Personality and Eating Behaviors: A Case–Control Study of Binge Eating Disorder

Caroline Davis, PhD[1,2,3]*
Robert D. Levitan, MD[2]
Jacqueline Carter, PhD[3]
Allan S. Kaplan, MD[2,3]
Caroline Reid, MA[1]
Claire Curtis, MA[1]
Karen Patte, MA[1]
James L. Kennedy, MD[2]

ABSTRACT

Objective: Questions have been raised about the validity of binge eating disorder (BED) as psycho-pathologically distinct from other forms of overeating. Our purpose was to ascertain whether BED individuals differed in important ways from nonbinging obese adults.

Method: BED adults were recruited from the community as were weight-matched (obese) and normal-weight control (NWC) groups. All groups were equivalent for age and gender distribution, and were assessed on several personality traits and eating behaviors.

Results: BED individuals and obese controls did not differ on the personality traits. Both were more reward sensitive, and had greater anxiousness, impulsivity, and addictive personality traits than NWC. However, BED individuals reported significantly greater hedonic eating compared with the obese, who had higher levels than NWC.

Conclusion: Our findings provided no evidence of a *psychological* identity unique to obese adults with BED although their eating behaviors are markedly hedonically driven—i.e., more responsive to factors external to physiological needs. © 2007 by Wiley Periodicals, Inc.

Keywords: binge eating disorder; obesity; personality; eating behaviors

(Int J Eat Disord 2008; 41:243–250)

Introduction

Inherent in the concept of a "psychiatric disorder" is that the proposed phenomenon is sufficiently *psycho*-pathological to differentiate it from the broad spectrum of normal psychology. Binge eating disorder (BED) presents an interesting case in this regard since its hallmark characteristics are largely *behavioral* and *somatic*—viz., episodes of excessive food consumption in the absence of any compensation, and not generally driven by physical hunger. A high proportion of cases is also overweight and obese.[1–3] The psychological distress that BED patients typically describe, in the form of guilt, embarrassment, and negative mood, could easily be construed as the natural emotional sequelae of a chaotic pattern of eating with associated weight gain, rather than as core etiological characteristics of the syndrome.

Such factors have spawned questions about the validity of BED as psychologically distinct from other forms of overeating, which are also related to elevated body weight. In other words, some have asked if BED is merely a behavioral subtype of immoderate food intake, or whether there is an underlying mental disturbance, which justifies its inclusion as an entity in the lexicon of the *Diagnostic and Statistical Manual of Mental Disorders*?[4] Concerns have been raised, for instance, about the content validity of the diagnostic criteria for BED as well as the reliability of the instruments used to diagnose BED.[5] Questions about the uniqueness of BED also stem from reports that these individuals are indistinguishable from obese nonbinge eaters in their response to obesity treatment.[6] Other related issues focus on whether BED is better understood as a variant or subtype of bulimia nervosa (BN), or as a point along a continuum of "binge spectrum disorder."[7] Debates such as these are at the heart of two broad foci in the area of BED research. Some studies have examined whether the binging behavior in BED differs qualitatively from that in BN, while others have investigated whether obese BED individuals differ in meaningful ways from those who are obese but do not binge-eat.

Accepted 30 April 2007.
Supported by the Canadian Institute of Health Research.
Correspondence to: Caroline Davis, York University, 343 Bethune College, 4700 Keele Street, Toronto, ON M3J 1P3, Canada. E-mail: cdavis@yorku.ca
[1] York University, Toronto, Canada
[2] Centre for Addiction and Mental Health, Toronto, Canada
[3] University Health Network, University of Toronto, Toronto, Canada
Published online 19 December 2007 in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/eat.20499

Binge Eating in BED

Very little is actually known about the mechanisms impelling the binge eating in BED. The *restraint theory* of BED[8] purports that calorie restriction plays a causal role in the development of compulsive overeating. However, most temporal sequencing studies refute this viewpoint because a sizeable percentage of BED individuals—in one as high as 81%[3]—began to binge before any of them dieted.[9,10] Moreover, a majority (63%) of treatment-seeking BED adults reported weight problems prior to regular binge eating and before attempts to lose weight.[11] Studies have also found strong links between binge eating and overweight/obesity in preadolescent children who are unlikely to have been on strict diets at such a young age.[12]

