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Research report

An fMRI study of obesity, food reward, and perceived caloric density. Does a low-fat label make food less appealing?☆

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ABSTRACT

We tested the hypothesis that obese individuals experience greater activation of the gustatory and somatosensory cortex, but weaker activation of the striatum, in response to intake and anticipated intake of high-fat chocolate milkshake versus an isocaloric milkshake labeled low-fat and a tasteless solution using functional magnetic resonance imaging (fMRI) with 17 obese and 17 lean young women. Obese relative to lean women showed greater activation in somatosensory (Rolandic operculum), gustatory (frontal operculum), and reward valuation regions (amgydala, ventralmedial prefrontal cortex (vmPFC) in response to intake and anticipated intake of milkshake versus tasteless solution, though there was little evidence of reduced striatal activation. Obese relative to lean women also showed greater activation in the Rolandic operculum, frontal operculum, and vmPFC in response to isocaloric milkshakes labeled regular versus low-fat. Results suggest that hyper-responsivity of somatosensory, gustatory, and reward valuation regions may be related to overeating and that top-down processing influence reward encoding, which could further contribute to weight gain.

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Introduction

The incidence of obesity has reached epidemic proportions, which is concerning because obesity increases the risk for high blood pressure, adverse lipoprotein profiles, diabetes mellitus, atherosclerotic cerebrovascular disease, coronary heart disease, colorectal cancer, and death from all causes (Calle, Thun, Petrelli, Rodriguez, & Heath, 1999). Obesity results in over 111,000 premature deaths annually in the US alone (Flegal, Graubard, Williamson, & Gail, 2005). Theorists have proposed that individuals who experience hyper-responsivity of the mesolimbic reward system in response to anticipated and actual receipt of food are at increased risk for overeating (Davis, Strachan, & Berkson, 2004; Dawe & Loxton, 2004; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008). In contrast, others have hypothesized that obese individuals have hypofunctioning dopamine-based reward circuitry, which leads them to overeat to compensate for this deficiency (Comings & Blum, 2000; Wang, Volkow, & Fowler, 2002).

Although data from self-report, observational, and operant studies suggest that obese relative to lean individuals rate high-fat, high sugar foods as more pleasant and work harder for such palatable foods on computer tasks (Drewnowski, Kurth, HOlden-Wiltse, & Saari, 1992; Epstein et al., 2007; Saelens & Epstein, 1996; White, Whisenhunt, Williamson, Greenway, & Netemeyer, 2002), few studies have used brain imaging to examine whether there is hyper-responsivity of the reward circuitry in obese relative to lean adults. Several neuroimaging studies have found that obese versus lean individuals show greater activation in the lateral OFC, amygdala, insula, nucleus accumbens, ventral striatum, pallidum, caudate, somatosensory cortex and hippocampus in response to pictures of high-calorie versus low-calorie foods (Rothemund et al., 2007; Stice, Yokum, Bohon, Marti, & Smolen, 2010; Stoeckel et al., 2008). Further, weaker activation in the frontal operculum, lateral OFC and striatum in response to images of high-fat food predict future weight gain for individuals at genetic risk for reduced dopaminergic signaling, whereas elevated activation in these same regions predict future weight gain for individuals not at-risk (Stice et al., 2010).

Only two studies have examined the relation of body mass index (BMI) scores to activation in response to actual food intake; activation in gustatory and somatosensory regions to intake and anticipated intake of chocolate milkshake have correlated positively with BMI, but activation in the caudate nucleus in response

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to milkshake taste have correlated negatively with BMI (Stice, Spoor, Bohon, & Small, 2008; Stice, Spoor, Bohon, Veldhuizen, et al., 2008). Gustatory regions play a role in encoding food taste, somatosensory regions in encoding food texture, and the striatum in encoding reward value of stimuli. Thus, these findings suggest that individuals who show greater activation in somatosensory regions and the gustatory cortex in response to anticipation and consumption of food, but weaker activation in the striatum during food intake, may be at risk for overeating. To date, however, research has not expressly tested whether obese relative to lean individuals show hyper-responsivity in somatosensory and gustatory regions in response to food intake and anticipated intake and hypo-responsivity in the striatum in response to food intake, which should provide a more sensitive test of relations. Thus, the first aim of the present study was to test the hypothesis that obese relative to lean individuals would show greater activation in somatosensory and gustatory regions in response to food intake and anticipated intake, and hypo-responsivity in the striatum.

