Binge-Eating Disorder: Reward Sensitivity and Brain Activation to Images of Food

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Background: The underlying neurobiological mechanisms that account for the onset and maintenance of binge-eating disorder (BED) are not sufficiently understood. This functional magnetic resonance imaging (fMRI) study explored the neural correlates of visually induced food reward and loathing.

Method: Sixty-seven female participants assigned to one of four groups (overweight BED patients, overweight healthy control subjects, normal-weight healthy control subjects, and normal-weight patients with bulimia nervosa) participated in the experiment. After an overnight fast, the participants' brain activation was recorded during each of the following three conditions: visual exposure to high-caloric food, to disgust-inducing pictures, and to affectively neutral pictures. After the fMRI experiment, the participants rated the affective value of the pictures.

Results: Each of the groups experienced the food pictures as very pleasant. Relative to the neutral pictures, the visual food stimuli provoked increased activation in the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and insula across all participants. The BED patients reported enhanced reward sensitivity and showed stronger medial OFC responses while viewing food pictures than all other groups. The bulimic patients displayed greater arousal, ACC activation, and insula activation than the other groups. Neural responses to the disgust-inducing pictures as well as trait disgust did not differ between the groups.

Conclusions: This study provides first evidence of differential brain activation to visual food stimuli in patients suffering from BED and bulimia nervosa.

Key Words: Binge-eating disorder, bulimia nervosa, disgust, fMRI, food, pictures

Binge eating has been recognized as a clinically relevant behavior among obese individuals for almost half a century (1). However, the proposal of a provisional mental disorder category, binge-eating disorder (BED), is relatively new (2). The research criteria of BED include the presence of recurrent binge episodes with impaired control and subsequent distress. Unlike bulimia nervosa (BN), in which inappropriate compensatory mechanisms (e.g., vomiting) are employed to counteract the effects of overeating, these regularly reoccurring behaviors are absent in BED. As a consequence, the afflicted individuals are often overweight or even obese. BED is a common mental disorder with a lifetime prevalence of approximately 3% in the general population (3). In samples drawn from weight-control programs, the prevalence is considerably higher (>30%) (2).

The neurobiological factors that account for the onset and maintenance of BED are not yet known in detail. Whereas in BN, dieting nearly always precedes the onset of binge eating, this pattern is typically reversed in BED (4). Here, binge eating usually predates the sporadic dieting attempts. Consequently, food restriction and malnutrition do not qualify as central binge triggers in BED. Negative emotional states (e.g., anger, sadness) have often been reported by BED patients to have preceded overeating episodes (5). It is of note that these triggers are not disorder-specific because they are also present in bulimia. Finally, the type of food seems to be of importance for binging in BED. The afflicted patients prefer high-caloric food with high fat and sugar content (6–8). This preference might be mediated by the reward value of these types of food.

Within this context, it has been suggested, based on the reinforcement sensitivity theory (RST) (9,10), that BED patients might have an elevated sensitivity for primary rewards such as food (11). The differences in sensitivity are mediated by the so-called behavioral approach system. Central underlying neural substrates are the nucleus accumbens, the amygdala, and the orbitofrontal cortex, which are innervated by dopaminergic pathways.

First evidence for a connection between reward sensitivity and overeating was supplied by questionnaire studies (11–13). Here, positive correlations between the degree of binging, the body mass index, and self-reports on reward sensitivity, were observed. Whether this response tendency in BED is mediated by the brain reward system has not yet been addressed and was therefore the focus of this functional magnetic resonance imaging (fMRI) investigation.

Only a few neuroimaging studies have analyzed brain responses to visual food cues in BED patients. To our knowledge, there is only one single photon emission computed tomography (SPECT) study on individuals with clinically relevant BED symptoms (14). After an overnight fast, the female patients were exposed to a freshly cooked lunch and a landscape picture (control condition). Relative to obese and lean nonbinging female subjects, the food presentation provoked increased left prefrontal activation in the patients. This activation was correlated with feelings of hunger. A further investigation (15) analyzed neural responses to pictures of high-caloric foods, low-caloric foods, and nonfood items in obese individuals with subclinical BED. The subjects were scanned after the intake of a standard meal. Compared with lean and overweight nonbingers, the viewing of high-caloric food provoked greater premotor

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Table 1. Characteristics of Study Samples

	BED M (SD)	BN M (SD)	C-NW M (SD)	C-OW M (SD)	Group Differences[a]
Age (years)	26.4 (6.4)	23.1 (3.8)	22.3 (2.6)	25.0 (4.7)	
BMI	32.2 (4.0)	22.1 (2.5)	21.7 (1.4)	31.6 (4.7)	BED, C-OW > BN, C-NW
Education (years)	13.0 (1.5)	12.7 (.8)	13.2 (.9)	12.5 (1.9)	
Blood glucose level (mg/dL)	92.5 (7.8)	83.2 (7.0)	86.9 (6.6)	92.8 (10.4)	BN < BED, C-OW
BDI	12.1 (4.6)	15.0 (8.8)	2.3 (2.4)	4.3 (2.7)	BED, BN > C-NW, C-OW
BAS	3.4 (.4)	2.7 (.6)	2.8 (.6)	2.8 (.4)	BED > C-NW, C-OW, BN
BIS	3.1 (.5)	2.7 (.9)	2.8 (.5)	2.6 (.5)	
QADS—total	2.3 (.6)	2.2 (.7)	2.3 (.4)	2.3 (.6)	
EDI—binging	14.4 (2.0)	15.8 (2.0)	.80 (1.4)	.65 (1.2)	BED, BN > C-NW, C-OW