A real-time examination of the antecedents and consequences of binge-eating in BED found that the self-defined primary motivation for this behavior was to improve mood; it was not a response to hunger or an abstinence violation.[13] Other clinical research has described the positive *hedonic* component of the binging in BED. Many have reported a strong liking for the taste, the smell, and the texture of the food while they are binging.[14] Indeed, they "speak of their binge eating with some degree of affection," although this positive response could also reflect a post hoc rationalization and/or justification for their behavior. Nevertheless, these findings could also suggest a heightened reward sensitivity in BED, at least compared with their normal-weight counterparts. The endogenous opioid peptides in the brain's mesolimbic reward pathway are known to regulate the palatability of foods high in sugar and fat[15,16]—those typically consumed in abundance during a binge. Significant genetic variation in this regulatory mechanism has recently been found between BED and normal-weight control (NWC) participants,[17] suggesting a biological basis to overeating, especially in an environment like ours where tasty and calorically-dense foods proliferate. There are other compelling links between compulsive overeating and addictive drugs, both of which activate brain reward pathways.[18,19]

Case–Control Studies

Differences in food intake between BED individuals, and those who are obese but do not binge eat, have mostly been studied using laboratory eating paradigms. The results suggest that BED individuals ingest more calories than weight-matched controls when instructed to binge eating from a multiple-item array of food, although no consistent differences have been found in the proportion of macronutrients consumed by each group.[20–22] These studies are limited, however, by small sample sizes, by the absence of a NWC group, and by the artificial nature of binge eating in a laboratory setting under surveillance. Less intrusive assessment methods are needed to broaden our understanding of eating behavior patterns in BED.

Moreover, the few field studies in this area have shown that results do not converge consistently with the laboratory research. For example, in one study—where handheld computers were used to measure factors associated with eating—episodes labeled as "binges" by the BED group did not differ in calorie content or composition from those of the non-BED obese controls, although the former did report more frequent binges.[23] In a second study, using a similar methodology with a small sample of overweight BED and weight-matched controls, the investigators found that the groups showed more similarities than differences in terms of the frequency of binge eating as well as the factors that trigger these episodes.[24] If the *ecological momentary assessment* technology employed in these studies is valid, an important concern is raised about the diagnostic validity of BED.

Other research—albeit also limited in scope—has investigated *psychological* differences between BED and non-BED obese adults. For example, in an early study of psychiatric comorbidity,[25] BED participants were more likely than weight-matched controls to have a lifetime history of depression and anxiety disorders as well as higher rates of personality disorder. Mitchell and Mussell[26] also found that BED individuals had elevated levels of psychopathology—particularly mood disorders—and they tended to be more impulsive. In a recent community study, obese BED individuals reported greater depression and anxiety, and a trend towards greater alcohol and nicotine use/abuse, compared with obese individuals who screened negative for BED.[2] However, on dimensions of normal personality, *harm avoidance* was elevated for both groups. A considerable limitation of this last study is that height and weight were self-reported, and BED diagnosis relied solely on responses to two items of a questionnaire.

In a recent study of patients with BED, anxiety was the most frequently cited emotion prompting episodes of overeating, and emotional eating was related to more frequent binge episodes and more severe eating disorder symptomatology.[27] Converging evidence also comes from a study in which BED participants reported greater hunger and desire to eat following a physical stressor than did

obese non-BED participants.[24] There has been a longstanding view that stress results in *hypo*-phagia as a consequence of sympathetic nervous system activation.[28] However, a subset of animals and humans seem to show the opposite eating response to stressors.[29] It appears that a key factor in appetite response is whether the stress is chronic or intermittent. Persistently elevated levels of stress hormones like cortisol tend to increase the salience of pleasurable activities like the ingestion of sweet and fatty foods.[30]

The Present Study

Our primary goal in the study reported here was to ascertain whether BED individuals differed in important ways from obese adults who do not binge eat. To this end, we examined several personality traits relevant to the clinical profile of BED, as well as a range of eating behaviors associated with over-consumption. In so doing, we have expanded on previous research in several important regards. For instance, BED participants were recruited from the general community rather than treatment facilities, and their diagnosis was confirmed by established clinical interview criteria. To avoid a potential confound of body weight, we also included both a weight-matched (i.e., obese) and a NWC group in the study design.

Method

Participants

Adults between the ages of 25 and 45 years who met criteria for BED (N = 53: women = 41; men = 12) were recruited from posters placed at universities, local hospitals, and other public institutions, as well as from advertisements in local newspapers. A normal-weight (N = 59: women = 52; men = 7) and a non-bingeing obese (n = 52: women = 38; men = 14) control group were recruited in the same manner. The percentage of men and women did not differ significantly among the groups (χ^2 = 4.209, df = 2, p = .122). The proportion of Caucasians in the BED, normal weight, and obese control groups was 86.8, 81.4, and 80.8, consecutively, and the χ^2 test of independence for these data was also nonsignificant (χ^2 = 0.833, df = 2, p = .659).

