained within 0.1 kg of baseline weight (energy balance), and those that gained ≥1 kg (positive energy balance) over this period.