The second aim was to examine the effects of perceived caloric content of food on reward circuitry responsivity to food. Given that people typically rate low-fat foods as less pleasant tasting and rewarding than high-fat foods (Yeomans, Lartamo, Procter, Lee, & Gray, 2001), we suspected that foods labeled as low-fat would activate reward circuitry to a lesser degree than foods labeled as high-fat. This is an important question because of the preponderance of low-fat foods in our current environment. If low-fat foods are in fact less able to activate reward circuitry, people may overeat these same foods, thwarting the weight control benefits of purchasing such foods. Societal pressure to eat low-fat foods can also influence food choice, even if such foods are unappealing (Nestle et al., 1998). Behavioral studies show that adults concerned with their weight consume more of a labeled fat-free food compared to a regular fat food in *ad libitum* taste tests (Miller, Castellanos, Shide, Peters, & Rolls, 1998). Critically, obese adults consume more of an isocaloric food that is labeled low-fat versus regular (Wansink & Chandon, 2006). No studies have used objective brain imaging to investigate the ability of foods labeled as low-fat versus high-fat to activate reward circuitry. Thus, we included a milkshake labeled as "low-fat" which was isocaloric to a milkshake labeled as "regular" so that we could investigate the effects of the perceived caloric content of the food independent of any difference in the actual taste of the food. We hypothesized that the "high-fat" milkshake would result in greater activation than the "low-fat" milkshake to food intake and anticipated food intake for obese versus lean individuals.

Method

Participants

Participants were 38 female college students recruited from introductory social sciences courses and fliers posted on campus. Participants were selected if they reported that their body mass index (BMI) was between 20.0 and 25.0 or between 30.0 and 40.0, based on National Institutes of Health (1998) guidelines. Exclusion criteria were past or current treatment for a psychiatric or neurological illness, current diagnosis of an eating disorder, ferromagnetic devices in or on the body that would be a danger to the participant or would cause artifacts in the images (e.g. braces, tattoos, pacemaker), and allergies to chocolate or lactose. Data from two participants were excluded due to excessive head movement greater than 1 mm. Two other participants were excluded because their BMI when directly measured did not fall within the range for lean or obese. The final sample of 34 women was 11% Asian, 77% Caucasian, 5% Hispanic, 5% American Indian or Alaskan Native and 5% who indicated more than one race. The mean age of the sample was 20.1 years old (SD = 1.4; range = 18–23). Paternal education, a proxy for socioeconomic status, was 28% high school graduate or less, 44% some college, 22% college graduate and 5% advanced degree in this sample. Informed consent was obtained from all participants under the IRB-approved protocol. Participants received $100 or research participation credit for completing the study.

Procedure

Participants were asked to refrain from eating and drinking (except water) for 4–6 h before the imaging session. They reported a mean of 5.6 hours (SD = 1.19) since their last meal. We selected this deprivation period to capture the hunger state that most individuals experience as they approach their next meal, which is a time when individual differences in food reward may impact caloric intake. Hunger ratings were collected in a subset of participants ($n = 10$), which showed no difference in hunger between obese ($M = .56$, SD = .24) and lean groups ($M = .41$, SD = .18; $t[10] = -1.07$, $p = .32$, Cohen's $d = .71$). Participants also completed a semi-structured interview assessing diagnostic criteria for eating disorders and a questionnaire about eating behaviors and attitudes, and height and weight were measured.

fMRI paradigm

In this event-related paradigm, colored pictures of glasses of water, regular chocolate milkshake, low-fat chocolate milkshake or an empty glass was presented to participants. The pictures were labeled correspondingly (e.g. the empty glass was labeled, "nothing"). The pictures signaled the delivery of 0.5 mL of the same chocolate milkshake for the regular and low-fat chocolate milkshake conditions, a tasteless solution for water, or nothing at all. The chocolate milkshake consisted of 1 cup of vanilla ice cream, 1.5 cups of 2% milk and 2 tablespoons of chocolate syrup. The tasteless solution, which was designed to mimic the natural taste of saliva, consisted of 25 mM KCl and 2.5 mM $NaHCO_3$. The tasteless solution was used because water has a taste that activates the gustatory cortex (Zald & Parvo, 2000)

The pictures were presented using MATLAB with a digital projector/reverse screen display system to a screen at the back end of the MRI scanner bore. Participants viewed the screen via a mirror attached to the birdcage head coil. Tastes were delivered using programmable syringe pumps (Braintree Scientific BS-8000) controlled by MATLAB to ensure consistent volume, rate and timing of taste delivery (0.5 mL over 5 s). Sixty-milliliter syringes filled with milkshake and tasteless solution were connected via Tygon tubing through a wave guide to a manifold attached to the birdcage head coil in the MRI scanner. The manifold fit into the participants' mouths and delivered the taste to a consistent segment of the tongue. This procedure has successfully delivered liquids in the scanner and has been described in detail elsewhere (Stice, Spoor, Bohon, Veldhuizen, et al., 2008).

The taste was delivered 4–11 s ($M = 7.5$) after the onset of the picture. The picture remained on the screen for 6–11 s ($M = 8.5$) after the taste was delivered and participants were instructed to swallow when the picture went off. The next picture appeared 1–5 s ($M = 3$) after the prior picture went off. As a result, each event lasted 11–27 s and each run consisted of 38 events. Duration of stimulus presentation and order of presentation were randomized across participants. Each picture corresponded with the delivery of a particular taste or no taste. On 40% of the milkshake and tasteless solution trials, the taste was not delivered in order to examine the neural response to anticipatory reward that was not confounded by the receipt of the taste (unpaired trials). Versions of this milkshake paradigm have been found to validly measure

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