The group mean ages were 35.0 (6.5) years for BED, 33.5 (7.5) years for normal weight controls, and 36.4 (6.5) years for the obese controls. Analysis of variance (ANOVA) procedures indicated that these differences were not statistically significant ($F_{2,161}$ = 2.49, p = .089). However, and by design, the three groups did differ significantly ($F_{2,161}$ = 81.80, $p < .0001$) on BMI (BED: 35.2 [8.9]; normal weight: 22.4 [2.8]; and obese: 39.0 [8.5]). Although both BED and the obese controls had a mean BMI in the Class II obesity range, according to World Health Organization criteria,[31] the least significant difference post hoc test indicated that BMI was significantly higher for the obese controls than for the BED participants (p = .008).

Measures of Personality

1. *Sensitivity to Punishment (SP) and Sensitivity to Reward (SR)* were assessed by the two scales of the *SPSR Questionnaire*.[32] Each comprises 24 forced-choice items reflecting the respondent's avoidance responses under conditions of punishment, and approach responses under various conditions of reward, respectively. These scales were developed to assess Gray's psychobiological model of personality[33,34]—a well-validated theory based on two independent neurobiological motivational systems: the behavioral inhibition system (BIS) and the behavioral activation system (BAS). The SPSR scales have shown good internal consistency, temporal stability, and concurrent validity.[35] The alpha coefficients for the present study were 0.88 for the SP and 0.79 for the SR.
2. *Impulsivity* was assessed by the 30-item, 4-point *Barratt Impulsivity Scale*,[36] which identifies three factors of impulsivity: the nonplanning aspects of this construct, as well as the tendency for one to act rashly, and to make quick decisions. Currently, this is the most widely used self-report measure of trait impulsivity. The highly significant correlations among the factors (r = .48–.52), and the high alpha coefficient in this study (0.84) for all 30 items, provides a good justification for our use of the total score in the statistical analyses.
3. *Novelty Seeking (NS), Harm Avoidance (HA), and Reward Dependence (RD)* were assessed by the 100-item *Tridimensional Personality Questionnaire*.[37] These scales were developed as measures of Cloninger's psycho-biological model of temperament. NS is characterized by exploratory activity, an aversion to monotony, and the tendency to respond to novel (rewarding) stimuli with excitement. HA is defined as the tendency to inhibit behavior in order to avoid punishing (or nonrewarding) stimuli. RD reflects the tendency to respond intensely to signals of reward—especially social reward—thereby fostering the maintenance and continuation of reward-inducing behaviors. In the present study, the alpha coefficients for the three scales are 0.75, 0.90, and 0.70, consecutively.
4. *Addictive Personality* was assessed by the 32-item Addiction Scale of the *Eysenck Personality Ques-*

tionnaire-Revised [EPQ-R].[38] This scale was derived empirically by identifying those items of the EPQ-R, at or beyond the 0.001 level of significance—and irrespective of scale—which differentiated male drug addicts from normal controls.[39] In addition to studies with drug addicts,[40] this scale has been validated with groups of problem drinkers,[41] pathological gamblers,[42] and participants with eating disorders.[43] The alpha coefficient in the present study was 0.79.

Measures of Eating Behaviors

1. *Emotional Eating and Externally-Driven Eating* were assessed by the *Dutch Eating Behavior Questionniare* [DEBQ].[44] The *Emotional Eating* subscale reflects the degree to which eating is prompted by emotional states like tension and worry rather than by hunger; and the *External* subscale, the degree to which one tends to overeat by the sight and smell of food. The third scale of the DEBQ (Dietary Restraint) was not deemed a useful index of overeating given its focus on dieting and calorie restriction. The alpha coefficients in the present study for Emotional Eating and External Eating were 0.96 and 0.82, respectively.
2. *Low-Fat Eating, Emotional Eating, Snacking on Sweets, Haphazard Planning, and Meal Skipping* were assessed by the *Eating Behavior Patterns Questionnaire*[45] [EBPQ].[a] These five factors reflect a broad range of eating behaviors that have been associated with health outcomes and BMI. The Low-Fat Eating items emphasis healthy food choices, attention to portion sizes, and care about the amount of fat in foods. The Emotional Eating scale is similar to the same-named subscale of the DEBQ (the two are correlated .79 in the present study). Snacking on sweets and meal skipping are self-evident from their titles. Haphazard planning reflects the tendency to eat fast food and restaurant meals and to dislike cooking and preparing meals at home. In the present study, the alpha coefficients for the scales were 0.75, 0.89, 0.75, 0.78, and 0.70, consecutively.