BDI, Beck Depression Inventory; BED, binge-eating disorder; BAS, Behavioral Activation Scale; BIS, Behavioral Inhibition Scale; BMI, body mass index; BN, bulimia nervosa; C-NW, normal-weight control subjects; C-OW, overweight control subjects; EDI, Eating Disorder Inventory, subscale: binging; QADS, Questionnaire for the Assessment of Disgust Sensitivity.

[a]Statistically significant group differences: *t* tests, all $p < .05$, uncorrected.

activation in the obese bingers. The authors suggested that this activation reflects mouth movement preparation for food ingestion.

Up until now, reactivity to visual food stimuli has been primarily studied in healthy individuals without eating disorders. Some of these neuroimaging studies contrasted brain activation to food versus nonfood pictures. Activated regions included the orbitofrontal cortex (16–20), the ventral striatum (17), the amygdala (21), and the insula (16,18). Other investigations examined cerebral responses to foods differing in caloric content (22–24). These fMRI studies showed that high-caloric foods provoked greater activity of the medial prefrontal cortex and the dorsal striatum than low-caloric foods. A subsequent analysis of the data showed that the degree of positive affect induction while viewing high-caloric food was positively correlated with orbitofrontal cortex (OFC) activation (23). Correlation approaches had also been applied in other investigations in which hunger/appetite ratings were positively associated with insular and OFC activation (14,16,18).

Few studies have focused on food-motivation-relevant trait factors, such as reward sensitivity, that might also influence affective and neural responses to food. An exception is the study by Beaver *et al.* (17), who demonstrated that subjects' reward sensitivity scores significantly predicted activation of the ventral striatum, the amygdala, and the OFC while they looked at food pictures.

Based on reinforcement sensitivity theory (9), this fMRI study explored whether BED patients would be characterized by an elevated reward sensitivity and increased activation in reward-processing brain areas (e.g., OFC, ventral striatum) while viewing pictures depicting high-caloric food. Further, we investigated whether BED patients would display lowered disgust responsiveness. Because food does not develop aversive qualities for BED patients when eating beyond satiety during binging (25), we hypothesized that this inhibited aversion development might be mediated by decreased trait disgust. This was the second exploratory question for this investigation.

Methods and Materials

Participants

Females suffering from BED ($n = 17$), BN (purging type, $n = 14$) according to DSM-IV (2), and healthy control subjects with no previous history of eating disorders (normal-weight [C-NW]; $n = 19$; overweight [C-OW]; $n = 17$) participated in this study. We restricted the sample to women because they are more likely to suffer from BED (1.5 times) and BN (10 times) than men (2). The subjects had been recruited through announcements in local newspapers and at the university campus. Written informed consent was obtained from each subject before entry. All subjects were nonmedicated and right-handed. The groups had a comparable mean age [$F(3,63) = 2.6$, $p = .07$] and education duration [$F(3,63) = .7$, $p = .56$; see Table 1]. The majority of participants were students. The BED patients and C-OW subjects were obese and had a comparable mean body mass index [BMI; $t(32) = .3$, $p = .76$]. Bulimic and C-NW subjects had a comparable mean BMI [$t(31) = .6$, $p = .54$]. Clinically relevant depression led to exclusion from the study. However, the groups differed in their BDI scores [$F(3,63) = 26.8$, $p < .001$]. Post hoc *t* tests showed that the patient groups had higher scores than the control groups (all pairwise comparisons, $p < .001$; all *p* values for self-report data are not corrected for multiple comparisons).

The BED and the bulimic group reported a similar degree of binging (Eating Disorder Inventory) (26): binging subscale, $t(29) = 1.95$, $p = .06$ (Table 1). Also, the duration of the eating disorder was comparable (BN: M = 7.3 years, SD = 3.6, BED: M = 6.8 years, SD = 4.0; $t(29) = .5$, $p = .66$). Those patients interested in treatment were assisted with referrals. The study had been approved by the ethics committee of the German Society for Psychology.

Stimuli and Design

The participants passively viewed pictures depicting high-caloric food (e.g., French fries, ice cream, cake, chips), disgust-inducing items (e.g., dirty toilets, maggots), and neutral items (household articles), which had been matched for complexity, brightness, and color composition. Each picture category consisted of 15 pictures. The scenes were shown for 4 sec each in an event-related design. The presentation was pseudo-randomized with the restriction that two pictures of the same category were not allowed to follow each other. In addition, a null condition was introduced, where a fixation cross was shown for an interval ranging between 3.5 and 6 sec. This was done to ensure design orthogonality. The design was repeated once. The duration of the total experiment was 19 min including functional pre- and postscans.

Procedure

The study was conducted on 2 days. After a telephone screening, the participants were invited to a diagnostic session