Procedures

Control participants were first screened during a structured telephone interview and excluded if they had any serious medical condition, were not fluent in English, were pregnant (or had recently given birth), and were currently being treated for (or had a history of) any psychiatric disorder, including eating disorders and substance abuse. BED participants were required to meet an operational definition of the disorder using ratings on the *Eating Disorder Examination*.[46] This definition was based on that provided in the main body of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV][4] where BED is defined as: "recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of bulimia nervosa" (p.550). This definition was operationalized in the following way. Participants had to report at least weekly objective binge episodes over the previous three months, but over this period they must not have vomited, fasted, or taken laxatives or diuretics as a means of controlling their shape or weight. Nor must they have met DSM-IV diagnostic criteria for BN or anorexia nervosa. BED diagnosis was established during a telephone interview carried out by trained personnel. The same exclusion criteria were applied to BED adults as to the control participants, except that we included BED and nonbinging obese participants who were being treated (or met criteria) for unipolar depression without psychotic symptoms (confirmed by a clinical interview prior to the beginning of the study) because of the high co-occurrence of BED, depression, and obesity. Four BED and two obese participants were comorbid for depression.

The procedures employed in this study were approved by the three Research Ethics Boards relevant to the institutional affiliations of the authors, and were carried out in accordance with the Declaration of Helsinki. On the day of testing, informed consent was obtained, and all relevant demographic information was obtained in a face-to-face interview. Participants then completed the questionnaire measures after which height and weight were measured. For BED participants, a structured clinical interview was carried out at the beginning of the testing session to confirm eligibility and identify comorbidities. For control participants, a brief nonpatient psychiatric screening took place, which included questions about substance use, depression, and disordered eating. At the end of the study, all participants were paid a stipend for their participation. The data reported in this paper are part of a larger study whose results will be published elsewhere.

Results

Personality Traits

A multivariate analysis of variance (MANOVA) compared the three study groups on seven personality variables. The multivariate F and all the uni-

[a]The EBPQ contains a 6th subscale not included in this study. We concluded that the Cultural/Lifestyle scale was problematic for a multicultural sample of participants because of its emphasis on aspects of Christian culture and religion (e.g. "On Sunday, I eat a large meal with my family" and "I eat at church socials"). The low alpha coefficient of this scale (0.64) also confirms its unreliability in our study.

TABLE 1. Multivariate and univariate analyses of variance for seven personality measures

	Multivariate		Univariate						
Source	df	F^a	SR[b]	SP[b]	Imp[b]	NS[b]	HA[b]	RD[b]	AD[b]
Group	2	3.78***	4.58**	10.94***	8.73***	5.49**	9.47***	1.44	15.87***
MSE			18.47	32.08	108.46	27.32	53.89	19.9	25.18

Note: The Multivariate *F* ratio was generated from Pillai's statistic.

SR, Sensitivity to reward scale of the SPSRQ; SP, sensitivity to punishment scale of the SPSRQ; Imp, Barrett impulsivity scale; NS, novelty seeking scale of the TPQ; HA, harm avoidance scale of the TPQ; RD, reward dependence scale of the TPQ; AD, addictive personality scale of the EPQ-R.

[a] Multivariate df = 14,300.

[b] Univariate df = 2,155.

** $p < .01$.

*** $p < .001$.

variate *F* values, except for the Reward Dependence scale, were statistically significant (see **Table 1**). Post hoc comparisons using the least significant difference test indicated that the BED and the obese control group did not differ on reward sensitivity, punishment sensitivity, impulsivity, harm avoidance, and addictive personality traits. However, both groups had higher mean scores than the normal weight control group. On the Novelty Seeking scale, the obese controls had significantly higher mean scores than their normal-weight counterparts, while the BED participants did not differ from either control group. **Table 2** presents means and standard deviations, and a summary of these findings.

TABLE 2. Mean scores on seven measures of personality as a function of participant group

	BED		Obese Control		Normal Control	
Variable	*M*	SD	*M*	SD	*M*	SD
Sensitivity to reward	11.0_a	4.1	11.2_b	4.2	$8.9_{a,b}$	4.5
Sensitivity to punishment	12.8_a	5.9	10.8_b	6	$7.7_{a,b}$	5.1
Impulsivity	67.0_a	11.5	67.7_b	10.9	$60.1_{a,b}$	8.8
Novelty seeking	16.8	5.8	18.4_a	5.1	15.1_a	4.8
Harm avoidance	17.8_a	7.6	17.3_b	8	$12.3_{a,b}$	6.4
Reward dependence	18.9	4.8	17.4	3.8	18.2	4.6
Addictive traits	15.5_a	5.1	14.3_b	4.2	$10.3_{a,b}$	5.6

Note: Means in a row sharing the same subscript (e.g. $_a$) are significantly different ($p < .05$), as calculated by Least Significant Difference post hoc tests.

Eating Behaviors

A second MANOVA compared the three groups on seven eating behaviors. Again, the multivariate *F* ratio, and all the univariate *F* values were statistically significant, except for the Meal Skipping variable (see **Table 3**). BED participants reported significantly more emotional eating (on both measures of this behavior), externally-driven eating, snacking on sweet foods, and haphazard meal planning than obese participants who, in turn, had greater values on all these variables than the NWC participants. **Table 4** lists means and standard deviations for each variable, and a summary of the post hoc tests.

Conclusion

Szasz[47] has argued that Western societies have transformed to "pharmacracies" where personal habits and problems are often defined as diseases, and treatments (of any sort) are prescribed only to people who are diagnosed as ill. In this spirit, BED has been criticized by some as the "pathologizing" of obesity. Others, however, maintain that it is a stable psychiatric syndrome, which is at least as chronic as other eating disorders,[48] and that it should be elevated from a provisional entry in the DSM-IV to an official diagnosis.[49] Our goal in the present study was to revisit this debate by employing a broad selection of psychological and behavioral measures to assess whether a community sample of individuals diagnosed with BED had an unique impairment, or whether they were better positioned as a subset of overweight individuals whose eating habits simply took the form of periodic episodes of great excess. An interesting dichotomy of results emerged.

Personality Traits

On five measures of personality, we found that BED and the nonbinging obese did not differ from each other, although both groups had significantly higher scores than the NWCs. The former were more sensitive to reward and to punishment, were more harm avoidant, more impulsive, and had more addictive personality traits. However, on the measure of novelty seeking, only the obese scored higher than the NWCs. In other words, obese individuals—whether characterized by binge eating or not—distinguish themselves from normal-weight adults by having a greater hedonic capacity, but a less stable emotional profile with more impetuousness and anxiety. Such findings provide no evi-

TABLE 3. Multivariate and univariate analyses of variance for seven eating behavior measures

	Multivariate	Univariate						
Source	F^a	Emot[b] (DEBQ)	Ext[b] (DEBQ)	Snack[b]	Haphaz[b]	Emot[b]	Low-Fat[b]	Skipping[b]
Group	9.25***	48.12***	27.07***	20.88***	21.44***	81.78***	4.48**	1.42
MSE		0.77	0.27	18.82	35.42	38.76	60.97	14.65

Note: The Multivariate *F* ratio was generated from Pillai's statistic.

Emot (DEBQ), Emotional eating scale of the DEBQ; Ext (DEBQ), external eating scale of the DEBQ; Snack, snacking on sweets scale of the EBPQ; Haphaz, haphazard planning scale of the EBPQ; Emot, emotional eating scale of the EBPQ; Low-Fat, low-fat eating scale of the EBPQ; skipping, meal skipping scale of the EBPQ.

[a] Multivariate df = 14,310.

[b] Univariate df = 2,160.

** $p < .01$

*** $p < .001$.

TABLE 4. Mean scores on seven measures of eating behavior as a function of participant group

	BED		Obese Control		Normal Control	
Variable	*M*	SD	*M*	SD	*M*	SD
Emotional eating (DEBQ)	3.8_a	0.9	3.2_a	1	2.2_a	0.8
External eating (DEBQ)	3.7_a	0.5	3.4_a	0.4	3.0_a	0.6
Snacking on sweets	20.4_a	4.2	18.0_a	4.7	15.1_a	4.2
Haphazard meal plans	28.2_a	6.1	24.3_a	5.8	20.8_a	6
Emotional eating	44.1_a	4.5	36.9_a	6.8	29.0_a	7
Low-fat eating	39.8_a	7.9	42.2	8.5	44.3_a	7
Meal skipping	14	4.2	14.4	3.2	13.2	4

Note: Means in a row sharing the same subscript (e.g. $_a$) are significantly different ($p < .05$), as calculated by Least Significant Difference post hoc tests.

dence of a psychological identity unique to obese adults with BED.

By contrast, most previous investigations[50] have reported a greater level of psychopathology in BED individuals, based largely on measures of psychiatric symptomatology and Axis I comorbid diagnosis. As a means of assessing BED psychopathology, however, comorbidity status has limitation since binging can be a *symptom* of psychiatric disorders such as atypical depression and seasonal affective disorder; as well as a drug-induced response to certain medications used to treat psychotic disorders. Nevertheless, it is important to acknowledge that BED adults in this study had a significantly lower mean BMI—albeit still in the obese range—than the nonbinging obese controls. Therefore it is possible that in BED adults with more severe obesity, a greater level of psychopathology may have emerged.

The relatively high levels of anxiousness found in both BED and obese participants may be a contributing factor to their elevated BMI. Glucocorticoid secretion is one of the most common hormonal responses to stress, and a key effect is to increase the compulsive nature of certain activities (such as addictive drug taking) and the salience of related stimuli.[30] Stress hormones also increase the consumption of what has been called "comfort food"—that is, highly palatable foods whose sensory qualities indicate increased calories.[30] This evidence meshes with reports that obese individuals are typically unable to distinguish between hunger and anxiety because they have learned to eat in response to both states.[51]

Eating Behaviors

When we examined group differences in eating behaviors, the pattern of findings was much different from the personality results. On five of the measures, BED individuals had significantly higher scores than the obese controls, who in turn had higher scores than the NWCs. BED individuals engaged in more emotionally driven eating, more eating in response to external/environmental cues, they snacked more on sweet foods, and reported more haphazard meal plans. Both BED and obese consumed fewer low-fat meals than NWCs, but in this regard they did not differ from each other.

There is accumulating evidence that overeating in obesity reflects heightened responsiveness to non-homeostatic stimuli, such as pictures of food or the aroma of cookies baking, rather than a defect in, or failure of, the regulatory systems involved in energy balance.[52] This finding is relevant since all the eating-behavior measures we used reflect food consumption that is motivated by something beyond the need to counteract the physiological signals of short-term energy depletion — what can be described as "hedonic eating." Lowe and Levine[53] make a nice distinction between *needing* food and *wanting* food, where the latter is primarily affected by the rewarding properties of the food and the reinforcement one receives from the experience of eating. Our data suggest that both BED and obese individuals are more responsive to factors external to their physiological needs such as the palatability, the availability, and even the con-

venience of the food. However, BED individuals, for reasons not identified in this study, have this tendency to a great degree. Indeed, their enhanced "wanting" of food could be a salient factor in the development of their binging behavior.

Summary and Conclusions

Our results suggest that from a psychological perspective, obese individuals with and without BED do not seem to differ—a finding which casts some doubt on the appropriateness of BED as a distinct psychiatric disturbance. However, what does distinguish the BED group from other obese adults is the degree of their hedonic eating behaviors. Given the strong biological basis of food-intake regulation,[54–56] a future research goal should be to examine neurophysiological differences between BED and nonbinging obese adults to investigate more thoroughly whether binge-eating is a phenomenon sufficiently different from other forms of overeating to warrant the label of "disorder."

Some strengths of our study are the inclusion of two weight-related control groups and the extensive set of outcomes measures we used. Clearly, however, there are other psychological traits that could be examined, and which may uncover salient differences between BED and non-binging obese individuals. For instance, personality measures of depressiveness and stress proneness may separate the two groups. It is also important to acknowledge that although we asked patients about feeling guilty, depressed and disgusted with themselves as part of the screening process (one part of Criterion B as set out the DSM-IV-TR for BED), we did not explicitly require that they meet the proposed Criterion C of "marked distress regarding binge eating". From clinical experience, we have found that all individuals meeting the other BED criteria have some degree of distress, and therefore we question the validity of differentiating an artificial threshold for "marked" distress in this population. This strategy may, however, have been conservative regarding the main study hypothesis as it limits the chance of biasing the BED group in terms of greater distress and therefore psychopathology. Finally, there is the possibility of a reporting bias on the part of BED participants. That is, people with BED may be more prone to provide emotionally significant reasons for eating as a way of explaining their aberrant eating behavior. On the other hand, and consistent with the findings of the present study, it may be that binging is simply a more severe form of overeating, and thereby a greater risk factor for obesity. In either case, stronger evidence than currently exists is needed to settle the debate.

References

1. Greeno CG, Wing RR, Marcus MD. How many donuts is a "binge"? Women with BED eat more but do not have more restrictive standards than weight-matched non-BED women. Addict Behav 1999;24:299–303.
2. Grucza RA, Przybeck TR, Cloninger R. Prevalence and correlates of binge eating disorder in a community sample. Compr Psychiatry 2007;48:124–131.
3. Manwaring JL, Hilbert A, Wilfley DE, Pike KM, Fairburn CG, Dohm F-A, et al. Risk factors and patterns of onset in binge eating disorder. Int J Eat Disord 2006;39:101–107.
4. American Psychiatric Association. Diagnostic and Statistical Manual—Version IV. Washington, DC: American Psychiatric Association Press, 1994.
5. Devlin MJ, Goldfein JA, Dobrow I. What is this thing called BED? Current status of binge eating disorder nosology. Int J Eat Disord 2003;34:S2–S18.
6. Stunkard AJ, Allison KC. Two forms of disordered eating in obesity: Binge eating and night eating. Int J Obes Relat Metab Disord 2003;27:1–12.
7. Hay P, Fairburn C. The validity of DSM-IV scheme for classifying bulimic eating disorders. Int J Eat Disord 1998;23:7–15.
8. Howard CE, Porzelius LK. The role of dieting in binge eating disorder: Etiology and treatment implications. Clin Psychol Rev 1999;19:25–44.
9. Abbott DW, de Zwaan M, Mussell MP, Raymond NC, Seim HC, Crow SJ, et al. Onset of binge eating and dieting in overweight women: Implications for etiology, associated features and treatment. J Psychosom Res 1998;44:367–374.
10. Mussell MP, Mitchell JE, de Zwaan M, Crosby RD, Seim HC, Crow SJ. Clinical characteristics associated with binge eating in obese females: A descriptive study. Int J Obes 1996;20:324–331.
11. Reas DL, Grilo CM. Timing and sequence of the onset of overweight, dieting, and binge eating in overweight patients with binge eating disorder. Int J Eat Disord 2007;40:165–170.
12. Goossens L, Braet C, Decaluwe V. Loss of control over eating in obese youngsters. Behav Res Ther 2007;45:1–9.
13. Stein RI, Kenardy J, Wiseman CV, Dounchis JZ, Arnow BA, Wilfley DE. What's driving the binge in binge eating disorder? A prospective study examination of precursors and consequences. Int J Eat Disord 2007;40:195–203.
14. Mitchell JE, Mussell MP, Peterson CB, Crow S, Wonderlich SA, Crosby RD, et al. Hedonics of binge eating in women with bulimia nervosa and binge eating disorder. Int J Eat Disord 1999;26:165–170.
15. Smith KS, Berridge KC. Opioid limbic circuit for reward: Interaction between hedonic hotspots of nucleus accumbens and ventral pallidum. J Neurosci 2007;27:1594–1605.
16. Will MJ, Franzblau EB, Kelley AE. The amygdala is critical for opioid-mediated binge eating of fat. Neuroreport 2004;15: 1857–1860.
17. Reid C, Davis C, Levitan RD, Carter JC, Kennedy JL, Kaplan AS, et al. The μ-1 opioid receptor (OPRM1), food preferences, and the risk for binge eating disorder. Presentation at the Western Obesity Summit, Alberta, Canada, May 2007.
18. Holden C. 'Behavioral' addictions: Do they exist? Science 2001;294:980–982.
19. Volkow ND, Wise RA. How can drug addiction help us understand obesity? Nat Neurosci 2005;8:555–560.

20. Goldfein JA, Walsh BT, LaChaussee JL, Kissileff HR, Devlin MJ. Eating behavior in binge eating disorder. Int J Eat Disord 1993;14:427–431.
21. Guss JL, Kissileff HR, Devlin MJ, Zimmerli E, Walsh BT. Binge size increases with body mass index in women with binge-eating disorder. Obes Res 2002;10:1021–1029.
22. Raymond NC, Bartholome LT, Lee SS, Peterson RE, Raatz SK. A comparison of energy intake and food selection during laboratory binge eating episodes in obese women with and without binge eating disorder diagnosis. Int J Eat Disord 2007;40:67–71.
23. Greeno CG, Wing RR, Shiffman S. Binge antecedents in obese women with and without binge eating disorder. J Consult Clin Psychol 2000;68:95–102.
24. le Grange D, Gorin A, Catley D, Stone AA. Does momentary assessment detect binge eating in overweight women that is denied at interview? Eur Eat Disord Rev 2001;9:309–324.
25. Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. Association of binge eating disorder and psychiatric comorbidity in obese subjects. Am J Psychiatry 1993;150:1472–1479.
26. Mitchell JE, Mussell MP. Comorbidity and binge eating disorder. Addict Behav 1995;20:725–732.
27. Masheb RM, Grilo CM. Emotional overeating and its associations with eating disorder psychopathology among overweight patients with binge eating disorder. Int J Eat Disord 2006;39: 141–146.
28. Gluck ME. Stress response and binge eating disorder. Appetite 2006;46:26–30.
29. Greeno CG, Wing RR. Stress-induced eating. Psychol Bull 1994; 115:444–464.
30. Dallman MF, Pecoraro N, Akana SF, La Fleur SE, Gomez F, Houshyar H, et al. Chronic stress and obesity: A new view of 'comfort food'. Proc Natl Acad Sci USA 2003;100:11696–11701.
31. WHO Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity. Geneva: World Health Organization, 1998.
32. Torrubia R, Avila C, Molto J, Caseras X. The sensitivity to punishment and sensitivity to reward questionnaire (SPSRQ) as a measure of Gray's anxiety and impulsivity dimensions. Personality Individual Differen 2002;31:837–862.
33. Gray JA. The neuropsychology of emotion and personality. In: Stahl SM, Iverson SD, Goodman EC, editors. Cognitive Neurochemistry. Oxford, UK: Oxford University Press, 1987, pp. 171–190.
34. Gray JA. Brain systems that mediate both emotion and cognition. Cogn Emot 1990;4:269–288.
35. Caseras X, Avila C, Torrubia R. The measurement of individual differences in behavioural inhibition and behavioural activation systems: A comparison of personality scales. Personality Individual Differen 2003;34:999–1013.
36. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsivity scale. J Clin Psychol 1995;51:768–774.
37. Cloninger CR. A systematic method for clinical description and classification of personality variants. Arch Gen Psychiatry 1987; 44:573–588.
38. Eysenck HJ, Eysenck SBG. Manual of the Eysenck Personality Scales. London: Hodder & Stoughton, 1991.
39. Gossop MR, Eysenck SBG. A further investigation into the personality of drug addicts in treatment. Br J Addict 1980;75:305–311.
40. Sigurdsson JF, Gudjonsson GH. Personality characteristics of drug-dependent offenders. Nordic J Psychiatry 1995;49:33–38.
41. Ogden ME, Dundas M, Bhat AV. Personality differences among alcoholic misusers in community treatment. Personality Individual Differen 1988;10:265–267.
42. Clarke D. Gambling and the trait of addiction in a sample of New Zealand university students. New Zealand J Psychol 2003;32:39–48.
43. Davis C, Claridge G. The eating disorders as addiction: A psychobiological perspective. Addict Behav 1998;23:463–475.
44. van Strien T, Frijters JE, Bergers GP, Defares PB. The Dutch eating behavior questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. Int J Eat Disord 1986;5:295–315.
45. Schlundt DG, Hargreaves MK, Buchowski MS. The eating behavior questionnaire predicts dietary fat intake in African American women. J Am Diet Assoc 2003;103:338–345.
46. Fairburn CG, Cooper Z. The eating disorder examination. In: Fairburn CG, Wilson GT, editors. Binge Eating: Nature, Assessment, and Treatment. New York: Guilford Press, 1993, pp.317–360
47. Szasz T. What counts as disease? The gold standard of disease versus the fiat standard of diagnosis. Independent Rev 2005; 10:325–336.
48. Pope HG, Lalonde JK, Pindyck LJ, Walsh T, Bulik CM, Crow S, et al. Binge eating disorder: A stable syndrome. Am J Psychiatry 2006;163:2181–2183.
49. Hudson JI, Hiripi E, Pope HG, Kessler RC. The prevalence and correlates of eating disorders in the national comorbidity survey replication. Biol Psychiatry 2007;61:348–358.
50. de Zwaan M. Binge eating disorder and obesity. Int J Eat Disord 2001;25(Suppl 1):S51–S55.
51. Canetti L, Bachar E, Berry EM. Food and emotion. Behav Process 2002;60:157–164.
52. Mela DJ. Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. Appetite 2006;47:10–17.
53. Lowe MR, Levine AS. Eating motives and the controversy over dieting: Eating less than needed versus less than wanted. Obes Res 2005;13:797–806.
54. Epstein LH, Leddy JJ. Food reinforcement. Appetite 2006;46: 22–25.
55. Kelley AE, Berridge KC. The neuroscience of natural rewards: Relevance to addictive drugs. J Neurosci 2002;22:3306–3311.
56. Kelley AE, Schiltz CA, Landry CF. Neural systems recruited by drug- and food-related cues: Studies of gene activation in corticolimbic regions. Physiol Behav 2005;86:11–14.